The Blue Cross Book

For the advancement of the veterinary profession

MSD Animal Health

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From Editor's Desk

The Editorial Board of the "Blue Cross Book" is happy to place before its esteem readers the 30th volume of this professional publication of MSD Animal Health. It has always been the endeavor of this publication to disseminate the latest information which way contribute to enhance health status, productivity and usefulness of domestic livestock and companion animals.

During last few issues, The "Blue Cross Book" tried to enlighten its readers about the 'Oxytocin', a hormone, though useful, surrounded by myths. The present issue is publishing yet another article, explaining the vital role of oxytocin in animal production and reproduction. Presently, the 'Blue Cross Book" is receiving large number of informative articles on management and health aspects of companion animals. 'Fading Puppy Syndrome' is an example, indicative of new dimensions the Indian Veterinarian is adding to companion animal management during early post-natel life. The Editorial Board of 'Blue Cross Book' expects in future such newer information on housing management, nutrition during health and illness, so also on health control measures in canines and felines.

Considering that pet animal practice has been gaining ground among practicing Veterinarians even in semi-urban areas, information about the infections of bacterial origin in canines and felines and their therapeutics with appropriate antibiotics in adequate dosages has been elaborated in detail for the pet practitioners.

Bovine infertility and toxic episodes of varying etiology are the topics of larger interest among field Veterinarians. The present issue deals with these topics, providing important and useful therapeutic considerations.

The 'Blue Cross Book' appeals to fellow Veterinarians and Animals Scientists to share their expertise and experience for the benefit of the larger cross section of Indian Veterinarians through this media.

Wishing all the readers a happy festive season which is in the offing.

Dr. Yash Goyal
Managing Director,
MSD Animal Health

Dear Professional Colleagues,

It gives me an immense pleasure and opportunity to present the 30th edition of our technical journal, "The Blue Cross Book''. With this, I wish to extend my sincere thanks to all the fellow professionals, who regularly contribute their knowledge and experience for updating this journal, dedicated to veterinary profession.

We, in MSD Animal Health, give utmost importance to clinical research that generates the field data. This we incorporate in ''The Blue Cross book'' to build confidence in Veterinary practitioners. We thus update a Veterinary practitioner, who uses this advanced technology as the key to his success in dealing with the problems related to livestock health and productivity.

The scientific innovations and advanced technologies today mean a combined effort of all stake holders including the field professionals and needy customers. We constantly partner with Veterinary Colleges and Universities, research institutions and Veterinary professionals for scientific update. Our close knit with field Veterinarians also helps us to strengthen our scientific base.

MSD-Animal Health has been in the Indian market with products of international repute in livestock, poultry and companion animal segments, offering hormones, anti-infectives, ecto and endo-parasitic controls, supportive medicines and biologicals, produced in our most sophisticated manufacturing sites, both locally and globally.

By virtue of having own manufacturing site for biologicals, supported by R&D and Service laboratory at Pune, MSD-Animal Health has been in a very strong position to cater the customer needs with its products at affordable prices, along with diagnostic support to combat the livestock and poultry diseases prevalent in India.

We sincerely believe that you would enjoy reading this compilation with professional interest and support MSD Animal Health further by providing your valuable feedback, which has helped us a long way in our mission to serve Veterinary profession and our customers in the best possible way, in the years to come.

Best Wishes,

Yash Goyal

THE SCIENCE NEVER STOPS

With a worldwide network of dedicated pharmaceutical and vaccine R&D and manufacturing sites, Merck Animal Health is better adapted to provide local solutions to our partners, helping them tackle both global and regional diseases.

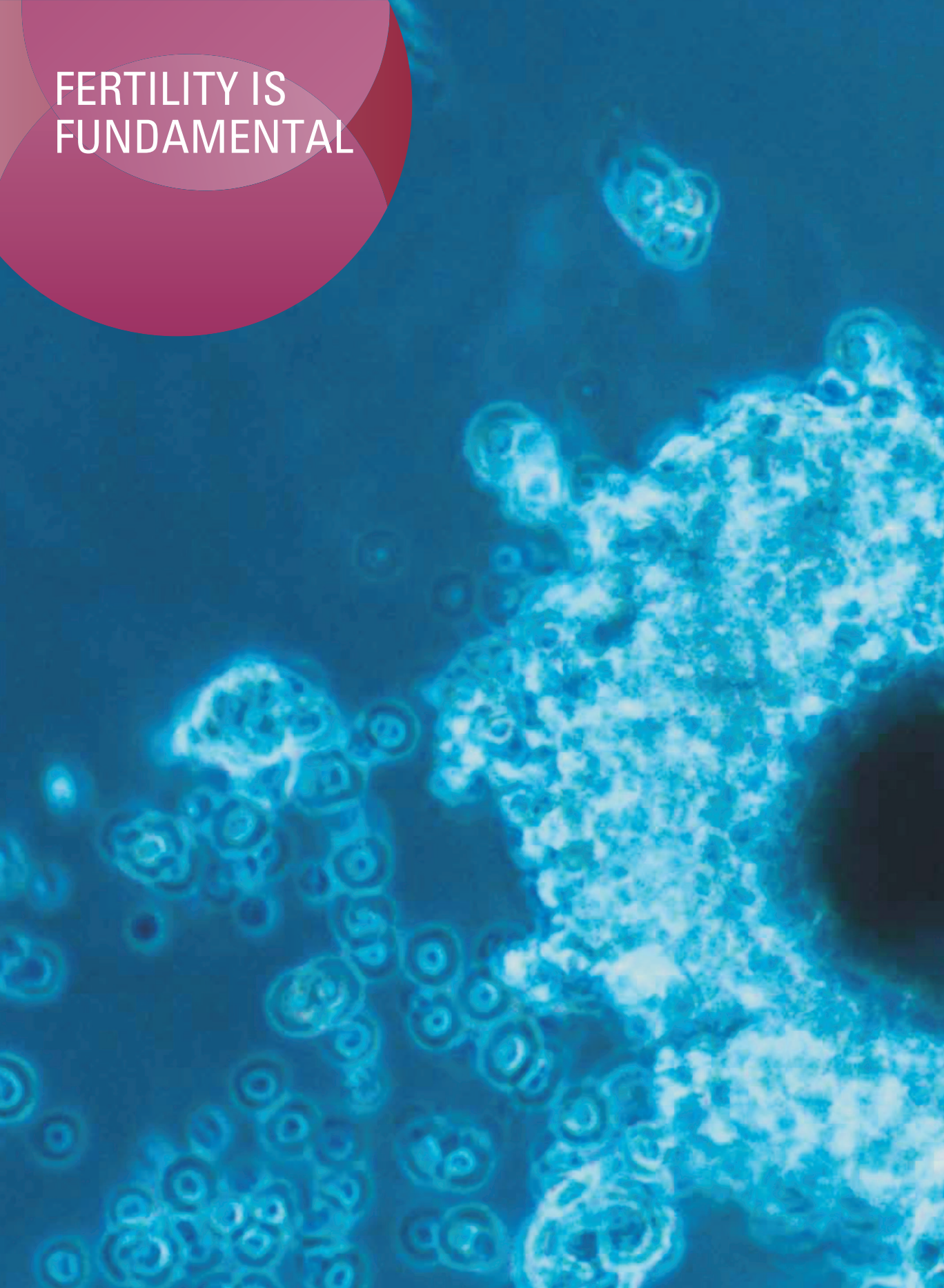
- Several dedicated world-class pharmaceutical research sites and multi-species R&D centers of excellence for vaccines
- More than 100 ongoing pharmaceutical R&D projects (focused on anti-parasitics, anti-infectives, endocrine drugs and pain medicine)
- More than 100 ongoing biological R&D projects (focused on zoonotic, respiratory, enteric and emerging diseases and enabling technologies)
- Global manufacturing network

Nevertheless, the pace of scientific innovation today means no company can afford to work in isolation. We combine our in-house R&D expertise with an active business development and in-licensing program, and constantly partner with specialist biotechnology companies, universities and research institutions.

Working closely with our human health colleagues also helps us exploit the strength of our combined science base. Our POSA TEX eardrops, for the treatment of otitis externa in dogs, was originally developed as ASMANEX to treat asthma in humans. Our antifungal drug posaconazole was first marketed as NOXAFIL for prophylaxis and treatment of fungal infections for people.

70-80% OF OUR PRODUCT PIPELINE REACHES THE MARKET. THE INDUSTRY-LEADING BREAKTHROUGHS OUR CUSTOMERS EXPECT FROM US REQUIRE THE BEST SCIENTISTS, AND THE BEST FACILITIES IN THE WORLD.

FERTILITY IS FUNDAMENTAL



Eradicating disease protects our food supplies. But good fertility is vital too. Merck Animal Health's fertility programs ensure consistent, healthy breeding and safeguard supplies of protein to the world's meat markets.



More meat and dairy in our diets increases pressure on producers to keep breeding levels high and predictable.

Merck Animal Health's fertility programs for production animals have several integral elements:

- Hormones that synchronize reproduction cycles
- Products that facilitate insemination and optimize breeding strategies like embryo transfer
- Vaccines that protect against infections that affect fertility and could pose a threat to human health
- Tools that help diagnose and treat uterine infections in cattle

For cattle, our hormone portfolio includes RECEPTAL (buserelin), and ESTRUMATE (cloprostenol). For swine, PG 600 (combination of gonadotropins) promotes a more fertile estrous cycle, and REGUMATE (altrenogest), is an oral treatment used in large farms to optimize the breeding process.

Our hormones are administered in parallel with vaccinations against diseases that damage reproduction. BOVILIS BVD for example, successfully protects cattle from abortions caused by bovine viral diarrhea virus. Similarly, OVILIS Toxovax and OVILIS Enzovax can prevent abortions caused by toxoplasmosis and chlamydophilosis in sheep and goats.

To reduce the movement of animals between farms, and the spread of undetected disease, artificial insemination is important to producers. Merck Animal Health also helps them build a stronger genetic pool with tools for more efficient artificial insemination.

Managing reproduction in horses is a complex process that requires expert knowledge. Our fertility products help professional stud farms and private individuals optimize their breeding programs.

Clinical Reports

The information on 'Brucellosis' has been accessed through internet.

Role of Oxytocin in animal Production and Reproduction

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Introduction:

Oxytocin is a nonapeptide hormone secreted from posterior pituitary. It is widely known for its role in female reproduction, which is released after distension of cervix and vagina during labor and after stimulation of the nipples thereby facilitating birth and breast feeding, respectively. Clinical use of this hormone is limited to the induction of parturition, expulsion of placenta and hastening of uterine involution. However, the hormone is widely used by dairy farmers for the induction of milk let down. Moreover, there are several other functions of oxytocin which are little known. Oxytocin plays an important role in pair bonding, blood pressure regulation, maternal behaviour, circadian timing of birth, initiation of parturition etc. The present review puts forward some uncommon functions of oxytocin, in animal production and reproduction.

Chemistry :

Oxytocin was discovered, isolated and synthesized by Vincent du Vigneaud in 1953, for which he received the Nobel Prize in Chemistry in 1955. Oxytocin is a polypeptide of nine amino acids (nonapeptide) and the aminoacid sequence is cysteine - tyrosine - isoleucine - glutamine - asparagine - cysteine - proline - leucine - glycine, wherein cystein forms a disulphide bridge. Oxytocin has a molecular mass of 1007 daltons. One international unit (IU) of oxytocin is equivalent to about 2 micrograms of pure peptide. The structure of oxytocin is very similar to vasopressin, which is also a nonapeptide with a sulfur bridge whose amino acid sequence differs by 2 amino acids (cysteine - tyrosine - phenylalanine - glutamine - asparagine - cysteine - proline - arginine - glycine). Oxytocin and vasopressin are the only known hormones released by posterior pituitary. However, oxytocin neurons secrete other peptides including corticotropin-releasing hormone (CRH) and dynorphin which act locally in the brain.

Synthesis, storage and release

Oxytocin is synthesized in magnocellular neurosecretory cells of supraoptic and paraventricular nuclei of the hypothalamus. Thereafter, it is transported and stored in posterior pituitary and from there released into the blood, following suitable stimulus. Oxytocin is also synthesized by some of the neurons of paraventricular nucleus that projects to other parts of brain and spinal cord. Oxytocin is derived from a large peptide fragment of the giant precursor protein molecule known as neurophysin by enzymatic cleavage. Secretion of oxytocin from the neurosecretory nerve endings is regulated by the action potential that propagates down the axons to the nerve endings in the pituitary. The nerve endings contain large numbers of oxytocin-containing vesicles, which are released by exocytosis as

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soon as the nerve terminals are depolarized. A number of factors can inhibit oxytocin release such as acute stress. Oxytocin release is suppressed by catecholamine released from the adrenal gland in response stressors.

Functions

Oxytocin has both peripheral (hormonal) and local actions. The actions of oxytocin are mediated by specific, high affinity oxytocin receptors. The oxytocin receptor is a rhodopsin-type (class I) G-protein-coupled receptor which requires Mg2+ and cholesterol for action. Oxytocin secreted from pituitary cannot re-enter the brain because of blood-brain barrier. Instead, the behavioral effects of oxytocin are thought to reflect release from centrally projecting oxytocin neurons which are different from those projecting to the pituitary gland. Oxytocin receptors are expressed by neurons in many parts of the brain and spinal cord, including the amygdala, ventromedial hypothalamus, septum and brainstem.

Functions of the oxytocin:

Uterine contraction - The pregnant uterus is one of the targets of oxytocin. In ruminants, oxytocin triggers the pulsatile release of PGF2 alpha by the endometrium at the end of an infertile estrous cycle to induce luteolysis. Oxytocin receptor is up regulated during the late luteal phase and at estrus. It is hypothesized to be down regulated during early pregnancy by the concept us derived trophoblastic interferon (IFNT) (Spencer and Bazer 2004). Around the onset of labor, uterine sensitivity to oxytocin markedly increases. This is associated with both an up regulation of oxytocin receptor mRNA levels and a strong increase in the density of myometrial oxytocin receptors, reaching a peak during early labor (Fuchs et al, 1995). Thus, at the onset of labor, oxytocin can stimulate uterine contractions at levels that are ineffective in the non pregnant state. After parturition, the concentration of oxytocin receptors rapidly declines. In rats, the uterine oxytocin receptor mRNA levels decrease more than sevenfold within 24 h (Zingg et al 1995). Possibly, the down regulation of the oxytocin receptors may be necessary to avoid unwanted contractile responses during lactation when oxytocin levels are raised.

Milk ejection - The stimulation of tactile receptors at teat/nipples site generates sensory impulses that are transmitted to the spinal cord and then to the secretory oxytocinergic neurons in the hypothalamus, leading to release of oxytocin into the bloodstream by which oxytocin is carried to the lactating breasts (Gimpl and Fahrenholz, 2001). There it causes contraction of the myoepithelial cells in the walls of the lactiferous ducts, sinuses, and breast tissue alveoli. This process is called milk ejection or milk let-down reflex and continues to function until weaning. Oxytocin administration increases the fat content of the milk. However, protein and lactose content do not differ significantly (Murray et al 1986). In an another study, Linzell and Peaker (1971) found increase in Sodium, Chloride, non-casein protein and decrease in Potassium and Lactose in milk following the oxytocin treatment. Lactating cows have more than 20 percent of milk as cisternal fraction (Bruckmaier and Blum 1998) but in buffaloes, cisternal milk accounts to about 5 percent (Thomas et al. 2004). This could be the reason for more milk let down time in buffaloes (Ludri 1980). Bidarimath M and Aggarwal A (2007) studied the effect of oxytocin on cisternal, alveolar fractions and milk quality in buffalo. Buffaloes were injected with oxytocin @ 2.5 IU (group I) and 5.0 IU (group II)

intramuscularly for a period of 1 month. There was no significant change in cisternal, alveolar and total milk yield by exogenous oxytocin administration. Fat percent in cisternal, alveolar and total milk decreased significantly ($P<0.01$) by 11.8% and 21.3% in groups I and II buffaloes, respectively. Protein percent increased significantly in group II. A significant increase was observed in somatic cell counts of milk by 5.36% and 6.22% in both the groups, respectively as compared to control group.

Sexual arousal – Oxytocin plays an important role in orgasms of both males and females. Oxytocin levels in the blood are significantly elevated in both sexes during sexual arousal and orgasm (Carmichael et al 1987). In males, oxytocin is said to facilitate sperm transport in ejaculation. Oxytocin injected into the cerebrospinal fluid causes spontaneous erections in rats ((Gimpl and Fahrenholz, 2001), reflecting actions in the hypothalamus and spinal cord.

Pair bonding - In females, oxytocin released into the brain during sexual activity is important for forming a monogamous pair bond with her sexual partner (Liu and Wang 2003). Plasma concentrations of oxytocin have been reported to be higher in people falling in love. Oxytocin has a role in social behavior in many species, and so it seems likely that it has similar roles in humans. It has been suggested that deficiencies in oxytocin pathways in the brain might be a feature of autism.

Anti-stress functions - Oxytocin reduces blood pressure and cortisol levels, increasing tolerance to pain, and reducing anxiety. Oxytocin may play a role in encouraging "tend and befriend", as opposed to "fight or flight" behavior, in response to stress (Uvnas-Moberg and Petersson 2005).

Increasing trust and reducing fear - In a risky investment game, nasally administered oxytocin displayed "the highest level of trust" twice as the control group (Kosfeld 2005). Nasally-administered oxytocin has also been reported to reduce fear, possibly by inhibiting the amygdala, which is thought to be responsible for fear responses (Kirsch et al, 2005).

Drug tolerance – Animal models have shown that oxytocin inhibits development of tolerance to various addictive drugs viz. opiates, cocaine, alcohol and reduces withdrawal symptoms (Kovacs 1998).

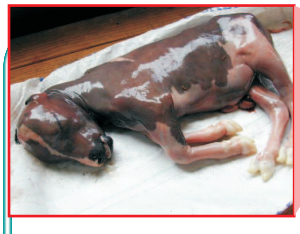
Learning & Memory - Certain learning and memory functions are impaired by centrally-administered oxytocin. Systemic oxytocin effects on memory retrieval might be mediated through an oxytocin-induced decrease in glucocorticoid release. It has been shown, for instance, that post-training systemic administration of oxytocin in mice produces an amnestic effect on the step-through inhibitory avoidance (Gimpl and Fahrenholz, 2001).

Embryonic development - Oxytocin and oxytocin receptors are found in the heart, and the hormone appears to play an important role in the embryonal development of the heart by promoting cardiomyocyte differentiation. (Paquin 2002, Jankowski 2004).

Establishment of maternal behavior: Successful reproduction in mammals demands that mothers become attached and nourish their offspring immediately after birth. It is also important that non-lactating females do not manifest such nurturing behavior. Oxytocin acts centrally to influence maternal and mating behaviour in rodents (Schulze and Gorzalka 1991). During parturition, there is an increase in concentration of oxytocin in cerebrospinal fluid,

and oxytocin acting within the brain plays a major role in establishing maternal behavior. Infusion of oxytocin into the ventricles of the brain of virgin rats or non-pregnant sheep rapidly induces maternal behavior. On the other hand, oxytocin antagonist administration into the brain prevented mother rats from accepting their pups.

Renal functions : Kallikrein-Kinin system Brattleboro rats were shown to concentrate their urine to hypertonic levels upon severe dehydration and the plasma levels of oxytocin increased 6-fold under this condition (Edwards et al 1984). The renal kallikrein-kinin system is thought to be a system that increases the urinary excretions of sodium and water (Schuster et al 1984). Urinary kallikrein is secreted from the distal collecting tubules, which acts on a low molecular weight kininogen to generate kinin in the renal tubular lumen. The kinin generated in the tubules binds to the bradykinin B2-receptors distributed over the collecting tubules (Tomita et al 1984), thereby inhibiting sodium and water reabsorption to induce the increase in urinary excretion of sodium and water. Renal kallikrein is also rapidly inactivated by kallistatin, a natural inhibitor, secreted from the renal tubules (Zhou et al, 1992). The increases in urine volume and excretions of sodium, chloride and potassium by OT-infusion were reduced by aprotinin, a kallikrein inhibitor, and Hoe 140, a bradykinin B2-receptor antagonist. Tomita et al. (1985) reported bradykinin, administered into the renal tubules, caused the inhibition of net sodium absorption without affecting net potassium transport or the transepithelial potential difference and bradykinin inhibits electroneutral NaCl absorption in the rat cortical collecting duct. These results indicate that kinin accelerates renal excretion of sodium, chloride and water, but not potassium. In conclusion, the natriuretic action of oxitocin is mediated through kallikrein kinin system.

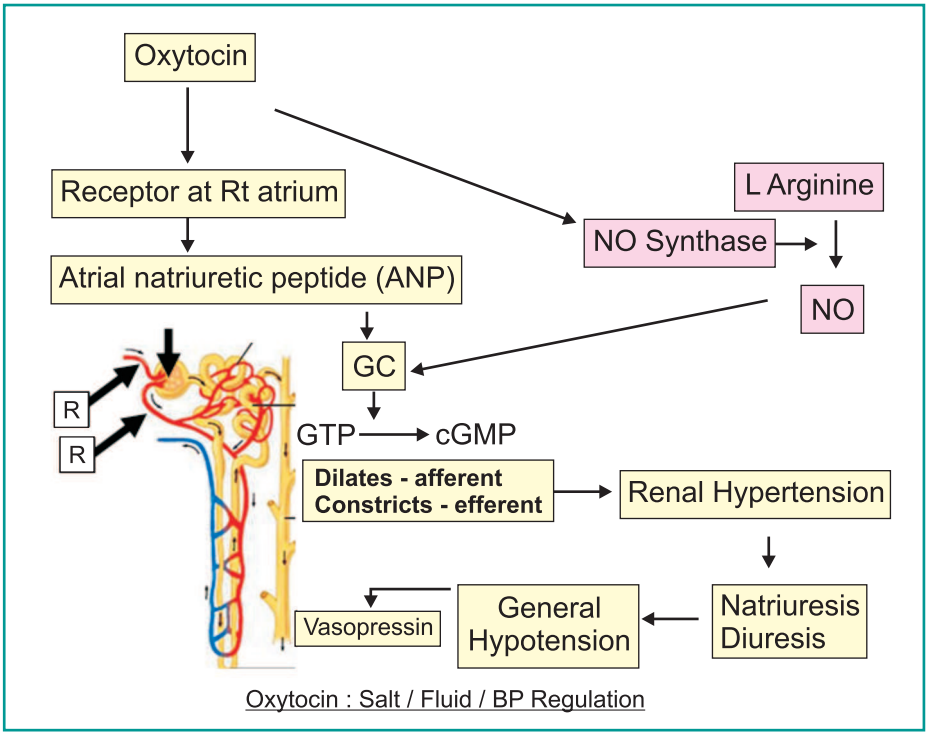


Brucellosis : A hurdle in Farm Livestock Development

What Do They Say?

WHO FAO OIE

"We regard **brucellosis** as the World's most widespread of all Zoonosis & apart from its toll on people, it has Enormous impact on the Animal Industry".



Oxytocin : Salt / Fluid / BP Regulation

Circadian Timing of Birth

The duration of pregnancy is guided by two interacting clocks - an interval timer measuring the overall length of gestation and a circadian timer defining birth time over a period of 24-hour (Viswanathan and Davis, 1992). Animals (hamsters, rats, mice, sheep, and humans) exhibit a robust circadian rhythmicity for the onset of labor, though this timing does not correlate with either active or inactive periods or position in a light-dark cycle. The circadian timing for birth depends upon the hypothalamic suprachiasmatic nucleus (SCN) (Reppert et al, 1987). However, the interval timer appears independent of the SCN, as lesioned animals deliver on average at the appropriate duration of gestation (Miller et al, 2004). One of the signaling pathways by which the SCN could communicate to control parturition is via the hypothalamic oxytocin. The oxytocin rhythm does not shift with changes in the light cycle during gestation indicating that it is controlled by a circadian pacemaker distinct from the master clock that regulates locomotor activity. The circadian pattern of oxytocin regulation is a primary determinant of the circadian timing of labor. This endogenous rhythm in oxytocin rather than environmental cues from light or darkness determines the hour of birth (Lincoln and Porter, 1976). In the absence of oxytocin, the circadian gating of birth is lost following a shift in the light cycle (Miller et al 2004). Further, oxytocin has both actions that delay the onset of labor by maintaining luteal function or promote labor by augmenting uterine contractions. Circadian release might enhance tissue-specific peripheral changes in oxytocin sensitivity to

focus the timing and progression of labor (Imamura et al 2000). Thus, oxytocin plays a critical role in minimizing disruption of labor in the face of resetting the master circadian clock.

Initiation of Parturition

Fetal brain is a possible source of oxytocin just before and during labor under hypoxic stress. Chen and Du (1999) showed that acute hypoxic stress induced by a hypobaric chamber triggered an intensity- and duration-dependent in situ release of oxytocin in the median eminence of adult rats. Pro-oxytocin forms accumulate in the magnocellular neurons of fetal rats starting from embryonic day 18-19 and a surge of fully processed oxytocin is seen in pituitary glands at 20-21 (Altstein and Gainer, 1988). Van der Sluis (1986) also established that the oxytocin content of fetal pituitary glands dramatically dropped during labor, which further argues in favor of a fetal pituitary release of oxytocin during this period.

Neuroprotection

Tyzio et al. (2006) indicated that maternal oxytocin exerted a neuroprotective action on fetal neurons during parturition. They induced anoxia and aglycemia in fetuses and demonstrated that the onset of anoxic depolarization (as a marker of neuronal death) in the fetal hippocampi was significantly accelerated when the mothers or the fetuses were previously treated with oxytocin receptor antagonists.

Female sexual maturation

Oxytocin has been shown to stimulate Gonadotropin-Releasing Hormone (GnRH) secretion from medial basal hypothalamic explants of adult male rats (Rettori et al 1997) and of cycling female rats in proestrus (Selvage and Johnston 2001). Sexual maturation involves an acceleration of pulsatile GnRH secretion (Bourguignon et al 1992). Oxytocin stimulates GnRH secretion in prepubertal male rats (Parent et al 2005) and the administration of oxytocin antagonist blunted the preovulatory LH peak in women (Evans et al 2003).

Oxytocin metabolism

The plasma protein binding of oxytocin is very low (~30%) and has minimum crossing through blood brain barrier and blood placental barrier. Oxytocin is biotransformed in plasma, placenta and uterus by the enzymes: Aminopeptidase and Oxytocinase by hydrolysis (Jenkins and Nussey, 1991). The enzyme activity increases with gestation i.e. maximum at parturition which declines after delivery. There is little or no degradation by plasma of male and non pregnant animals. In kidneys, lactating mammary gland and liver, biotransformation is through breaking of disulphide bond to form glycinamide which is mainly excreted through urine as inactivated glycinamide (65-70%) and oxytocin (30-35%). Less than 1% is excreted unchanged in milk. Being polypeptide, it is rapidly degraded in stomach. However, acute oral and S/C toxicity have been shown @ 20.5 mg/kg b.wt in rats (Syntocinon data sheet, 2006).

Conclusion

Apart from playing an important role in uterine contraction and milk ejection, oxytocin also regulates blood pressure, maternal behavior, antistress effect, female sexual maturation, neuroprotective, initiation of parturition, circadian rhythm in parturition and sexual arousal.

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Antibiotic Therapy for Pet Practitioners

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Introduction :

Antibiotics are most commonly prescribed for suspected infections and also for prophylactic use. This article provides guidelines for selection of antibiotics under these two circumstances. Although the term 'antibiotic' strictly refers to chemical substances produced by micro-organisms, information in this article is extended to include synthetic antibacterial agents, such as sulphonamides and quinolines.

There is a temptation to use broad-spectrum antibiotic regimens, with activity against gram-negative, gram-positive, aerobic and anaerobic bacteria. An extended spectrum of antibacterial activity is often not needed unless there is a mixed infection, as overuse of broad-spectrum antibiotics may promote emergence of microbial resistance. Patient factors, which influence the selection of antibiotics, include the status of the immune system, the location of the infection, the presence of concurrent diseases (e.g. hepatic or renal failure) and the physiological status (e.g. pregnant or neonatal animal). An antibiotic regimen with an extended spectrum of activity using bactericidal drugs may be required in a neutropenic patient, particularly in situations where microbial culture tests from a patient with a suspected infection are pending. However, in patients with normal neutrophil function and numbers, it is not necessary to select an antimicrobial agent based on bactericidal versus bacteriostatic properties of the drug.

When an infection is suspected, the ideal antibiotic dosing regimen would be designed after results of bacterial culture and antimicrobial susceptibility testing are known. Cytological evaluation of infected material, including gram stain, may assist in preliminary identification of involved bacteria. However, absolute bacterial identification and antimicrobial susceptibility is usually not known at the time therapy is initiated, and antibiotics are administered empirically. Practitioners hardly rely on bacterial culture results (only about 10%) when instituting an antibiotic therapy. Empirical selection of antibiotics can be made using a rational approach. This approach should be based on a knowledge of which bacteria are most likely to infect specific sites, and the tissue distribution and spectrum of activity of specific antibiotics. Table 1 is used to define classes of antibiotics that are referred to in the text. Initially, antibiotics of choice should be selected. Once the site of infection has been identified, alternative antibiotics should be tried for those animals that do not have a favorable response to the initial antibiotic therapy, when culture results are not yet available or if the drugs of choice are contraindicated in a particular patient. Local factors at the site of infection should be considered because they will influence the activity of antibiotics. For example, pus or necrotic debris in an infection will decrease the activity of aminoglycosides and trimethoprim-sulphonamides.

Table 1 : Classification of antibiotics

Class	Antibiotics
Tetracycline's	Oxytetracycline, Doxycycline
Aminoglycosides	Amikacin, Gentamicin, Tobramycin
Macrolides and lincosamides	Erythromycin, Lincomycin, Clindamycin
Penicillins	PenicillinG, Ampicillin
Penicillinase-resistant penicillins	Cloxacillin, Dicloxacillin, Oxacillin
Clavulanic acid-potentiated amoxicillin	Ticarcillin-clavulanate, Imipenem-cilastin
First generation Cephalosporins	Cephalexin, Cefazolin, Cephalothin Cephapirin, Cefadroxil
Second-generation cephalosporins	Cefotetan, Cefoxitin
Third-generation cephalosporins	Cefotaxime, Cefixime
Fourth-generation cephalosporins	Cefquinome
Quinolones	Enrofloxacin, ciprofloxacin, Orbifloxacin, Marbofloxacin, Difloxacin

An anaerobic environment inhibits the antibacterial activity of aminoglycosides. Certain drugs should be used judiciously or not given to animals that are pregnant or growing or have hepatic or renal failure. Client concerns, particularly costs and dosing frequency of an antibiotic, may also affect the selection of specific antimicrobial agents.

Antibiotic selection for treatment of bacterial infections :

A. Cutaneous infections :

Almost all superficial pyoderma in dogs and most in cats are caused by *Staphylococcus intermedius*. Cytological evaluation of exudate from pustules can be used to confirm the presence of gram-positive cocci. Antibiotic therapy can be started without submitting specimens for bacterial culture and susceptibility. While most deep pyoderma in dogs is caused by *Staphylococcus intermedius*, some are complicated with a mixed infection of gram-negative bacteria. For dogs with deep pyoderma, cytological evaluation and bacterial culture and susceptibility testing of purulent exudate is warranted before initiation of antibiotics. Final antibiotic selection should subsequently be modified, if needed, based on these results. Whereas three weeks of treatment may be sufficient to treat superficial pyoderma, six to eight weeks, or longer, may be needed for the treatment of deep pyoderma. Although cats frequently get abscesses, deep pyoderma is rare. Cat abscesses are usually caused by *Pasteurella multocida* but can be caused by mixed infections involving anaerobic bacteria. Cat abscesses can usually be managed by establishing drainage and administering amoxycillin, ampicillin, clavulanic acid-potentiated amoxycillin or cephalexin. If abscesses do not resolve with this treatment,

L-form bacteria may be present, and a therapeutic trial with tetracyclines should be considered.

B. Urinary tract infections : (UTI)

Bacterial urinary tract infections are common in dogs but uncommon in cats. The clinical signs depend upon the location of infection. Animals with UTI localized to the bladder or urethra have clinical signs limited to the urinary tract, whereas, those with pyelonephritis or prostatitis may have systemic signs (depression, fever, evidence of septicaemia). Many dogs respond to one of several empirically administered antibiotics, but dogs with non-responding or recurring chronic infections require a more thorough evaluation including localization of infection, identification of predisposing causes and antimicrobial susceptibility testing of the involved pathogen.

Urinalysis and urine culture are particularly important for animals with a previous history of Urinary Tract Infection or with medical risk factors for developing subclinical Urinary Tract Infection, such as diabetes mellitus or hyperadrenocorticism. Many antibiotics reach very high concentrations in the urine, allowing for the relatively straightforward treatment of acute, uncomplicated Urinary Tract Infection by the administration of an antibiotic for ten to 14 days. If, after one course of empirical antibiotic therapy, signs of Urinary Tract Infection recur, microbial culture and susceptibility testing of the urine is always indicated. Disorders that can lead to chronic relapsing Urinary Tract Infection include pyelonephritis, prostatitis, urolithiasis, hyperadrenocorticism, chronic glucocorticoid treatment, anatomic defects and diabetes mellitus. Prostatitis should be suspected in any sexually intact male animal presenting with bacterial cystitis.

Empirical antibiotic therapy should not be initiated in animals suspected to have any of the above conditions but antibiotic selection should instead be based on bacterial culture and susceptibility testing. Antibiotics that reach effective concentrations in the urine do not necessarily reach effective concentrations in the prostate and kidneys. In addition, some antibiotics are less active in acidic urine. The quinolones (e.g. enrofloxacin) and the potentiated sulphonamides (e.g. trimethoprim-sulphamethoxazole) reach high concentrations in the prostate and are the drugs of choice for the treatment of prostatitis. Antibiotics should be administered for four to eight weeks to animals diagnosed with prostatitis, pyelonephritis or chronic cystitis. Susceptibility tests should ideally be repeated three to five days after therapy is initiated, to ensure effectiveness, and monthly for several months after therapy stops, to detect recurrence.

C. Respiratory tract infections :

Bacterial rhinitis in the dogs and cats almost always occurs secondarily to another disorder. Clinical improvement associated with antibiotic therapy is usually transient, with signs recurring either during treatment or after antibiotic therapy is stopped. The nasal passages have abundant normal flora, making it difficult to interpret culture results; however, heavy growth of one or even two pathogenic organisms may suggest infection and aid in antibiotic selection. If a favourable response to an antibiotic selected on the basis of culture is noted, treatment should continue for six to eight weeks.

Antibiotic therapy may not be helpful in the treatment of canine infectious tracheobronchitis because most antibiotics do not reach sufficiently high concentrations in

bronchial secretions and infections are often self-limiting. However, *Bordetella bronchiseptica* can colonise the lower airways and antibiotic therapy may be warranted. Antibiotic therapy should be administered for at least ten days, or five days beyond resolution of clinical signs.

Because it is often difficult to predict which bacterial species is responsible for the infections of the lower respiratory tract, tracheal or bronchial specimens should be obtained for cytological examination and microbial culture before initiating antibiotic therapy. For most antibiotics, interstitial and alveolar drug concentrations are similar to serum concentrations; however, bronchial concentrations may be lower and, for most commonly used antibiotics, are not influenced by the presence of mucosal inflammation. Because diffusion of drugs into bronchial secretions is low, antibiotic choices are limited. In all cases, appropriate supportive care should be provided. The response to therapy should be monitored through repeated physical examination and thoracic radiographs. If the antibiotic and dosage regimen is appropriate, a favourable response should be noted within 48-72 hours. Antibiotic administration should be continued for at least one week beyond resolution of clinical signs.

Effective management of pyothorax requires antibiotic therapy based on culture and susceptibility test results, drainage of the pleural cavity, and supportive care. During the initial management, antibiotics should be administered parenterally because these animals are frequently septicaemic. Antibiotic therapy should be continued for four to six weeks after drainage of the pleural cavity is complete.

D. Skeletal infections :

Septic arthritis requires rapid intervention and aggressive management in order to eliminate the infection and salvage the joint. Parenteral antibiotics may be necessary during the initial treatment period to ensure sufficiently high drug concentrations in the joint. Once inflammation has been reduced, oral antibiotic therapy can be started and should continue for four to six weeks, or for two weeks beyond complete resolution of clinical signs. The high end of the dose range listed for antibiotics will usually produce adequate drug concentrations in synovial fluid. In some cases, arthrotomy and lavage may be required, particularly if clinical signs are severe or if the animal has open growth plates.

Appendicular osteomyelitis also requires aggressive management. Most antibiotics reach adequate concentrations in bone when administered at recommended doses. However, antibiotic penetration into the site of infection may be impeded if blood supply is decreased by a sequestrum, haematoma or implant, making the treatment of appendicular osteomyelitis difficult. Acute osteomyelitis may require only four to six weeks of antibiotic therapy. However, in chronic osteomyelitis, antibiotic therapy alone is usually ineffective without surgical removal of sequestra and débridement. Fractures must be stabilised, with grafts placed in severe bone defects. In contrast, uncomplicated discospondylitis often responds readily to appropriate antibiotic therapy with dogs that have mild or no neurologic dysfunction, response commencing within five days of starting antibiotic therapy.

Parenteral antibiotics may be needed if there is evidence of bacteraemia or septicaemia, but in most cases oral antibiotics administered for six

to eight weeks is effective. **Tetracyclines should never be administered for bone infections because their antibacterial activity is reduced in bone.**

E. Mastitis :

Bacterial mastitis is uncommon in dogs and quite rare in cats. Before beginning therapy, a sample of milk or aspirated fluid should be obtained aseptically and submitted for bacterial culture. Milk antibiotic concentrations after systemic treatment are highest for macrolides, quinolones and trimethoprim and lowest for beta-lactams and aminoglycosides. The use of quinolones (i.e. ciprofloxacin, orbifloxacin, difloxacin or enrofloxacin) and tetracyclines (i.e. oxytetracycline or tetracycline) should be avoided in animals that are still nursing pups or kittens as these antibiotics can be harmful to the growing animal. Antibiotics should continue for ten to 14 days beyond resolution of inflammation. In severe cases of mastitis, antibiotics may be needed throughout lactation. Hot compresses applied to the affected glands and milking may be useful in promoting drainage and preventing abcessation. Occasionally, surgical débridement is required.

F. Septicemia/bacterial endocarditis :

In the case of life-threatening septicaemia, antibiotics with activity against gram-negative, gram-positive, aerobic and anaerobic bacteria should be administered intravenously. Because it is often difficult to predict which bacteria are responsible for the infection. Serial blood cultures should be initiated before administering antibiotics. If antibiotics have already been administered, an antibiotic removal device should be used. A search for the site of infections should be conducted.

Initial antibiotic therapy typically includes combinations of drugs. Once the site of infection is identified and results of microbial culture and susceptibility testing are available, the spectrum can be narrowed to target the known pathogen. Some antibiotics, including cefoxitin, cefotetan, imipenem-cilastatin, third-generation cephalosporins and ticarcillin-clavulanate, are usually reserved for infections with gram-negative bacteria that have proven multiple drug resistance rather than be used for routine broad-spectrum empirical therapy. Furthermore, third-generation cephalosporins and imipenem-cilastatin induce resistance by stimulating beta-lactamase synthesis.

G. Perioperative antimicrobial prophylaxis:

Antimicrobial prophylaxis is defined as the systemic administration of an antimicrobial agent in the absence of infection. The aim of prophylactic therapy is to prevent infection by reducing the number of viable bacteria present in the wound at the time of surgery to a level that normal host defences can handle. Antimicrobial prophylaxis is not a substitute for good surgical technique. In order for antimicrobial prophylaxis to be effective, the surgeon must take into account the type of surgery, the potential pathogens, the competence of the host and the pharmacologic and antibacterial properties of the antimicrobial agent.

Contaminating bacteria enter the wound from either exogenous sources or the patient's endogenous flora. Exogenous sources include surgical equipment, the operating room and operating personnel. Contamination with the patient's endogenous flora probably accounts for a higher percentage of postoperative infections. The source is typically the patient's skin or mucosal surfaces that have been



transected during surgery. Bacteria can be haematogenously spread to the surgical incision from distant overt or occult septic foci or dental procedures. Whenever possible, septic foci should be treated or eliminated before an elective surgical procedure. The pathologic bacteria that most frequently contaminate a surgical wound are *Staphylococcus sp*, followed by *Escherichia coli*. However, other gram-negative bacteria and enterococci may also contaminate wounds. Anaerobic bacterial contamination may occur during colonic surgery.

The infection rates in patients with contaminated wounds is about 15-20% and antimicrobial prophylaxis is generally warranted. Open, fresh contaminated wounds may not require systemic antimicrobial prophylaxis if wounds are appropriately managed within four hours of their occurrence. Regardless of the type of wound, antimicrobial prophylaxis may be warranted if there is extensive tissue damage, large accumulation of blood or drainage material in the wound or an immunocompromised patient is suspected. Antibiotic administration will probably not prevent infection in wounds with indwelling drains or catheters in place but may, in fact, promote development of infections with antibiotic-resistant bacteria. Whenever possible, antibiotic therapy should be withheld until drains and catheters placed in the wound have been removed.

Drugs should be administered using a regimen that maintains therapeutically effective concentrations in the tissues, with peak concentrations being achieved at the time of incision. Ideally, a single intravenous bolus of drug should be administered 20-30 minutes before incision. If the procedure lasts longer than three to four hours, a maintenance dose should be given so that effective concentrations are sustained. The risk of contamination in primarily closed wounds is present until a fibrin seal develops between wound edges (approximately three to five hours postoperatively). Another bolus of drug should be given just before the end of surgery so that effective serum concentrations of antibiotic are present, provided that the risk of bacterial contamination exists and the patient is compromised from anaesthesia and intraoperative stress. This last dose also ensures that high concentrations of drug are incorporated into the healing tissue. In the absence of documented infection, antibiotic administration should not be continued beyond 24 hours of surgery.

When selecting a drug for antimicrobial prophylaxis, those that have the potential for producing systemic toxicity (e.g. aminoglycosides) should be avoided. The selected drug should have activity against the pathogens most likely to contaminate the site. For most procedures, drugs active against beta-lactamase-producing Staphylococcus sp. should be administered. This would include beta-lactamase-resistant penicillins and first-generation cephalosporins. For those procedures that are more likely to be associated with gram-negative bacteria, cephalosporins may be more effective. Owing to its ability to reach high tissue concentrations, and to its efficacy and relative low cost, cefazolin is probably the cephalosporin of choice although cephalothin and cephapirin could also be used. Cefazolin should be administered at 22 mg/kg every two hours throughout the procedure.

When anaerobic contamination or more resistant gram-negative bacilli are a concern, cefoxitin or cefotetan may be more active.

When surgery is planned for the lower gastrointestinal tract, multiple enemas and oral antimicrobial agents efficacious against anaerobic and gram-negative bacteria should be given starting 36-48 hours before surgery, to reduce the number of bacteria in the colon. Neomycin sulphate plus metronidazole, or metronidazole alone, could be used. In emergency colon surgery, cefoxitin or an aminoglycoside-clindamycin combination should be administered parenterally.

Table 2 : Antibacterial therapy for cutaneous infections in dogs and cats

Type of infection	Pathogens involved	Antibiotic of choice	Alternative antibiotic	Culture/ susceptibility testing
Superficial pyoderma (Dogs and Cats)	*Staph intermedius*	Clavulanic acid-potentiated amoxycillin First-generation cephalosporin Penicillinase-resistant penicillin	Potentiated sulphas, Erythromycin, Fluoro-quinolone, Lincomycin Clindamycin	Usually not needed but indicated if no response to primary antibiotic therapy
Deep pyoderma (Dogs)	*Staph intermedius*	Penicillinase-resistant penicillin 1st-gen. cephalosporin Clindamycin	Quinolone Erythromycin Aminoglycoside	Culture of exudate or surgically prepared skin biopsy sample before initiating antibiotic therapy
Abscessation (Cats)	*Pasteurella multocida*	Clavulanic acid-potentiated amoxycillin 1st-gen cephalosporin	-	Usually not needed but indicated if no response to primary antibiotic



Table 4 : Antibacterial therapy for Respiratory Tract Infections in Dogs and Cats

Type of infection	Pathogens involved	Antibiotic of choice	Alternative antibiotic	Culture/ susceptibility testing
Tracheo-bronchitis (Dogs and Cats)	*Bordetella bronchiseptica, Mycoplasma spp*	Tetracyclines Potentiated sulphonamide Quinolone	-	Not needed in uncomplicated tracheo-bronchitis. Tracheal wash fluid if non- responding or recurrent
Bronchi and pulmonary parenchyma (Dogs)	*Escherichia coli, Klebsiella spp* *Pasteurella spp* *B. bronchiseptica*	Potentiated sulphas 1-gen. cephalosporin Quinolone, Clavulanic acid-potentiated amoxycillin	Doxycycline Aminoglycoside	Tracheal or bronchial wash fluid before initiating antibiotic therapy
Pleural cavity (Dogs)	*Actinomyces spp* *Nocardia spp* *Anaerobes*	Ampicillin-sulbactam Clavulanic acid-potentiated amoxycillin	Potentiated sulphas Clindamycin	Pleural fluid sample before initiating antibiotic therapy
Bronchi and pulmonary parenchyma (Cats)	*Pasteu. multocida* *B. bronchiseptica* *Mycoplasma spp*	Potentiated sulphas 1-gen cephalosporin Quinolone	-	Tracheal or bronchial wash fluid before antibiotic therapy
Pleural cavity (Cats)	*Pasteurella multocida* *Anaerobes*	Amoxicillin or ampicillin Clavulanic acid-potentiated amoxicillin Clindamycin	-	Pleural fluid sample before initiating antibiotic therapy

Table 3 : Antibacterial therapy for Urinary Tract Infections in Dogs and Cats

Type of infection	Pathogens involved	Antibiotic of choice	Alternative antibiotic	Culture/ susceptibility testing
Cystitis / Nephritis	*Escherichia coli* *Staph. intermedius* *Enterococcus spp* *Proteus spp* *Pseudomonas spp*	Amoxycillin or ampicillin Clavulanic acid-potentiated amoxycillin Potentiated sulphonamide 1-gen.cephalosporin	Quinolone Aminoglycoside Oxytetracycline or tetracycline	Not needed in patients with acute, uncomplicated cystitis. Urine sample, obtained by cystocentesis under conditions listed in text
Prostate (dogs only)	*Escherichia coli* *Staph. intermedius* *Proteus mirabilis* *Enterococcus spp*	Potentiated sulphonamide Quinolone	-	Prostatic fluid, collected by prostatic massage or ejaculate, urine by cystocentesis, before antibiotic therapy

Table 5 : Antibacterial therapy for Musculo-skeletal System infections in Dogs and Cats

Type of infection	Pathogens involved	Antibiotic of choice	Alternative antibiotic	Culture/ susceptibility testing
Septic arthritis (Dogs)	*Staph. spp* *Strepto. spp* *Escherichia coli* *Anaerobes*	1-gen cephalosporin Clavulanic acid-potentiated amoxycillin Doxycycline	Aminoglycoside Quinolone Penicillinase-resistant penicillin	Joint fluid before initiation of antibiotic therapy
Septic arthritis (Cats)	*Pasteurella spp* *Mycoplasma spp*	1-gen. cephalosporin Clavulanic acid-potentiated amoxycillin Doxycycline	-	Joint fluid before initiation of antibiotic therapy
Osteomyelitis (Dogs and Cats)	*Staph. spp* *Escherichia coli* *Proteus spp* *Enterococcus spp*	1-gen. cephalosporin Clavulanic acid-potentiated amoxycillin Clindamycin	Aminoglycoside Quinolone Penicillinase-resistant penicillins	Exudate or bone specimen or serial blood samples before antibiotic therapy
Discspondylitis in dogs and cats	*Staph. spp* *Strepto. spp* *Brucella canis*	1-gen. cephalosporin Clavulanic acid-potentiated amoxycillin	Quinolone Penicillinase-resistant penicillin	Serial blood samples before initiation of antibiotic therapy

Table 6 : Antibacterial therapy for Mastitis and Septicemia in Dogs and Cats

Type of infection	Pathogens involved	Antibiotic of choice	Alternative antibiotic	Culture/ susceptibility testing
Mastitis (dogs and cats)	*Escherichia coli* *Staph. spp* *Strepto. spp*	1-gen. cephalosporin Clavulanic acid-potentiated amoxycillin Potentiated sulphas	Quinolone (Contra-indicated in nursing females)	Sample of milk or aspirated fluid before initiation of antibiotic therapy
Septicaemia (dogs and cats)	*Staph. spp* *Escherichia coli* *Strepto. spp* *Klebsiella spp* *Salmonella spp* *Proteus spp* *Pseudomonas spp* *Enterococcus spp* *Anaerobes*	Aminoglycoside + ampicillin sodium Aminoglycoside + ampicillin-sulbactam Aminoglycoside, 1-gen. cephalosporin + clindamycin Quinolone + clindamycin Quinolone + ampicillin sodium + imipenem-cilastatin	-	Serial blood samples, three, collected one to two hours apart from peripheral veins before initiating antibiotic therapy



Table 7 : Suggested dosage regimens for selected orally administered antibiotics

Antibiotic	Species	Dose	Dose interval	Remark
Amoxicillin	Dog, Cat	10-22 mg/kg	8 hr	-
Clavulanic acid	Cat	62.5 mg	8-12 hr	-
Azithromycin	Dog	3.3 mg/kg	24 hr	A, B
	Cat	5 mg/kg	48 hr	-
Cefadroxil	Dog, Cat	22 mg/kg	8-12 hr	-
Cefixime	Dog	10 mg/kg	12 hr	-
Cephalexin	Dog, Cat	22 mg/kg	8 hr	B
Chloramphenicol	Dog	40-50 mg/kg	8 hr	A, B, C, D, F
	Cat	50 mg	12 hr	C, D, E
Ciprofloxacin	Dog, Cat	10 mg/kg	24 hr	-
Clindamycin	Dog, Cat	5-11 mg/kg	12 hr	A
Cloxacillin	Dog	22 mg/kg	12 hr	-
Difloxacin	Dog	20-40 mg/kg	8 hr	-
Doxycycline	Dog, Cat	5 mg/kg	12 hr	A, B, C
Enrofloxacin	Dog, Cat	5-20 mg/kg	24 hr	C, D, E
Erythromycin	Dog	10-20 mg/kg	8-12 hr	-
Lincomycin	Dog	15-25 mg/kg	24 hr	-
Metronidazole	Dog	10-20 mg/kg	8-12 hr	A
	Cat	10-20 mg/kg	24 hr	-
Orbifloxacin	Dog, Cat	2.75-5 mg/kg	24 hr	C, D, E
Ormetoprim-sulfa	Dog, Cat	27 mg/kg	24 hr day 1	D, E
		13.5 mg/kg	24 hr day 2+	-
Oxacillin	Dog, Cat	22-40 mg/kg	8 hr	-
Oxy/tetracycline	Dog, Cat	20 mg/kg	8-12 hr	A, B, C, D, E
Trimeth-sulfa	Dog, Cat	30 mg/kg	12 hr	D, E

A. Avoid or reduce dose in patients with severe liver failure
B. Administer with food if gastrointestinal upset occurs
C. Avoid in young animals
D. Avoid in breeding or pregnant animals
E. Avoid or reduce dose in patients with renal failure
F. Do not use for more than 14 days in cats

Table 8 : Suggested dosage regimens for selected parenteral antibiotics

Antibiotic	Species	Dose	Route	Dose interval	Remarks
Amikacin	Dog, Cat	5-10mg/kg	IV, IM, SC	8 hr	
Amoxicillin	Dog, Cat	10-22mg/kg	SC	8 hr	
Amoxicillin-clavulanic acid	Dog, Cat	12.5mg/kg	IM, SC	12 hr	
Ampicillin sodium	Dog, Cat	10-20 mg/kg	IV, IM, SC	8 hr	
Ampicillin-sulbactam	Dog Cat	12.5-25mg/kg 62.5mg	IV, IM, SC IV, IM, SC	8-12 hr 8-12 hr	
Cephalexin	Dog, Cat	10-25mg/kg	IM, SC	8-12 hr	
Cefazolin	Dog, Cat	20-25mg/kg	IV, IM	8 hr	
Cefotaxime	Dog, Cat	25-50mg/kg	IV, IM	8 hr	
Cefotetan	Dog	30mg/kg	IV	8 hr	
Cefoxitin	Dog, Cat	30mg/kg	IV	8 hr	
Cephalothin, Cephapirin	Dog, Cat	10-30mg/kg	IV, IM	6-8 hr	
Chloramphenicol Na succinate	Dog Cat	40-50mg/kg 50 mg	IV, IM, SC IV, IM, SC	8 hr 12 hr	c, d, e, f
Clindamycin	Dog Cat	5-11mg/kg 5-11mg/kg	IM, SC SC	12 hr 12 hr	e
Doxycycline	Dog, Cat	5mg/kg	IV	12 hr	c, e
Enrofloxacin	Dog, Cat	5-10mg/kg	IV, IM	12 hr	a, c, d
Gentamicin	Dog, Cat Dog, Cat	2-4mg/kg 6-8mg/kg	IV, IM, SC IV, IM, SC	8 hr 24 hr	a-b
Imipenem-cilastatin	Dog, Cat	2-5mg/kg	IV	6-8 hr	a
Ticarcillin+clavulanate	Dog	40-110mg/kg	IV, IM	6 hr	g
Tobramycin	Dog, Cat Dog, Cat	2mg/kg 10mg/kg	IV, IM, SC IV, IM, SC	8 hr 24 hr	a, b
Trimethoprim-sulfa	Dog, Cat	30mg/kg	IV	12 hr	a, d

a. Avoid or reduce dose in patients with renal failure
b. Therapeutic drug monitoring advised, particularly in young animals
c. Avoid in young animals
d. Avoid in breeding or pregnant animals
e. Avoid or reduce dose in patients with severe liver failure
f. Do not use for more than 14 days in cats
g. Used primarily for treatment of Pseudomonas spp. infections

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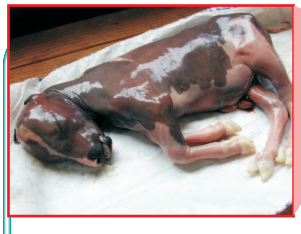

Brucellosis : A hurdle in Farm Livestock Development

Brucellosis, a disease of bacterial origin, manifests itself principally by reproductive failure in farm livestock (cattle, buffaloes, goats, sheep and pigs). This includes abortions, still births, births of unthrifty newborns, retention of placenta, metritis and subsequent infertility in females, whereas, orchitis, epididymitis and subsequent infertility/sterility in males.

Persistent and lifelong presence of infection in reproductive tract, mammary glands, joints and lymphnodes; and shedding of organisms in reproductive and mammary secretions is the characteristic of brucella infection.

Brucellosis in animals has also a zoonotic importance, as it produces a chronic, debilitating disease in human population. The major impact of brucellosis on economics of livestock production, so also its zoonotic potential, has prompted most of the countries in the World, including India, to initiate brucellosis control and eradication programmes in the respective countries.

Effect of timed artificial insemination Protocols in effectuating successful fertilization in Repeat Breeding Crossbred Cattle

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Introduction

Repeat breeding among cyclic crossbred cows prolongs service period, inter-calving period and results in huge economic losses to dairy farmers in India. Although the factors responsible for repeat breeding are multiple viz. anatomical, physiological, nutritional, managemental and infectious, a higher incidence of repeat breeding has been attributed in Kerala to that from a combination of ovulatory disturbances and reproductive tract infections (Ibraheem Kutty and Ramchandran 2003).

Synchronization of ovulation in cows has been extensively investigated and many protocols have been developed for timed artificial insemination (Orkun Demiral et al 2006). Gonadotropin-Releasing Hormone (GnRH) together with Prostaglandin F2α (PGF2$\boldsymbol{\alpha}$) are used successfully to synchronize estrus and ovulation among dairy and beef cows (Geary et al 2001). Successful ovulation is a prerequisite for fertility and to the success of all timed AI protocols to improve fertility among repeat breeders. As a comparative study among various timed AI protocols namely Ovsynch, Cosynch and Selectsynch to elicit successful fertilization in cyclic repeat breeding crossbred cows in field condition had not been done in Thiruvananthapuram. Krishi Vigyan Kendra, Thiruvananthapuram, Kerala in the year 2009-2010 conducted an on Farm Testing to determine comparatively efficient timed AI protocol among the three known procedures.

Materials and Methods

Cyclic crossbred cows that returned for insemination for the third consecutive time in a span of six months were identified as repeat breeders to be included in the study. Sixty cows from animals so identified were selected and distributed into three groups having twenty animals each as uniformly as possible with regard to age, breed, parity and body weight. All animals included in the study were provided with vitamin and mineral supplements and uterus of all animals were douched with 15 ml Povidone Iodine fifteen days prior to execution of protocols. Animals in Group I were subjected to Ovsynch protocol (Pursley et al., 1994) and were injected with 0.1mg intramuscular injection of Buserlin (Inj. Receptal) on starting day of trial, 0.5mg intramuscular injection of Cloprostenol (Inj. Cyclix) seven days later, second dose of 0.1mg intramuscular injection of Buserlin 48 hours after Cloprostenol injection and followed with timed AI 24 hours after second Buserlin injection. Animals in Group II were subjected to Cosynch protocol (Geary et al 2001) and were injected with 0.1mg intramuscular injection of Buserlin (Inj. Receptal) on the starting day of trial, 0.5mg intramuscular injection of Cloprostenol (Inj. Cyclix) seven days later, another 0.1mg Intramuscular injection of

Buserlin 48 hours after Cloprostenol injection followed with AI on the same day immediately after second Buserlin injection. Animals in Group III were subjected to Selectsynch protocol (Stevenson et al., 2000) and were injected with 0.1mg Intramuscular injection of Buserlin (Inj. Receptal) on the day of start, 0.5mg Intramuscular injection of Cloprostenol (Inj. Cyclix) seven days later and AI 72 hours after Cloprostenol injection. Days of handling and cost of protocol per cow were recorded for all the three fixed time AI protocols. All the animals included in the study were checked for pregnancy via per rectal examination on 75th day from AI.

Results and Discussion

Twelve out of the twenty animals that underwent timed AI via Ovsynch protocol when checked for pregnancy via per rectal examination were diagnosed to be pregnant. Of the twenty animals that underwent timed AI via Cosynch protocol, only nine were diagnosed pregnant while only five out of the twenty animals that underwent timed AI via Selectsynch protocol were diagnosed to be pregnant on per rectal examination at 75th day from AI. Results similar to this study were obtained earlier by Geary and Whittier, (1998) who had reported that although Cosynch protocol requires one less handling of cows it resulted only in 49% successful pregnancy compared to Ovsynch protocol that yielded 57% success in their study on timed AI protocols in beef cows. Days of handling, pregnancy and cost of protocol per cow obtained in present study were as tabulated in Table 1 and described in Fig 1 and 2. Out of the six heifers included in the three treatment groups for the present study, four heifers in Group II that underwent timed AI with Cosynch protocol became pregnant compared to two heifers that received Ovsynch protocol and one heifer that became pregnant after undergoing Selectsynch protocol. This finding was in accordance to Orkun Demiral et al., (2006) who found Cosynch protocol to be more effective in heifers than multiparous cows. Rs 1100 spent per cattle for timed AI through Ovsynch protocol ensured a 60% fertility rate in Group I compared to 45% fertility obtained from Cosynch protocol at a cost of Rs 1000 per cattle in Group II and 25% fertility provided from Selectsynch protocol at a cost of Rs 800 per cattle in Group III.

Fig 1. Fertility rates obtained from the three timed AI protocols in Repeat Breeders

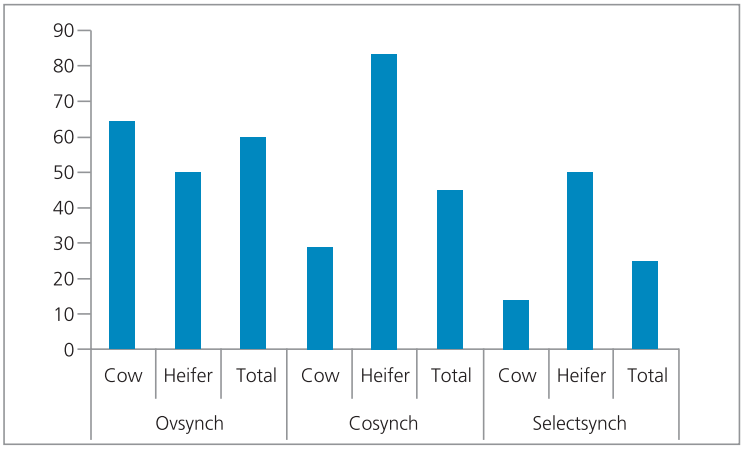

Fig 2. Cost/Cow for the three timed AI protocols in Repeat Breeders

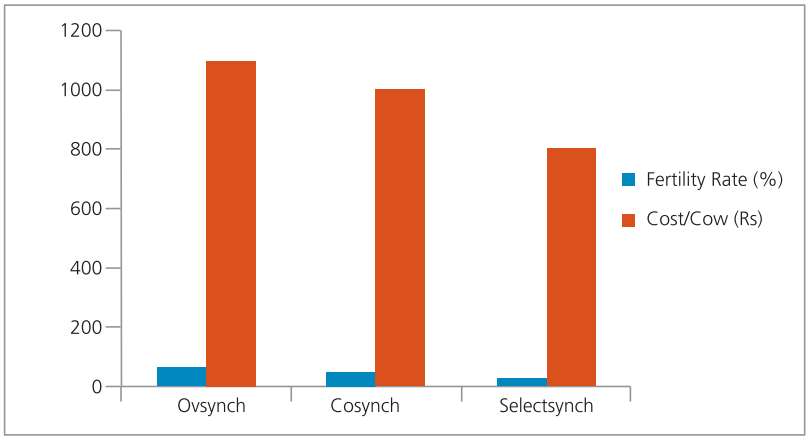

Table 1. Comparative Performance of the three timed AI protocols in Repeat Breeders

Day of Handling	Protocol I	Group I: Ovsynch			Cost of Protocol/Cow
		Cow	Heifer	Total	
		14	**6**	**20**	
0	GnRH	do	do	do	300
7	PGF 2α	do	do	do	350
9	GnRH	do	do	do	300
10	AI	do	do	do	150
75	Pregnancy Diagnosis	**10**	**2**	**12**	
			Total Cost/ Cow		**1100**
Days of Handling	**Protocol II**	**Group II: Ovsynch**			**Cost of Protocol/Cow**
		Cow	Heifer	Total	
		14	**6**	**20**	
0	GnRH	do	do	do	300
7	PGF 2α	do	do	do	350
9	GnRH & AI	do	do	do	400
75	Pregnancy Diagnosis	**5**	**4**	**9**	
			Total Cost/ Cow		1050

Days of Handling	Protocol III	Group III: Ovsynch			Cost of Protocol/Cow
		Cow	Heifer	Total	
		14	**6**	**20**	
0	GnRH	do	do	do	300
7	PGF 2α	do	do	do	350
9	AI	do	do	do	150
75	Pregnancy Diagnosis	**4**	**1**	**5**	
			Total Cost/ Cow		800

Acknowledgement

Funds provided by the Zonal Project Directorate, ICAR ToT, Zone-VIII to fund the On Farm testing is humbly acknowledged.

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Techniques of Fracture management in Small Ruminants

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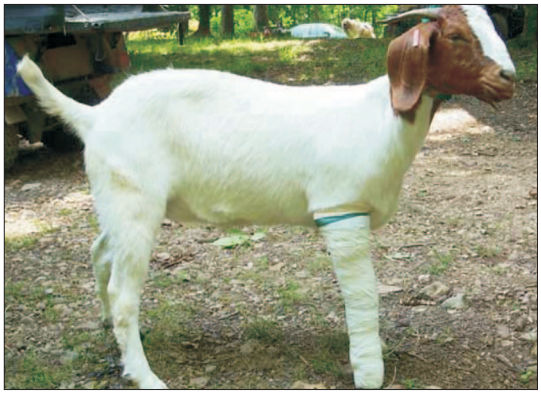


Introduction

Fractures in small ruminants occur frequently and is a concern of economic importance to the owner. The practice of keeping them as pets in households rather than in sheds or in confinements have increased the chance of encountering accidents like fall from terrace, limbs being trapped in drains, being hit by vehicles, dog bites etc. leading to fracture. Unlike in large ruminants, Small ruminant fracture management can be successfully achieved owing to their light weight, they tolerate external coaptation relatively well and can ambulate in three limbs well in order to spare the injured limb. Fracture in small ruminants can be repaired successfully with regional anaesthetic techniques which they can tolerate very well. Owing to the high economical value of these animals which are kept for ceremonial purposes or for sacrifices, the owner can insist fracture repair. The constraint of cost effectiveness can be balanced by carefully selecting the appropriate technique according to the weight and limb involved. Many economical skeletal fixation techniques have been evolved which can well fit into the budget of the owner. Knowledge of simple techniques can enhance healing in cases of bone loss and management of infection.

Fracture site stabilisation

Fracture repair is usually elective, hence initial support and care of the fracture is necessary to prevent further trauma and complications like communication of fracture site to the exterior through wound or sinus and chances of infection. Initial support to splint the bone not only limits the movements, but also supports the soft tissues from untoward consequences. Limb splinting reduces edema and pain, preserves neurovascular supply to the limb till the day of operation and helps to transport the animal safely. Fracture to limbs caudal to mid radius ulna/tibia can safely be stabilised by splints or full limb casts. Wood, pliable bamboo or split poly vinyl chloride pipes positioned on the caudal and lateral aspects of the limbs and supported firmly with tapes or bandages can serve the purpose. Fracture proximal to mid radius ulna or tibia should not be stabilised using splits since the fulcrum effect created due to this can augment soft tissue injury. Careful assessment of the viability of the structures caudal to fracture site should be assessed prior to a decision regarding surgery as devitalised tissue hamper healing and is a subject to be considered for amputation.

Signs of fracture

The history of improper limb carriage or non weight bearing, associated with a traumatic event or as a sudden finding can implicate a possibility of fractured limb. Pain and acute swelling may hinder palpation of the site, though most animals are stoic without much pain and shrieks. Discontinuity, crepitus and abnormal alignment of the bone with tenderness can be appreciated. Radiography should be undertaken with orthogonal views (anterio-posterior and lateral), unless in specific situations which elaborate the nature of fracture, enough to plan and execute fracture repair. Good technique and proper positioning is imperative to detect even the subtle abnormalities like hair line, the involvement of joint or the level of comminutuion. Sedation with butorphanol (0.05 mg/kg IV), Xylazine (0.05-0.1 mg/kg im) or midazolam (0.2 mg /kg iv) can give adequate muscle relaxation, if required for taking radiographs.

Considerations for fracture management

The position of fracture which is either open or closed, the level of soft tissue damage and the viability of limb distal to fracture, suitability of implants experience of the surgeon and cost effectiveness of techniques are to be considered and the technique should be planned.

Anaesthesia for surgery

Regional analgesic techniques are better suited to small ruminant fracture repair owing to the safety and simplicity of the technique and lesser peri- operative complications. Intravenous regional analgesia (IVRA) technique is popular in this regard . For forelimb surgeries, a rubber tourniquet approximately 1-1.5 cm wide and 10-15 cm length can be used. The forelimb to be operated is restrained with the same side lateral recumbancy, and the bandage is applied cranial to the elbow joint, the limb cau dal to this is aseptically prepared for surgery. The cephalic vein is located on the medial middle third of the forearm, 3-4 ml 2% lidocaine is injected into the vein. For hind limb surgery, the limb considered is to be restrained up, opposite to the site of recumbancy, the bandage is applied above the hock joint and limb caudal to this site is aseptically prepared. The recurrent tarsal vein is located at the groove in front of the gastrocnemius tendon and 3-4 ml 2% xylocaine is injected into this. The analgesia will remain for 30-45 min after which the tourniquet can be released. For surgery involving the femur or hind limb, lumbar epidural /spinal analgesia with lidocaine (0.2-0.4 mg/kg bwt) or Xylazine-ketamine (0.05 mg/kg and 2.5 mg/kg, 0.75 ml each) is suitable. General anaesthesia or balanced anaesthesia is often employed for surgery involving humerus or femur when the technique is time consuming.

Limb casting

The casting material according to the availability and economic concerns can vary. Plaster of Paris (POP) cast is easily available and economical. POP casts can be made locally or readymade material can be purchased. PMMA (Poly Methyl Methacrylate) or fibre glass is other choice. Fibre glass casts have advantages of strength and stiffness, they do not weaken by moisture and stress. Adequate cotton padding or foam resin to cover bony prominence, put in place with the help of bandages or stockinette precedes cast application. POP cast is adequate to manage closed, un displaced fractures of caudal radius

ulna, metacarpals or metatarsals without comminutuion or sharp ends. This can be put in place for 15 days at a stretch and evaluate the strength, and reapply the cast if required. Bamboo splints or PVC strips can reinforce the strength of the cast when incorporated with the initial padding. Care must be taken that the cast is applied not too tight to obstruct the venous return which can lead to swelling of the toes.

The casts can be applied as either full limb casts or half limb casts according to the bone to be immobilised. Half limb casts can be used to immobilise closed fractures of metacarpal or metatarsal bone, undisplaced physeal fractures and fractures involving phalanges. Cast application ensures the immobilisation of one limb above and below the bone fractured. Comminuted fractures and fractures involving the articular surface require rigid fixation techniques where cast application is uncalled for. Full limb casts can be applied for closed transverse fracture of distal radius ulna or proximal metacarpal or metatarsals. The time for fracture healing under casts depends upon the stability of the cast, age of the animal, presence or absence of infection and fracture stability. Clinical union can be appreciated by a bridging callus covering the fracture site and ambulation of the fractured limb. An average 20 -30 days time is sufficient for the retention of cast. Cast complications like sudden onset or pain, swelling or sores alerts cast removal.

Thomas splint

Thomas splint can be useful for fractures at proximal radius ulna or tibia, caudal to humerus or femur. The round frame and the hoop of the Thomas Splint helps additional anchorage and weight transfer into the frame from the body, thus sparing the fracture site. The complications involve inadequate fixation of the frame with the limb leading to rotation of the frame and displacement of the fracture fragments. Adequate space should be provided while the initial frame built in order to incorporate cotton padding. The hoop of the frame should support the axilla or inguinal area for support with body wall and weight transfer into the frame. The animal may need to accustom with this before proper weight bearing.

Hanging limb pin cast

A hanging limb pin cast refers to the placement of transcortical pins through the proximal fracture fragment and incorporating the pins into a cast. The cast begins proximal to the pin application, incorporating the transcortical pins and the entire limb caudal to it including the foot. The forces acting on the bone spares the fracture site, gets transmitted through the pins and the cast into the ground. This can be helpful to treat comminuted metacarpal or metatarsal fractures by biological osteosynthesis since it causes minimal fracture site disruption. This technique is cheaper and effective once the size and placement of the pins are appropriate. Care must be taken to spare the joints, articular surfaces and growth plate in young animals.

The diameter of the pins should not exceed 20% of the diameter of the concerned bone; the stoutest pin should be placed most cranially since it takes away majority of the forces acting on the bone. Pins should spare at least six pin diameter space from the fracture ends and the individual pins placed at four pin diameter spacing. Align the pins at a angle of 300 to provide rotational stability of the construct. The cast material should be applied closer to the skin

after bandaging. The problem of poor access to wound at the area is a disadvantage, the windows if left for wound care should not weaken the strength of the cast. Look for window oedema which can be managed by bandaging the wound at window and regular check for swelling and infection.

Transfixation pins and cast

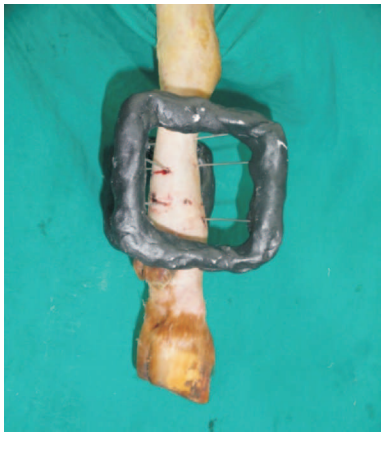
Transcortical pins through both the proximal and distal fracture fragments and casting with either POP or fibreglass can be achieved in closed fractures of midshaft radius ulna or tibia in larger ewes and goats. Here the cast will cover the bone conserved proximal and distal to the trasfixation pins. The amount of casting material is less and the animal can ambulate in its legs after its application.

External skeletal fixator

External skeletal fixation requires at least two transcortical pins placed proximal and distal to the fracture site and connected through connecting bars, fibre glass cast, epoxy putty, or PMMA (Fig.1and Fig.3). External skeletal fixation is versatile, easily applied, augments biological fracture repair and wound care. Pin size can be selected according to the size of the animal and bone involved. The fracture forces are shared equally by the individual pins. Fibreglass or epoxy putty are more economic, easy to apply and are readily available in the market. The construct can be eventually disassembled to increase axial weight bearing. This technique is well suited to manage compound diaphyseal fractures involving radius ulna, tibia, metacarpal or metatarsals.

Intramedullary pins and cerclage wires

Open fracture management of small ruminants allows precise anatomic reduction and more stable fixation. This is also relatively simple technique to manage fracture femur, tibia and humerus. Simple Intramedullary pin should fit nearly 70% of the intramedullary diameter. Normograde pin placement is recommended for tibial fracture since retrograde pin placement can injure the stifle joint or cruciate ligaments. Cerclage wiring has to be done in long oblique fractures, (fracture line exceeding twice the



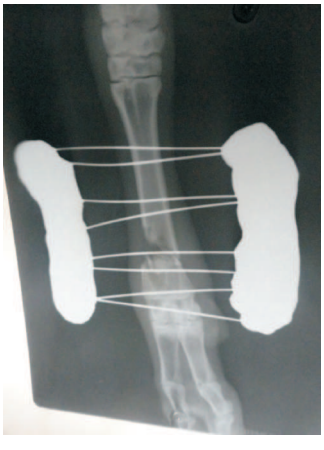


Fig.1: External skeletal fixator made up of Epoxy for metacarpal fracture repair.

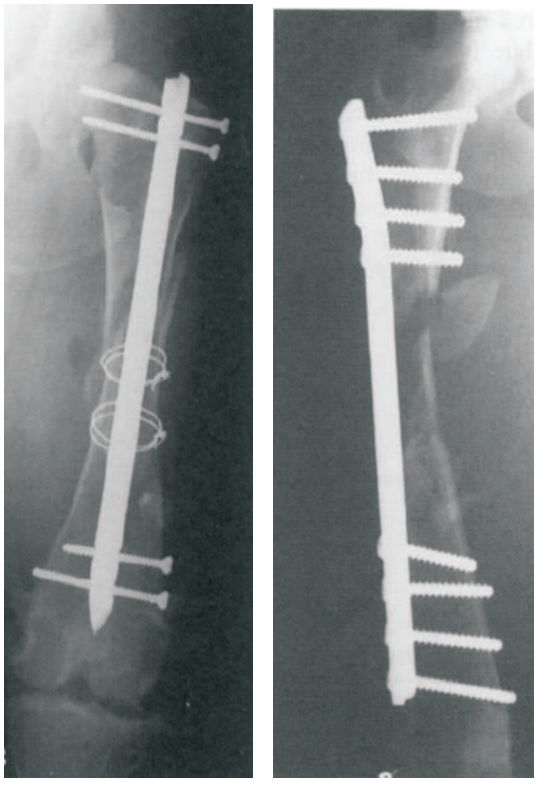


Fig.2. Radiographs of femur fracture repaired with Intramedullary interlocking nails and Bone plating

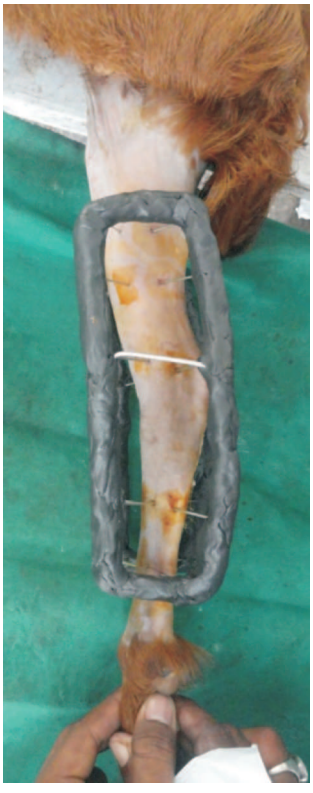


Fig.3. Management of hock joint dislocation with External skeletal fixator (Epoxy).

diameter of the bone) which offers some rotational stability to the construct. Unless there is complication of pin migration or osteomyelitis, the pin wire construct does not require surgical removal.

Bone plating

The advancements in orthopaedic research and due to the availability of instrumentation for plating have facilitated long bone fracture repair in small ruminants as well. Bone plating has most of the biomechanical advantages to facilitate fracture healing like rigid fixation, neutralise axial and rotational forces acting on the bone and early healing. The limitations due to disruption of extra osseous blood supply and subsequent osteopenia are very well dealt with newer implants like point contact dynamic compression plates, locking plates and percutaneous fixators (fig.2). Single or double plating techniques are used according to demand of fracture morphology and technique of the surgeon. Tibia is particularly suitable to plating because of the ease of approach to the bone and moderate extraosseous blood supply since two third of the bone is covered by the muscle when compared to the scanty muscle cover of metacarpal or metatarsals.

Intramedullary interlocking nails

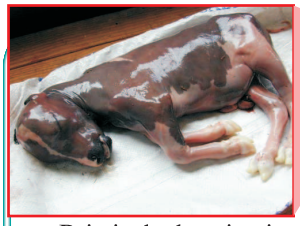
Locking Intramedullary implants have the advantage to facilitate biological fracture fixation with least disturbance of the fracture site (fig.2). This technique offers rotational stability when compared to simple Intramedullary implants and the locking bolts of screws bypass the fracture site off the axial forces to facilitate healing. Post operative dynamization has the added advantage of augmenting fracture repair by facilitating controlled axial loading. Intramedullary interlocking nailing can be opted for comminuted fractures of humerus, femur and tibia.

Complications of fracture repair

Post operative immobilisation/ limited ambulation is challenging in small ruminants. Infection, delayed union, malunion, recurrence of fracture, implant migration etc. are frequently encountered in these species. Adherence to proper aseptic protocol, minimally invasive technique and least surgical trauma and preserving maximum extraosseous blood supply to the fracture site can minimise such complications.

Conclusion

Fracture repair in small ruminants can pay very well to the efforts of the surgeon if conducted with appropriate implants and proper technique. Commonly available and economical implants and awareness about the techniques can render fracture repair in small ruminants a success and can prevent economic losses to the owner. The common and effective fracture fixation techniques and anaesthetic management are elaborated here.



Brucellosis : A hurdle in Farm Livestock Development

Historical

- Britain had maintained a military base on the Island of Malta in Mediterranean region between 18th/19th centuries. During this period, several British physicians described a chronic illness among the troops, characterized by debility with complications of rheumatism. The disease was described as "Malta Fever" or "Mediterranean Fever".
- Captain David Bruce, a British Physician, was sent to Malta (1884) to investigate the illness. He isolated the causative organism from the spleens of four fatal cases (1887) and named it as *Micrococus melitensis* (melitensis : Latin - Honey).
- "A Mediterranean Fever Commission" was formed (1905), with David Bruce as its chairman. Dr. Themistoles Zammit, a Maltase Physician, was one of the members of the Commission.
- Those days, British soldiers were given raw goat milk for many illnesses. Dr. Zammit demonstrated that Maltase goats - often with non-clinical signs of illness - carried the organisms and served as a source of infection through consumption of non-pasteurized goat milk by military personnel. Thus, the role of goats in Mediterranean Fever was established.

Canine Transmissible Veneral Tumor

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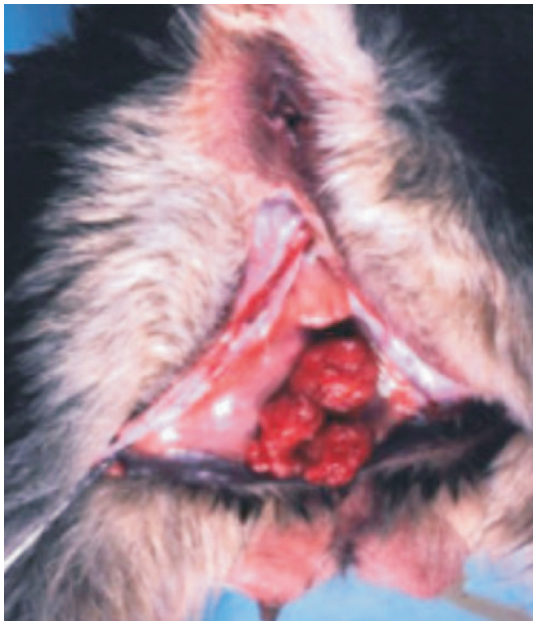

Introduction :

Canine transmissible veneral tumor (CTVT), also known as veneral granuloma, primarily occurs on the external genitalia of the male and female dogs. As coitus is the main pathway of transmission, it mainly occurs in young , sexually mature animals.

History/Observations

A 2 year old female dog (Japanese Spitz cross bred) weighing 14 kg was presented with the complaint of a hard, pink, spherical mass protruding from vulva since three months. It was slow growing in nature, looked like cauliflower, with dirty discharge from vagina. Physical examination revealed hard tumorous pedunculated mass having smooth and glossy surface protruding from the vulva. It had difficulty in urination and defecation. The temperature, pulse rate and respiration rate were normal.

Diagnosis

From the history and physical examination, the case was tentatively diagnosed as vaginal tumor and it was decided to go for surgical excision.

Surgical treatment

The animal was anesthetized with xylazine + ketamine (2.5 ml) and positioned in lateral recumbency while the table was tilted to elevate the perineum. The site was prepared by shaving the area with sterilized blade and cleaning with povidone solution and the mass was surgically removed.

Procedure

Purse string suture was placed around the anus and the vagina was exposed. Tumors were carefully differentiated and excised. Hemorrhage was controlled with ligatures and cautery ($Kmno_4$). Mucosa was closed with 3-0 chromic catgut in interrupted manner. A simple interrupted suture was also placed to close muscle and sub cutaneous tissue. Then non-absorbable suture in interrupted fashion was placed on to close the skin.

Post-operatively, the animal was provided with Ringer's lactate solution @200-300 ml iv. Course of antibiotic (ampicillin + cloxacillin;

magapen) was given for 5 days @ 10mg/kg. Anti-neoplastic drug, Vincristine @ 0.025mg/kg body weight iv with 20 ml normal saline once a week was administered for four weeks. Douching of vagina with 2% povidone iodine was done. Supportive therapy included B-complex syrup @ 10ml orally for 7 days.

Discussion

CTVT is a transmissible, usually non-malignant growth, wherein surgery is effective for localized lesion; by episiotomy. Local or extensive tumors respond well to Vincristine (0.5 ml/week) by I/V administration. Vincristine is a plant alkaloid and in some cases peripheral neuropathy and constipation may be seen as side effects. Surgical removal is the only treatment of choice. Chemotherapy is rarely applied in animals. While removing the tumor, the tissues around the tumor must be removed as much as possible to reduce its recurrence.

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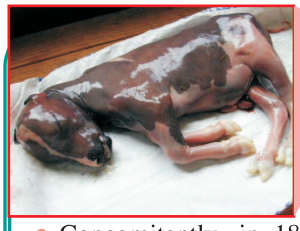

Brucellosis : A hurdle in Farm Livestock Development

Historical - contd.

- Concomitantly, in 1897, a Danish Veterinarian, Dr. Bernard Bang studied a disease in cattle characterized by abortions and isolated an organism. He named it as '*Bactillus abortus*' and the disease was named as 'Contagious abortion' or 'Bang's Disease'.
- In 1918, Alice Evans, an American Microbiologist, showed that *Bactillus abortus* also causes Malta Fever Syndrome in humans and the organism was closely related morphologically and biologically to Bruce's *Micrococcus melitensis*. Unfortunately, Alice Evans, in her efforts to develop improved techniques to recover the organisms and diagnosing the disease, got her self infected in 1925.
- A Veterinary scientist at the Hooper Foundation, San Francisco, Karl Meyer, proposed to group these organisms under the Genus "*Brucella*", in honor of Dr. Bruce, to settle the nomenclature issue.
- The United States Department of Agriculture's Bureo of Animal Industry, between 1920 - 1940, conducted research on effective vaccine development. Dr. John M Buck of the Bureo, maintained a group of Brucellosis cultures on his desk at room temperature for evaluating them for immunogenecity and stability. He observed that the 19th culture was significantly less pathogenic and sufficiently immunogenic. This culture was later developed as a vaccine strain, first licenced in 1941, as a **live attenuated strain 19 vaccine.**
- The same strain 19 live attenuated vaccine was successfully employed during subsequent decades to eliminate brucellosis from the United States' cattle population through calf-hood vaccination programme.

Ultrasonographic diagnosis of Fetal Mummification in a Bitch

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Introduction

Fetal mummification is a common problem in polytocous and rare in monotocous animals (Perumal and Srivastava, 2011). In canine, it is uncommon and sporadic in nature (Roberts, 2004). Death of fetus and fetal fluid are reabsorbed by the uterus causing dehydration of fetal tissue and associated membranes with persistence of the corpus luteum so that the products of conception are retained within the uterus is called fetal mummification (Noakes, 1986). Death of the fetus after ossification of fetal bones generally leads to fetal mummification if there is no bacterial infection concurrent with or causing death of the fetus (Robinson et al, 2003). The uterus contracts on the fetus, placental fluids get absorbed and fetal membranes become shriveled and dried (Roberts, 2004). In polytocous species, if mummification occurs in some embryos, it does not interfere with continuation of pregnancy of viable fetus; instead mummified fetus may be delivered with live fetus at the time of normal parturition (Arthur et al., 2001).

History and Clinical Observations:

A 2-year-old German Shepherd female with a history of mating 55 days before was brought to TVCC, Apollo College of Veterinary Medicine. The owner reported anorexia, fever, and vomiting, abnormal vaginal discharge since 4 days. The bitch did not show any clinical manifestations of approaching whelping. The rectal temperature was 102.9°F and the animal was weak and lethargic. The perineum of the animal was soiled with vaginal discharge. The ultrasonographic examination was carried out to know about pregnancy status of the bitch.

Result:

Ultrasonographic examination was performed through trans-abdominal approach. Ultrasonographic evaluation on 55th day of pregnancy reveled presence of five fetal skeletons. All fetuses were small and without heart beats. Fetal fluid was absorbed and fetal membranes adhered to the fetus. So, the present clinical case was diagnosed as fetal mummification.

Discussion:

The fetal mummification is common problem in polytocous and rare in montocous species (Perumal and Srivastava, 2011). In this study, all the fetuses were mummified and surrounded by dark capsules with wet surface. The main reason for the lack of expulsion of dead mummified fetus in present case may be primary uterine inertia which is common in canine species (Romagnoly et al., 2004). Walett and Linde (1994) also reported uterine inertia as main cause of dystocia in bitches. In elderly female

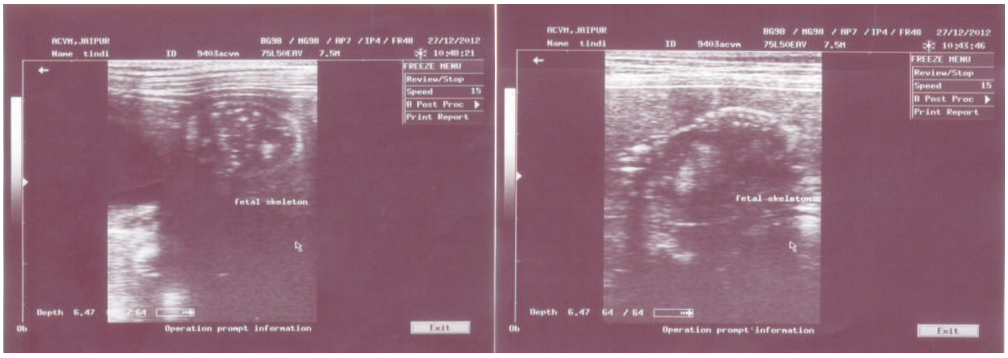

Fig 1 and 2: Shows the presence of fetal skeleton without fetal fluid

dogs, due to poor abdominal muscle tone, there is difficulty in producing uterine contraction in second stage of labor (Jackson, 2004b). This primary uterine atony might have been the cause for the maternal dystocia (Vorwald et al., 2012). It was also believed that fetuses fail to produce sufficient ACTH and cortisol to initiate the birth process (Johnston et al., 2001a; Linde-Forsberg, 2010).

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Successful management of Theleriosis in a Crossbred Cow

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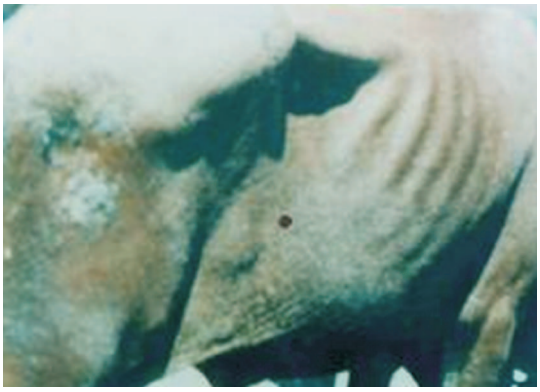

Introduction

Theleriosis are those tick borne protozoan diseases caused by Theleria spp in cattle, sheep and goats as well as in wild and captive ungulates .The diseases are characterized by fever and lympho proliferative disorders, which may be associated with leucopenia and or anemia (Radostits, 2000). The Theleria undergoes sequential development in leucocytes, erythrocytes of the mammalian host and causes an acute , often fatal disease (Singh et al ,2012). In India, the disease has assumed paramount importance with the intensification of crossbreeding programmes, aimed at enhancing milk production (Benewal et al,1997).The present case report deals with the successful management of theleriosis in a crossbred cow.

Case history and Observations

A three year old cross bred cow was presented to Campus Veterinary Hospital, College of Veterinary Science , Rajendranagar with the history of anorexia, dullness, depression and sudden drop in milk yield. Clinical examination of the cow revealed rise in body temperature(103.5°F), pallor mucous membranes, reduced ruminal motility (1 per 3 minutes) and salivation. The physical examination revealed severe tick infestation and enlarged superficial lymph nodes. Blood was collected for complete blood count and blood smear was prepared, stained with Giemsa stain.

Treatment

The affected animal was treated with the single dose of Buparvoquone (Inj. Zubion @2.5 mg/kg b.wt deep intramuscularly, Inj. Ferritas (Iron sorbitol citric acid complex) @10 ml intramuscularly at 3 days interval for 2 weeks, Inj. Tribivet (Vit B1,B6,B12) 10 ml intramuscularly for one week, Inj.Anistamin (Chlorpheneramine maleate) @ 10 ml intramuscularly for 3 days and Deltamethrin (2ml in one litre of water) was used externally. There was marked improvement in the condition within 3 days of treatment and blood smear was found negative .

Results and Discussion:

Clinical findings such as rise in body temperature, enlarged superficial lymph nodes and anaemia are in agreement with Radostits, (2000).However, severe anaemia, jaundice, drop in milk production and abortion in

advanced pregnancy was seen in buffaloes suffering with Theleriosis (Dayaram and Pavan,2012). The microscopic examination of thin blood smears stained with Giemsa stain, revealed round to oval piroplasms in the erythrocyte resembling theleria organisms (Soulsby,1982). Hematological examination revealed low haemoglobin (5.8 gm%) and RBC count(3.18 millions /cumm), PCV 16% and Total Leucocyte Count (8700X10³/cumm) indicating anaemia. Marked anaemia of varying intensity with anisocytosis, poikilocytosis and polychromacia with no significant change in monocyte and eosinophil count has been reported (Singh et al 2012).On the basis of history, physical, clinical examinations and finding the schizonts in the blood smear, the case was diagnosed as theleriosis. Single dose therapy with Buparvoquone was considered to be most effective for controlling bovine theleriosis (Singh et al,2012., Sumathi and Veena 2012). However, combination of Buparvoquone and Oxytetracycline along with supportive therapy was found to be most effective in treatment of theleriosis (Jayanna et al 2012).

Conclusion

A three year old crossbred cow was presented with the history of anorexia, dullness, depression and decreased milk yield. Clinical examination of the animal revealed high temperature, pale visible mucous membranes and swelling of superficial lymph nodes. Theleriosis was microscopically diagnosed by determining the presence of piroplasms in erythrocytes in Giemsa stained blood smear. Treatment with Buparvoquone along with supportive therapy was effective leading to the improvement in the clinical condition of the animal.

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Auto-haemo therapy in clinical cases of Papilloma

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Introduction:

Auto haemotherapy is a simple technique where patient's own blood is injected parenterally (sub cutaneous and intramuscular route).This method is preferable as it is simple and economical compared to other therapeutic procedures. The beneficial effect of autohaemotherapy is due to its immune stimulatory effects. There is an increased production of both humoral and cell mediated immunity following auto haemotherapy. In the present study a case of buffalo, affected with warts on teats and another case of dog affected with warts on four different sites of the body are described.

Case presentation:

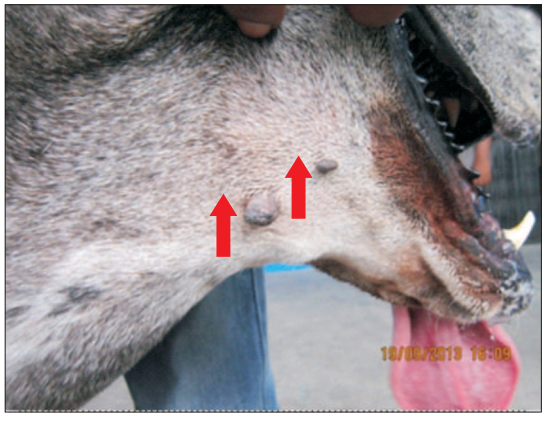
Case 1: She buffalo was presented to the medical ward, College of Veterinary Science, Proddatur with the history of soreness of teats and difficult milking. Examination of teats revealed small, sessile papillomatous growths on the hind teats.

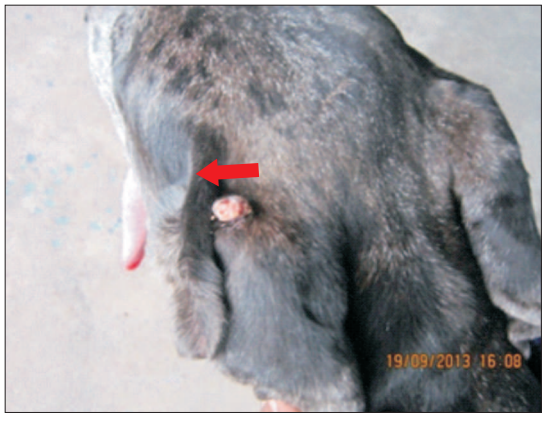
Casell:

A Grate Dane pup exhibited warts at the left hind foot (8mm), right thorax (8mm), left ear (1mm) and right mandible region (10mm).

Clinical management:

The animals were subjected to autohamotherapy . About 10ml of Avilin was given to the she buffalo;1ml of Avilin was given to Grate Dane dog in order to stabilize the animals and avoid untoward allergic reactions. After a period of 15 minutes, 20ml of blood was taken from the she buffalo and administered 10ml I/M and other 10 ml S/C at the neck region. Similar procedure was adopted in the dog also but the quantity blood was 5ml(2.5ml S/C&2.5ml I/M).The treatment was repeated at weekly intervals.





Results & Discussion:

The animals recovered after 2-4 injections .She buffalo received four(4) injections while the dog required only two(2) injections for clinical recovery.

Autohaemotherapy is a very useful and simple method wherein patient's own blood is given parenterally. This method is preferable as it is simple and economical compared to the other clinical methods. The action or effectiveness of this may be due to its immune enhancing effects(Athulya et al;2011).Enhanced production of both humoral and cell mediated immunity was reported after autohaemotherapy by Chetan Kumar (2011) on a five and half year old cow.

Autohaemotherapy might be an alternative method in the field (Biricik et al.,2003). Autohaemotherapy was successfully employed on five buffaloe calves, 3 female calves and buffaloe heifer aged eight(8) months to three(3) years in West Bengal by Jana & Jana (2009).

It may be recommended as a good therapeutic tool as it is very simple, safe and economical method compared to other conventional procedures. This is a good alternative method in the field.

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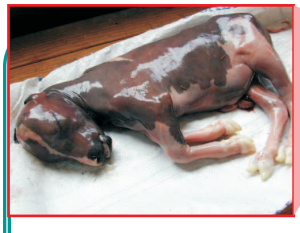

Brucellosis : A hurdle in Farm Livestock Development

The Organisms

- Brucella group of organisms are cocco-bacilli or short rods which are arranged singly and less frequently in pairs and small groups. They are non-motile, gram-negative and do not show bipolar staining. They do not form spores, neither they produce true capsules or flagella.
- Brucella organisms belong to α-2 Proteobacteriacea family. They are not acid fast, but resist decolouration by weak acid, thereby staining red with the Stamp's modification of Ziehl - Neelsen staining method, which is usually used for the microscopic diagnosis of Brucellosis from the smears taken from placenta, dead fetus , uterine discharge etc.
- Brucella are facultative, intracellular organisms and in-spite of having more than 94% similarity amongst themselves, have different host preferences. Therefore, Brucella spp. are capable of causing disease in a variety of animal species, including human beings.

Prolene Mesh Perineal Hernioplasty

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Introduction:

Perineal hernia is characterized by disruption of the pelvic diaphragm and herniation of the abdominal or pelvic organs through the ischiorectal fossa in the perineal sac, especially in middle-aged or aged intact male dogs (Stoll et. al. 2002, Head et. al., 2002, Gilley et. al., 2003, Bellenger and Canfield, 2003, Bongartz, et. al. 2005). Approximately 59% of the perineal hernias are unilateral while 41% are bilateral (Bellenger and Canfield, 2003).

Perineal hernia may be associated with sacculation, dilatation, deviation and diverticulation of rectum, retroflexion of urinary bladder or uretral obstruction (Brissot et. al. 2004, Vnuck et. al. 2006). The recurrence of the hernia, tenesmus and rectal prolapse are not rare with standard herniorraphy (Brissot et. al. 2004, Gilley et. al., 2003). Castration is recommended due to the effects of male hormone (Head et. al., 2002 and Niebauer et. al., 2005) on the prostate gland and perianal musculature. Adequate analgesia protocol is also needed for prevention of straining and concomitant recurrence.

Case report:

A Dalmatian dog aged 9.5 years was presented for straining in defecation and swelling at the right perineal region. The clinical examination revealed the ruptured perineal diaphragm and rectal diverticulum. The case was diagnosed as perineal hernia.

Treatment:

The dog was premedicated with Atropine sulphate @ 0.04mg/kg i/m and Xylazine HCL @ 1.5mg/kg i/m. The general anaesthesia was maintained by Ketamine administration @ 8-10 mg/kg intravenously. The dog was placed on the sternal recumbency. The operation table was tilted 10 to 15 degrees in head-down position. Anal sacs of the dogs were evacuated. A curvilinear skin incision was made 1 to 2 cm lateral to the anus, beginning at the base of the tail and extending 1 to 2 cm ventral to the ischium. The rectal diverticulum determined by assistant surgeon inserted his index finger through the rectum. The size of the rectal diverticulum was reduced by placing a few

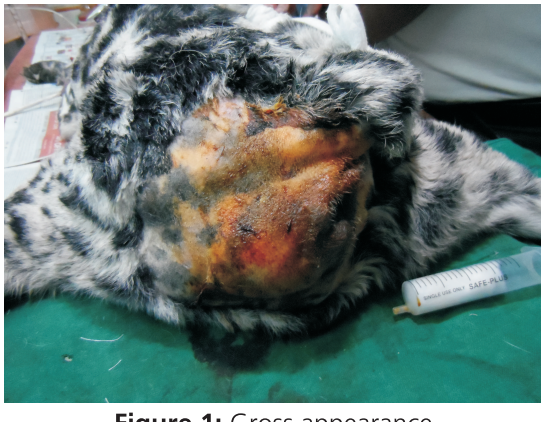

Figure 1: Gross appearance

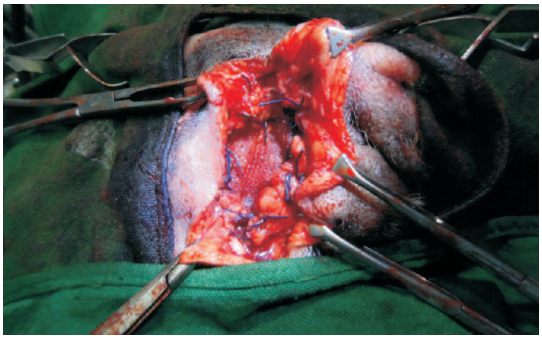

Figure 2: Prolene mesh in place

interrupted Lambert sutures (Vicryl Plus No. 1-0). After the rectum was restored, the internal obturator muscle was elevated. It was sutured medially and laterally by coccygeus and levator ani muscles along with the Prolene mesh using mattress pattern 1-0 polypropylene sutures.

The entire area was applied with povidone iodine and metronidazole topical solution. The available subcutaneous tissue and then after skin edges were approximated with 1/0 vicryl and black braided silk suture respectively. The routine dressing with antibiotic coverage (Ceftriaxone @ 250mg I/M, OD for 7 days) and fluid therapy was given for 5 days and the case recovered without any complication.

Discussion:

The postoperative pain causes continual straining for a long time before healing may lead to recurrence of herniation (Vnuck et. al. 2006). Prevention or relief of straining could have a role in preventing and retarding the progression of development of perineal hernia and postoperative analgesia is important. In the present case, no complications were recorded associated with analgesia obtained by parental nonsteroidal anti-inflammatory drugs administration.

Perineal wound infection is the most common complication, ranging from 5 to 45 percent (Bellenger and Canfield, 2003). In the present study careful placement of sutures was carried out by the guide of an assistant who everted the rectum by his finger during the correction of diverticulum, and antibiotics were used subsequently for a week to prevent infection and it recovered the case.

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Cutaneous Leishmaniasis in a Dachshund dog

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Cutaneous leishmaniasis is a vector borne parasitic disease caused by the protozoan parasite of genus Leishmania .The disease is transmitted by the bites of Sand flies of the genus Phlebotomus and is of zoonotic importance .The disease is endemic in India and has been reported in dogs from Rajasthan (Ahuja et al.,1993;Ahuja et al.,2012) and in man from different parts of India (Kochar et al.,2006, Sharma et al.,2007 ; Purohit et al.,2012).Cutaneous leismaniasis in dogs seems to have not been reported from Gujarat. Therefore the present case of cutaneous leismaniasis in a dog from Gujarat is reported and discussed.

History and Detailed Examinations:

An adult (27 month old) male Dachshund, weighing 15.1 kg, was referred to the hospital with slight persistent unilateral nasal bleeding, posterior ataxia and non-healing lesions at mouth commissure and nose from Rajkot (Gujarat). History revealed that the dog was ill with erratic temperature reaching upto 103.6^{0}F, erratic unilateral nasal bleeding, posterior weakness and non-healing lesions at mouth commissure and nose for last 5-6 months and had remained refractory to routine treatment. Detailed clinical examination revealed dullness, normal temperature (101.3^{0}F), weakness ,lethargy, blood spots at right nare, muco-cutaneous lesions at mouth commissures ((Fig.1), and nose.

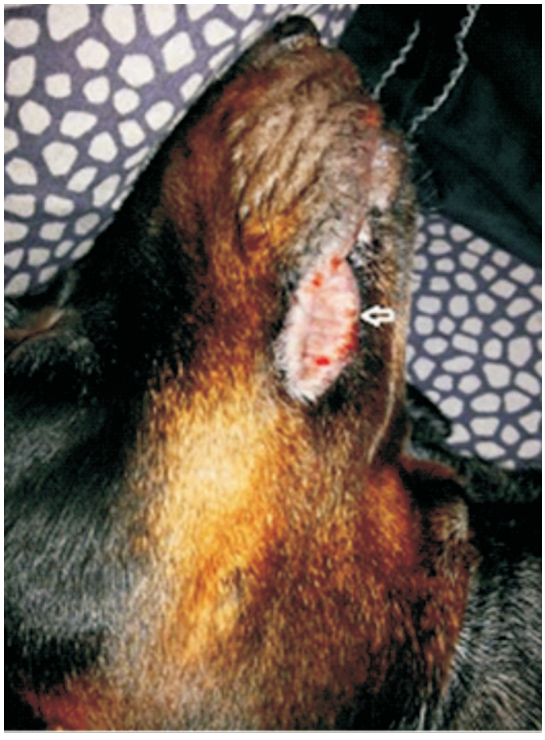

Fig.1. Non-healing muco-cutaneous lesion at right mouth commissure

Blood smear was negative for haemoprotozoan and rickettsial parasites. Total leucocytes (25000/mm3), total erythrocytes (4.50 millions/mm3), haemoglobin (9.2 g/dl), packed cell volume (28.0 %), thrombocytes (89000/mm3), lymphocytes (20%), monocytes (2%),eosinophills (2%) and neutrophils(76%) counts revealed leucocytosis with mild anaemia and thrombocytopenia. SGPT (74.8 U) and serum bilirubin (total 0.63, direct 0.52 and

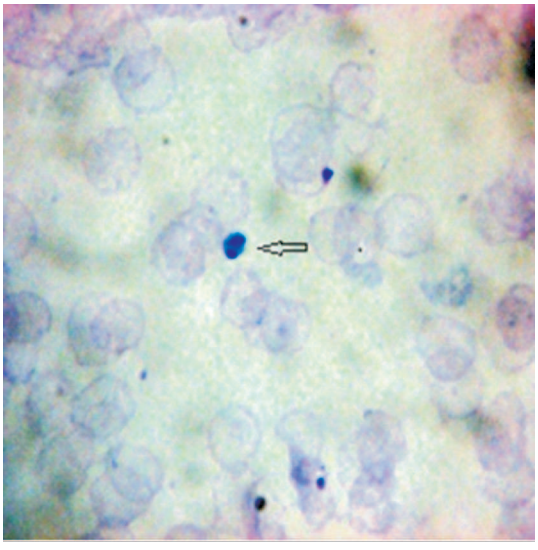

Fig.2. Amastigote of Leishmania in smear taken from lesion. Giemsa stain x1000

indirect 0.11 mg/dl) were within normal range , however, serum creatinine (2.4 mg/dl) , BUN (184 mg/dl), total serum proteins (8.9 g%) and serum globulin (6.8 g%) levels were high. Impression smears prepared from muco-cutaneous lesions after slight scrapping showed amastigotes indistinguishable from those of Leishmania (Fig. 2). Ultrasonogram revealed loss of echo differentiation between renal cortex and medulla (Rt. K 55x 35 mm, Lt.K 37X40 mm), hypoechoic liver and normal spleen. Electrocardiogram and chest X-rays were normal.

Diagnosis:

Based on cytological finding of amastigote in Giemsa stained smears prepared from muco-cutaneous lesion and clinical signs , the diagnosis of cutaneous leishmaniasis was arrived at.

Treatment:

Treatment with domperidone (0.5 mg/kg PO BID), ketoconazole (10 mg/kg PO OD), arachitol (3.0 lac IM fortnightly), prednisolone (1.0 mg/kg PO OD), allopurinol 10.0 mg/kg PO BID and Silybon 10 mg/kg PO BID was initiated and advised to continue for 30 days.

Discussion:

Cutaneous leishmaniasis in the present case was diagnosed on cytological evidence of amastigote in the smears prepared from muco-cutaneous lesion. Sporadic cases of cutaneous leishmaniasis in dogs have been reported from Rajasthan (Ahuja et al.,1993; Sharma et al.,2003). Despite reports of human leishmaniasis from Gujarat (Sharma et al.,2007; Purohit et al.,2012), the disease seems to have not been reported in dogs. Symptoms observed in the present case were in agreement with those described for cutaneous leismaniasis in dogs by previous workers. Unilateral epistaxis, might be the result of both ulcerative lesions of the nasal mucosa and/or impaired coagulation, due to hyperglobulinemia and

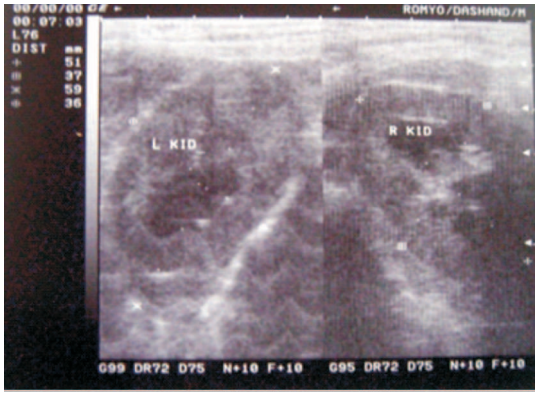

Fig.3. USG of the above dog showing loss of normal echo texture of kidneys

thrombocytopenia (Slappendel and Ferrer 1998).Increased serum creatinine and blood urea levels indicated renal insufficiency (Ciaramella et al.,1997) indicating the grave prognosis. The pentavalent antimonials sodium stibogluconate and meglumine antimoniate have been the first-line treatment for leismaniasis, their nonavailability makes the treatment frustrating. Further, antimonials are toxic with frequent, sometimes life-threatening, adverse side effects, including cardiac arrhythmia and acute pancreatitis. Various oral medications such as ketoconazole (Belazzuog et al.1985; Singh et al.,1996), itraconazole, metronidazole, allopurinol, dapsone, etc. have been used in humans but their effects are inconsistent. Therefore allopurinol (Vercammen and De Deken,1996) was used with Ketoconazole and prednisolone was added in the treatment regime because of renal involvement (high serum creatinine and blood urea and sonographic evidence). Domperidone was given to control emesis and silybon to guard liver stress on allopurinol and ketoconazole therapy. Unfortunately the dog succumbed to renal failure on 3rd day of therapy.

This seems to be the first case report of cutaneous leishmaniasis from Gujarat.

Acknowledgements:

Sincere thanks are due to Managing Trustees and Board of Trustees for providing necessary facilities, and the owner of the dog for her pains and cooperation in investigations.

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Demodicosis in a German Shepherd Dog

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Introduction and Case History :

Demodex mites live in hair follicle and sebaceous glands. It is a very obstinate skin disease and is refractory to most of the treatment. A German shepherd dog was brought to Teaching Veterinary Clinical Complex, College of Veterinary Science, Proddatur with a history of generalized alopecia, presence of pustules on the skin, slight itching, swollen body parts (legs, base of tail). It was reported that the dog was under the antibiotic treatment since two months and there was recurrence of the condition. Temperature, pulse were normal and conjunctival mucous membrane was pale.

Microscopic and Hematological Findings

Microscopic examination of the skin scrapings revealed presence of 6 motile demodex mites per field, indicating the severity of the condition. Hematological evaluation revealed severe anaemia with eosinophilia and leukocytosis. Biochemical analysis of serum indicated hypoproteineamia.

Treatment:

Treatment of canine generalized demodicosis is multimodal. In addition to effective acaricidal therapy, treatment of concurrent bacterial skin infection, internal parasitism and underlying systemic disease must be undertaken to maximize the potential for successful treatment. The hair present on the body were clipped for easy identification of the lesions.

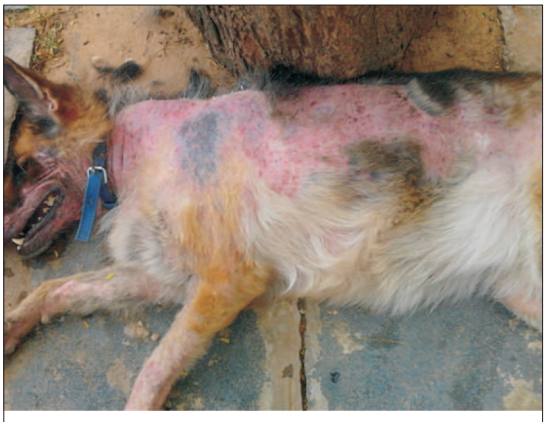

Fig. 1: GSD dog showing generalized alopecia and nodular lesions of mange.

Following medications were given :

1. Injection IVERMECTIN 0.2 mg/Kg B WT S/C once in 4 days.
2. Suspension ENDECTIN 1ml/4Kg B WT oral daily for 45 days
3. Injection Tefrocef 2 mg/Kg B WT for 5 days.
4. Injection Avilin 3ml/kg B Wt daily.
5. Advised Taktic (Amitraz) 10 ml/1lit of water once in 4 days locally.

Supportive therapy:

1. Injection Haemocoel ½ lit I/V for 4 days once
2. Oral sharcoferal ½ table spoon BID daily
3. Injection Inferon 2ml I/M for 4 days once
4. Advised Petben shampoo topical once in 4 days.

Advised to continue the treatment up to 90 days.

After 2 weeks, hyperpigmentation of the skin was observed and the hair growth started. After 45 days, skin scrapings examination revealed presence of dead mites. Complete recovery and negative skin scrapings were observed after 90 days.

Discussion:

Chronic generalized canine demodicosis is a frustrating disease to treat. General health and management of dogs with demodicosis should be addressed initially. Secondary pyoderma associated with localized or generalized disease should be treated with appropriate antibiotic therapy. Discontinuation of antibiotic therapy should be based on clinical re-evaluation. Corticosteroid therapy in any form is contraindicated in dogs with localized or generalized demodicosis.

Benzoyl peroxide (2–3%) and chlorhexidine-based shampoos (3–4%) are commonly recommended for dogs with demodicosis. They have a prolonged antibacterial activity on skin (Kwochka et al,1991). Benzoyl peroxide is degreasing, thus drying, and may be irritant, so it may be prudent to follow up with a moisturizer to prevent drying of the skin (Mueller, 2008). The frequency of topical therapy depends on the dog, owner and concurrent miticidal therapy, but weekly bathing is most commonly recommended. Daily use of ivermectin, at a dosage of 0.6 mg/kg, PO, was found to be effective in the treatment of generalized demodicosis in dogs(Ristic et al 1995).

The recommended concentration of Amitraz varies from 0.025 to 0.06%, with a frequency of once weekly to every 2 weeks. Clinical efficacy increases with increasing concentration and shorter treatment intervals (Hugnet et al, 2001). It is not sufficient to rely on clinical appearance as the end-point of treatment. Clinically normal dogs may still harbour mites on skin scrapings. Microscopic cure, defined as multiple negative skin scrapings, in addition to resolution of clinical signs is needed to determine the therapeutic end-point.

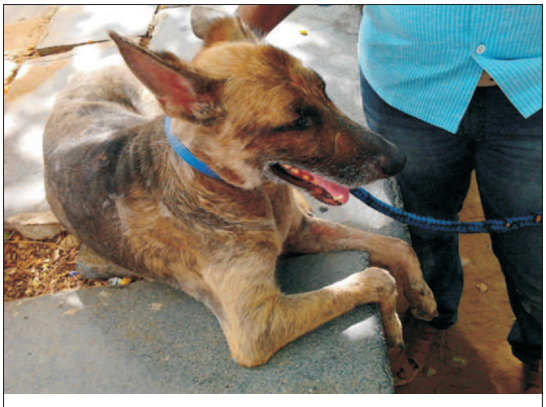

Fig. 2: GSD demodex affected dog showing clinical recovery

Fig. 3: GSD demodex affected dog showing clinical recovery

Based on published studies, a recurrence of the disease in the first 1–2 years after cessation of therapy does occur in a small number of dogs. (Mueller, 2004). The majority of these cases achieve remission with a repeat of the same treatment regimen or with another type of therapy(Mueller, 2004). In more recent studies, a follow-up period of 12 months is recommended to monitor for relapse.

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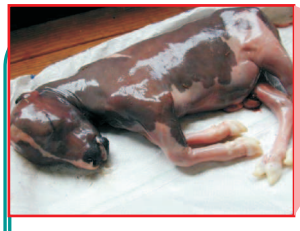

Brucellosis : A hurdle in Farm Livestock Development

The Organisms - contd.

Genus Brucella has been divided into **six** classical species, with different biotypes.

Brucella melitensis - biotypes I, II, III

Brucella abortus - biotype I

Brucella suis - biotype I, II

Brucella canis -

Brucella ovis -

Brucella neotomae -

The first three have small ruminants, cattle (buffaloes) and pigs as preferred hosts respectively and are consider most pathogenic for humans.

Importantly, *Brucella canis* (dogs) has low zoonotic potential, while *Brucella ovis* (sheep) and *Brucella neotonae* (desert rats) are considered not associated with human brucellosis.

To Brucella spp. isolated and identified front marine mammals, *Brucella ceti* and *Brucella pinnipedialis*, have also been found to cause human brucellosis

The epidemic potential, absence of reliable vaccine, the drawbacks of available vaccine strains in terms of safety, so also, the efficacy of brucella to produce aerosol infection has made this organism to be classified as "Biosafety level - 3" pathogen and considered as a potential bio-terrorist agent.

Surgical management of umbilical hernia in a female calf

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Introduction:

Hernia is a protrusion of an organ or tissue through the opening (Venugopalan., 2000). The opening may be caused by tear in the abdominal wall or diaphragm or it may be a natural opening. Umbilical hernia occurs frequently in bovine calves and this condition is comparatively more frequent in females than in males (Tyagi and Jith Singh, 2008). The contents usually consist of intestine and omentum. Umbilical hernia is common in young animals and embryonic defect like imperfect closure of the umbilicus is the predisposing factor for the umbilical hernia in young ones. Umbilical hernias are the most common bovine congenital defects and can occur in any breed, although they appear to be most common in Holstein-Friesian cattle. Increased intra abdominal pressure and blunt injuries also lead to umbilical hernia. Umbilical hernia is more commonly seen in foals, pigs, calves and pups.

History and clinical observation:

Two month old, Holstein- Friesian cross bred female calf weighing around 45 kg was reported with compliant of swelling in the umbilical region with a gradual increase in its size (fig 1). The animal was showing symptoms like anorexia and pain during defecation. On clinical examination, hernial swelling at the umbilicus was observed. By physical examination, it was differentiated from hematoma and abscess. Careful palpation of mass revealed a reducible hernial contents. By

Fig.: 1. Calf showing swelling in the umbilical region

restraining the animal in dorsal recumbency, hernial ring was palpated.

Surgical procedure:

Calf was fasted overnight. Pre-operatively, Streptomycin + penicillin (Dicrysticin) 1.5ml was given intramuscularly. Surgical site was prepared aseptically under field condition. Site

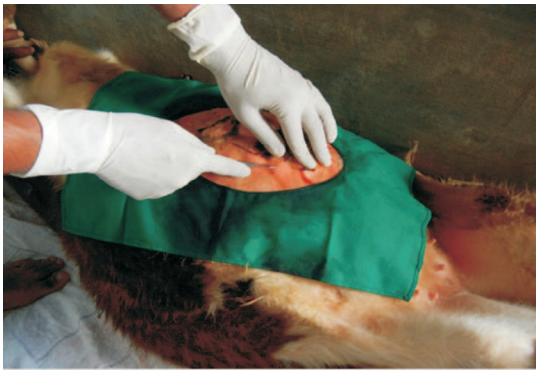

Fig 2 : An elliptical incision made on skin

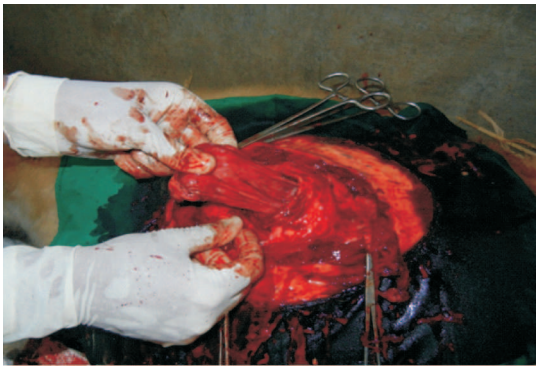

Fig 3 : reducing the hernia content through hernia ring

was shaved and scrubbed with povidine iodine and anesthetized with local anesthetic lignocaine HCl 2%. Animal was restrained in the dorsal recumbancy and an elliptical incision was made on the skin and the hernial ring was explored (fig 2). Hernial content noticed was the omentum. Adhesions around the hernial content were cleared. Henial content was successfully reduced (fig 3) and hernia ring was closed by overlapping suture pattern using silk(fig 4). Skin was closed by simple interrupted

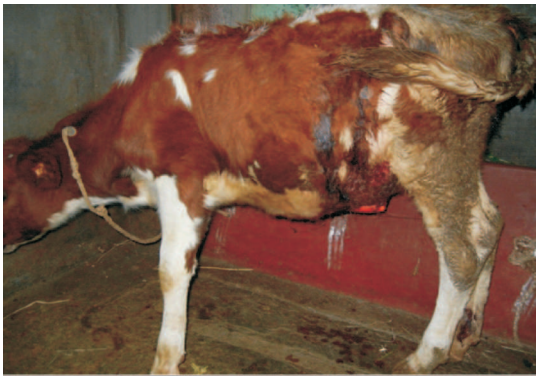

Fig 5 : Calf after surgery

pattern using nylon. Post-operatively, suture line was dressed and meloxicam 2.5ml was given

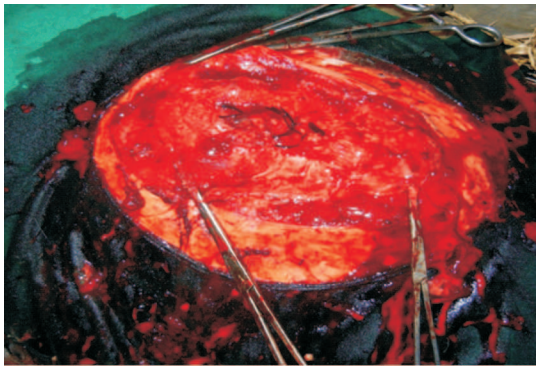

Fig. 4 : closing the hernia ring by overlapping suturing method

intramuscularly. Dicrysticin 1.5ml intramuscularly and meloxicam 2.5ml intramuscularly was continued for another six days. Animal recovered uneventfully (fig 5) and the suture was removed on the tenth day.

Discussion :

Umbilical hernia might be congenital or acquired and are seen in foals, calves and pigs (Turner and Mcllwraith, 1989). Congenital umbilical hernias are common defects in calves, particularly in Holstein Friesian cattle, where frequencies between 4 % to 15 % had been observed (Hondele, 1986; Muller et al., 1988; Virtala et al., 1996). Restraining of animal in the dorsal recumbency made the force to move contents into the abdominal cavity. An elliptical incision on skin made to have clear view of contents and with minimum damage to it. The content was moved carefully. Hernial ring was closed with non absorbable suturing material by overlapping suture technique (Simon et al., 2013 and Thangadurai and Vijayakumar., 2013). Inhalation anaesthesia is preferrable in foals for herniorraphy. (Marke et al., 1987). In the present case, calf herniorraphy was performed under local analgesia with lignocaine

HCl 2%. Skin sutured by non-absorbable suture material nylon by interrupted suture pattern. Complications of umbilical hernia repair observed were seroma formation, abscess, haematoma and breakdown of hernia repair (Weaver et al., 2005). However, in the present case of umbilical hernia, there were no complications and animal recovered uneventfully.

To avoid post operative wound infection, antibiotic strepto-penicillin and pain killer meloxicam (Melonex) was given for next five days. Animal owner was advised to withhold the solid food for next 36-48 hours to reduce the rumen volume, thereby avoiding excessive pressure on the abdominal cavity.

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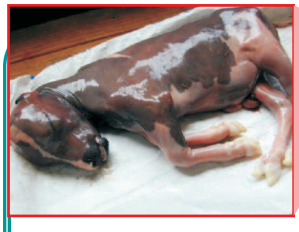

Brucellosis : A hurdle in Farm Livestock Development

The Pathogenic Potential

The pathogenic potential of Brucella organisms is highly dependent on their ability to enter, survive and replicate within the host cells. These organisms do not have classical virulence factors like exotoxins or endotoxic lipopolysaccharides. The major virulence mechanism is due to host cell invasion and intracellular survival/multiplication.

It is interesting to note that Brucella organisms survive and multiply in macrophages and dendrite cells, the very cells designed to eliminate the invading bacteria and provide prime adaptive immunity.

Successful management of Uterine Prolapse In non-decript Buffalo under field conditions

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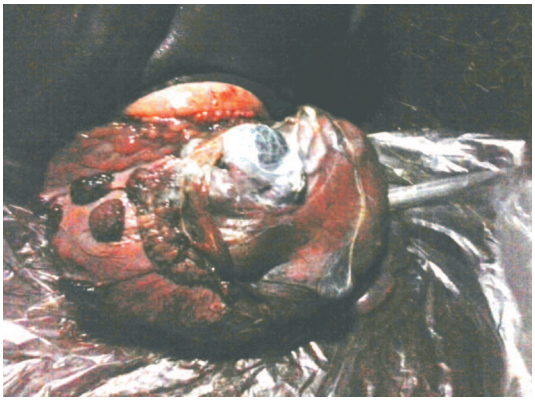


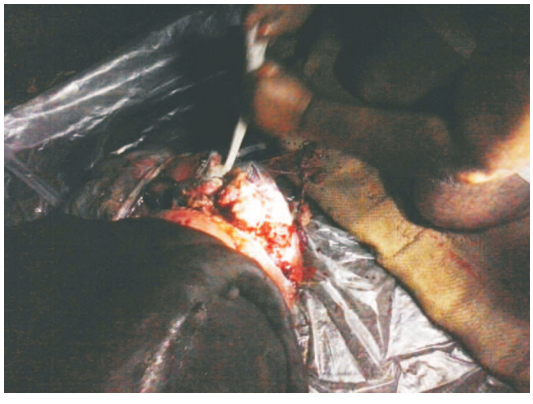
Prolapsed uterus with fetal membrane, the attachment of placenta to caruncles

Introduction:-

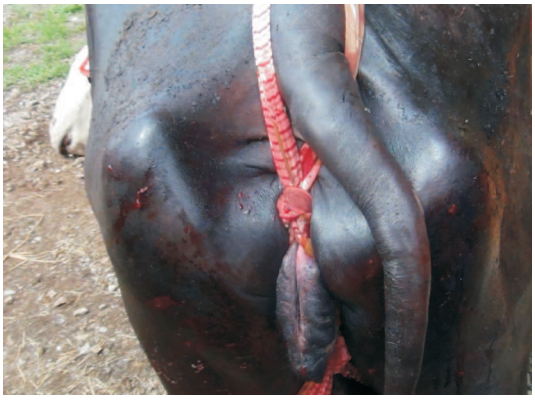
Prolapse can be defined as eversion of the genital organs from its original position outside the body. Prolapse may be partial or complete. The eversion of the genital organs in farm animals occurs during pregnancy (pre-partum) or after the parturition (postpartum). Prolapse of the uterus is the common complication of third stage of labour in cows and ewes. In ruminants, the prolapse is generally complete inversion of gravid cornu. The incidence varies from 2 per 1000 calvings in range beef cattle in America (Patterson et al 1979) to 3 per 1000 cows per year in Scandinvian dairy cattle (Rasbech et al 1967; Ellerby et al 1969; Odegaard, 1977; Roine and Soloniemy, 1978). Uterine prolapse is a disorder of ruminants most frequently observed in buffaloes. It is observed after parturition and rarely observed unconnected with pregnancy and parturition. Prolapse is seen in all species of animals, however, more common in dairy animals as compared to drought breeds (Sane 1993). The causes of the prolapse are probably multiple. The incidence of prolapse is more common during last two months of gestation, probably due to large amount of estrogenic hormones being secreted by the placenta (Robert 1975). The present communication reports complete uterine prolapse in buffalo and its successful management.

History and diagnosis:

A 5-6 year old buffalo, weighing about 400 – 450 kg, in her second gestation, was presented with a history of normal calving, retention of



Cleaning of mass with antiseptics and removal of placenta



Application of rope truss to prevent the recurrence of prolapse

fetal membranes and complete eversion of uterus 4-6 hours after calving. The gynaeco-clinical examination of the buffalo revealed that the uterus was completely everted, fetal membranes were not expelled completely, temperature, heart rate, respiratory rate of the animal were 98°F, 83 BPM and 20 per min. respectively. The buffalo was in recumbent position. Uterus was enlarged and edematous covered with dirt and blood clots. The straining was continuous by the animal. The case was diagnosed as the complete uterine prolapse.

Treatment and Management:

The treatment of the prolapse was carried out in three steps viz. Reduction, Reposition and Retention of the prolapsed mass. The epidural anesthesia was achieved by administration of low dose of 2% lignocaine hydrochloride to prevent straining and easy manual correction. The prolapsed uterus was thoroughly cleaned with antiseptic potassium permanganate solution (1:1000). The fetal membranes and other debris material were removed by gentle squeezing the placentomes which aids in early involution of the caruncles. The ice cubes were applied to reduce the size of the mass. The reposition of the prolapsed mass was done manually starting from the lateral walls of the uterus and then towards the central part. The force was applied by using the palm, and the uterus was placed to its original position. The retention was achieved by applying rope struss. Intravenous infusion of 450 ml calcium preparation (Mifex) was given for correction of hypocalcemia and to maintain the tonicity. The routine post-operative treatment of antibiotic, analgesic and fluid therapy was followed for three days. The eventful recovery of the animal was confirmed by removing rope struss and recurrence of prolapse if any after seven days.

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Indian Squill Toxicity in Ruminants and its treatment

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Indian squill
(*Urgenia indica / Drimeria indica*)

Introduction

Scarcity of feed and fodder during summer season makes livestock to graze on plants that may be poisonous. The situation is grave especially in drought prone areas. Some farmers of Chittoor dist in Andhra Pradesh reported deaths in white cattle upon grazing of leaves and bulbs of unknown plant. An investigation was conducted and the plant responsible for toxicity was identified to be Indian Squill, *Urginea indica / Drimeria indica* named in different languages as Sea onion (English); Adavi tellagadda; Nakka vulligadda (Telugu), Jangir kanda (Gujarati) ; Jangli kanda or Jungali pyaj (Hindi); Kattulli (Malayalam) Vanapalandam (Sanskrit) Nari vengayam (Tamil) and Sarpkand (Marathi). The plant is a robust perennial with a globose bulb, to 15cm (6 in) across, composed of overlapping tunic scales and narrowly lanceolate, glaucous leaves, 30 cm to 1 m (1-3 ft) long. Small, star-shaped, white flowers are produced in a dense spike in late summer and early autumn, followed by new leaves.

Toxicology of Indian Squill

The plant is cyanogenic. The leaves and bulbs are eaten by ruminants in drought. The major toxic principles present in the plant are hydrocynic acid, scilliroside and scillaren which is a mixture of scillaren A and B. (Frank, 2004). Scilliroside is a cardioactive bufadienolide glycoside and is the main active toxic principle (Verbiscar, 1986). The toxic dose in ruminants has been reported to be 200-250mg/Kg (Sandhu and Brar, 2009)

Symptoms of Toxicity

The toxic symptoms include ataxia and hyperaesthesia followed by paralysis, bloody diarrohea, ruminal atony, posterior paresis, frequent micturition, convulsions, tremors, tachycardia, arrythmia followed by bradycardia and cardiac arrest. The symptoms of diarrhoea, gastritis and enteritis are due to the direct actions on gastrointestinal tract. Deaths in cattle and buffaloes were reported earlier in Tarikere district of Karnataka (Narayana, 2003). Further it

was also reported that death occurs immediately following the drinking of water after eating the plant leaves and bulbs of *Drimeria indica*, which may be due to hydrolysis of glycosides releasing toxic principles. The initial signs of poisoning occur within 12 h and death usually follows within 3 days. The clinical course seldom is longer than 24-36 hours. Generally animals exposed to sub-lethal dose recover in less than 48 hours.

Post-mortem Lesions

Post mortem of dead animals revealed enlargement and congestion of abdominal and thoracic organs, gastritis and enteritis. Kidneys, liver, lungs and myocardium show signs of congestion and swelling. Histopathology of the organs revealed degenerative changes.

Diagnosis

Diagnosis is based on the history of grazing on the plant, identification of the plant and clinical symptoms.

Hyperkalemia is a primary manifestation of acute cardiac glycoside toxicity and an early predictor of need for therapy. Hyperkalemia (>5.0 mEq/L) in acute toxicity and life threatening hyperkalemia (>6.5 mEq/L) may be seen.

Confirmatory diagnosis is established by identification of the plant, the glycoside or both in ingesta in association with myocardial lesions. (Radostits et al.,2005)

Differential diagnosis with other toxicants causing cardiomyopathy and similar clinical signs include *Urechites lutea*, Albizia sps, Fluroacetate, Gossypol, Ionophore antibiotics, nutritional deficiencies of vitamin E, selenium, and copper.

Treatment

No specific antidote is available for treatment: A large number of animals consuming this plant material at sub lethal doses can survive without any treatment. Care is needed while handling the animals having consumed a large dose of poison as they will struggle fiercely when caught and held for treatment. This may lead to excitement, resulting in death due to cardiac arrest. Hypercalcemia and hypomagnesemia exacerbate cardiac glycoside toxicity. Therefore, drug of choice include agents that promote potassium redistribution from extra cellular to intracellular compartments.

Treatment consists of supportive therapy and evacuation of the GI tract.

- Activated charcoal: 1-3g/Kg body weight in all species at 4-8 hr interval which is used to bind the glycosides remaining in rumen and intestine. Even though a single dose is sufficient, due to enterohepatic / enteroenteric recirculation of cardiac glycosides, multiple doses can be given at 4-8 hr intervals to help enhanced elimination.

- Glucose i.v and Sodium bicarbonate i.v : Glucose causes redistribution of potassium intracellular and the onset of action is 30 min and duration of action is 4-6 h. The sodium in sodium bicarbonate counteracts potassium effects, while alkalosis created by bicarbonate leads to redistribution of potassium intracellularly. The onset of action is 5-10 min and duration of action is 1-2 h. In chronic cases, however there will be hypokalemia, hence potassium chloride at 1g/kg should be used in addition to activated charcoal.

- Atropine sulphate: Cattle 30 mg per 50 kg. The recommended average initial dose should be split, injecting one quarter (1/4) to one-third (1/3) slow i.v. and the remainder i.m or s.c. Atropine abolishes vagal tone and thus increases heart rate. Hence used for bradycardia and conduction block.
- Lidocaine 0.5 mg/kg i.v. : Lidocaine is a class IB antiarrhythmic agent that increases electrical stimulation threshold of the ventricle, suppressing automaticity of conduction through the tissue.
- Propranolol : It is a β blocker and contributes for antiarrhythmic action.

Contraindications

Calcium supplements, Corticosteroids, Quinidine, Stimulant laxatives and diuretic drugs are known to produce interaction with squill hence need to be avoided.

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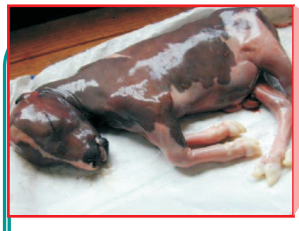

Brucellosis : A hurdle in Farm Livestock Development

The Pathogenic Process

Brucella organisms have the unique ability to persist and replicate within the phagocytic cells of reticulo-endothelial system, like macrophages and dentrite cells as well as in non-phagocytic cells like trophoblasts (placental cells providing nutritional and hormonal support for maintenance of pregnancy). The endoplasmic reticulum association of Brucella intra-cellularly favors the intracellular replications of organisms in above cells. Intracellular growth and multiplication of Brucella is more pronounced in dentrite cells.

Trophoblastic cells are key target cells of brucella infection during the late phase of gestation in ruminants. Growth and multiplication of Brucella in trophoblasts is enhanced in the presence of high concentration of streroidal hormones and **erythritol** (Brucella growth / multiplication promoting secretion of trophoblasts) during the final third of gestation. Rapid multiplication of organisms inside the trophoblasts disturb the integrity of the cells, resulting into placentitis and infection to fetus. The placentitis promotes enhanced release of hormones like PGF2α, estrogens and cortisol with concurrent reduction in progesterone that ultimately results into expulsion of dead/infected fetus.

Oestrous ovis infestation in a lamb

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Introduction :

Oestrosis is a world wide infection caused by the larvae of the fly *Oestrous ovis* (Diptera, Oestridae), that develops from the first to the third stage larvae. This is an obligate parasite of the nasal and sinus cavities of sheep and goats (Gunalan et al 2011). The adult female lays larvae near external nares and the larvae crawl towards ethmoid region where they develop for some months and from there go to the frontal sinus or maxillary sinus where they become mature in few weeks. Altogether, larvae may be in sheep for about 10 months. They leave the host during the spring. They are sneezed out and drop on to the ground and pupate (Sastry,2000). The present paper describes the *oestrous ovis* infestation in a lamb and its management.

Case history and Observations:

A six month old male lamb presented to Veterinary Ambulatory clinic, Mylardevpally, College of Veterinary Science, Rajendranagar with the history of sneezing, head shaking, pressing the nose against wall, restlessness and slight inappetance for a week. Upon clinical examination, there was unilateral mucoid nasal discharge(Fig 1),mild respiratory distress with normal temperature and pulse rate. Upon sneezing, nasal bot (*Oestrous ovis*) were sneezed out (Fig 2) which were identified as per Soulsby, (1982). Morphological examination

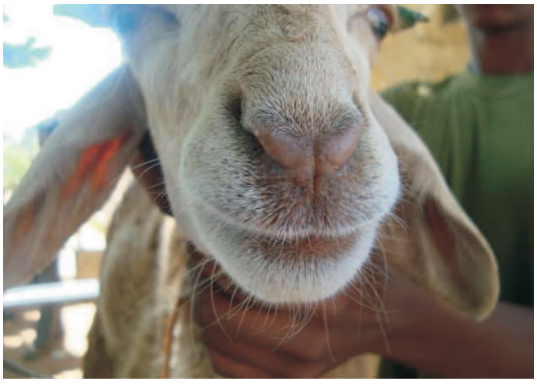

Fig. 1: Lamb with unilateral nasal discharge

revealed that bot was at mature stage of larvae of *Oestrous ovis* which was creamy in color with dark transverse bands on the dorsal aspect indicating typical characteristics of Oestrous ovis. Faecal examination revealed presence of strongyle eggs. Hematological parameters were normal except for increase in eosinophilic count. Based on history , clinical manifestations of head shaking, mucoid nasal discharge and morphological characteristic of the nasal bot confirmed the infestation of Oestrous ovis larvae.

Treatment

The affected lamb was treated with Ivermectin (Inj. Neomec) 0.2 mg/kg body weight subcutaneously once , followed by Inj . Tribivet 1ml intramuscularly and Inj. Chlorpheneramine maleate 1ml intramuscularly for 3 days. On day 2, there was reduced sneezing, nasal discharge

Fig. 2: Nasal bots (*Oestrous ovis*)

and improvement in appetite. The lamb made an uneventful recovery within 7 days post therapy.

Discussion

Clinical signs such as breathing difficulties, nasal discharge, emaciation together with annoyance caused by adult flies leads to production and economic losses (Dorchies et al 1998) which are in agreement with the present findings. *Oestrous ovis* impairs breathing because it induces tenacious nasal discharge to which grass, straw and dust adhere , clogging the air passages (Dorchies et al 1993).The first stage larva(L1) stage is commonly found in sheep less than one year of age due to existence of a period of hypobiosis. Respiratory distress such as loud, noisy breathing, poor body scores (severe emaciation), ill thrift and poor kidding rates are observed. (Gunalan et al 2011). The spiny surface of the larvae causes irritation of the nasal mucosa resulting in catarrhal rhinitis with sneezing, mucopurulent discharge, snoring respiration (Radostits, 2000).The *Oestrus ovis* has immunosuppressive effects with consequent association with respiratory pathogen (Dorchies, 1993). Prompt use of Ivermectin/ Doramectin reduces the oestrous ovis burden in sheep together with other sheep parasites or helminthiasis. Treatment with closantel @7.5 mg/kg and Ivermectin @0.2 mg/kg are effective in controlling nasal bots (Radostits, 2000).

It is concluded that all unilateral discharges may be suspected for *Oestrous ovis* infestation, which will respond well to the treatment with Ivermectin.

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Melanoma in Ruminants

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Introduction

Melanoma is a benign tumour of melanoblasts, the melanin containing cells, present in the basal layer of epidermis. Melanomas originate from neuroectoderma melanoblasts, which migrate at the beginning of the development period into the epidermal-dermal junction of the skin, follicles, and dermis. They are also found in ocular structures, meninges, adrenal glands, endocardium, and intima of blood vessels (Pulley and Stannard, 1990). In India, melanomas are mostly seen in dark coloured cattle, buffaloes, sheep and goat. These growths are black in colour, raised as nodules on the surface and contain black brown pigment. The melanomas may occur on the skin, mucus membrane and eye. Most melanin pigment tumours are benign in cattle, however in some cases malignancy may occur. (Tyagi and Singh, 2008) Melanomas are encountered in all animals that vary in size from tiny specs to large tumours. They are black or brown in colour and may be round, nodular, flat or pedunculated. Cutaneous tumous are prone to trauma and so may become ulcerated and infected. Benign tumour is called melanoma and their malignant counterpart is called melanocarcinoma. (Shastry G.A. and Rama Rao P., 2001)

History :

Case 1:

A 6 year old cow was presented with two large growths on craniolateral aspect of left thigh (Fig. 1 and 2) since last 9 months. The mass was increasing in size day by day. On palpation, the mass was hard in consistency and fine needle aspiration revealed frank blood.

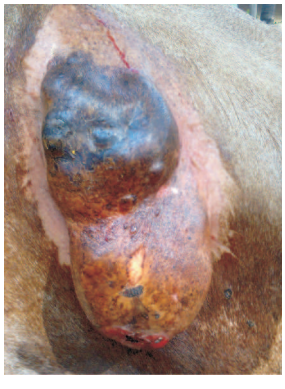

Fig.1 & 2: Multiple large hard masses at craniolateral aspect of left thigh

Fig.3: Tennis ball size large growth hanging on the ear pinna of a goat

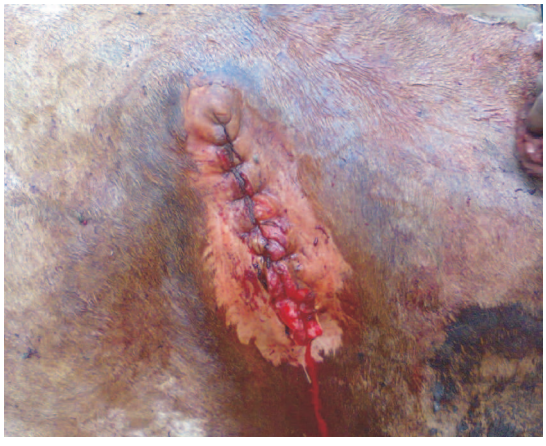

Fig.4: Surgical wound at the thigh region of cow after growth excision

Case2:

A two year old female non descript goat was presented with large tennis ball size hard mass with signs of ulcerations hanging on the tip of right ear pinna (Fig.3) causing discomfort to the animal in its daily routine activities. The mass was small in size initially and was increasing in size since last 8 months.

Treatment and management

Case 1:

It was decided to surgically excise the growth under local anaesthesia lignocaine. An elliptical incision was made and growth was aseptically removed. Since no loose skin was available at thigh region, the mass was excised deep inside along with a part of subcutaneous tissue to bring skin in apposition (Fig. 4). Post operatively, animal was given Inj.AC-Vet Forte-D 3gm sid IM for 4 days along with Inj. Maxxtol, 30ml IM. Animal recovered uneventfully and any reoccurrence of mass was not seen.

Case 2:

Under local lignocaine infiltration, the mass along with normal ear cartilage was excised (Fig.5). Bleeding was countered by thermo cauterisation. Skin was apposed using horizontal mattress pattern, followed by post operative care.

Discussion

Melanomas arise from specialized cells containing melanin (melanoblasts) situated in the stratum germinativum of the epidermis. Initially the epidermis overlying the tumour may be intact, but becomes ulcerated with rapidly growing tumors (White et al., 2002). They may occur as solitary (Babic et al., 2009) or multiple lesions. Neoplasms in goats are rare, in comparison with other species of animals (Zubaidy, 1976).The causes of melanomas are uncertain and in humans, risk factors such as race, lack of skin pigmentation, excessive exposure to sunlight have been described (Madewell and Theilen, 1987). However, in

Fig.5: Surgical wound after removal of growth along with a part of healthy ear flap

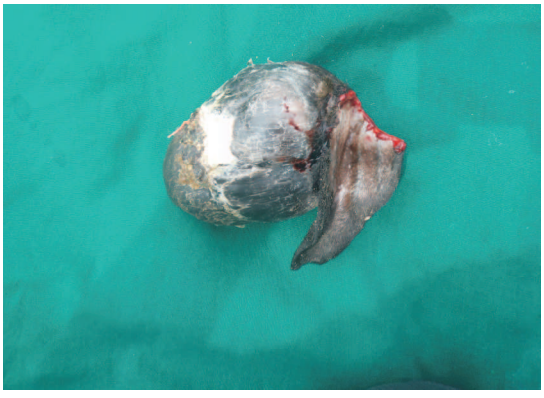

Fig.6: Excised mass along with a part of ear flap of a goat

most domestic animals, risk factors for melanomas have not been fully described. In horses, the loss of melanin pigment with age and a tendency to develop grey or white hair coats have been identified as risk factors (Madewell and Theilen, 1987). Both the cow and the goat in this report were nondescript, local animals and had normal hair coats. The goat was dark black and the cow was brown in colour. Some reports suggest that dark-skinned and hairy goats are more often affected (Venkatesan et al., 1979; Radostits et al., 2007).

Other domestic animals in which melanomas are associated with dark skin include pigs and dogs (Radostits et al., 2007; Madewell and Theilen, 1987). In goats, the sites of occurrence tend to be sparsely covered with hair, such as the ear, face, anus, vulva, tail, and udder (Venkatesan et al., 1979). Some reviews suggest that the occurrence of melanomas in cows and goats is secondary to mutations induced by ultraviolet solar radiation (Smith et al., 2002). In cattle and goats melanoma occurs commonly in perineal region and root of the tail followed by skin and udder. The horn base has been reported as primary site for malignant melanomas in goats (Mavangira et al., 2008). Prognosis of malignant melanomas in goats is guarded to poor or grave (Smith et al., 2002). Reportedly, melanomas of goats are highly malignant, locally aggressive, and commonly metastasize to other organs via the lymphatics and the blood stream (Smith et al., 2002). Tumors arising on the digits and on mucocutaneous junctions are reported to be more aggressive and behave in a similar manner to oral melanoma.

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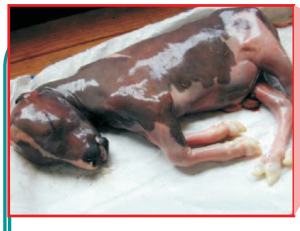

Brucellosis : A hurdle in Farm Livestock Development

The Role of Erythritol

Erythritol (H(CHOH)4H) is a tetrahydric, four-carbon Sugar Alcohol, produced by placental trophoblasts in increasing amounts during the later stages of pregnancy to serve as the preferred carbon and energy source for the developing fetus in cows, buffaloes, ewes, does and sows.

Erythitol being also a preferred carbon and energy source for multiplying Brucella organisms in the placental trophoblasts, it is postulated that when the availability of erythritol is abundant in late stages of pregnancy, the brucella organisms multiply rapidly and produce placentitis, which results into fetal death and abortions in farm livestock.

Cardiomyopathy in a Parrot

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Introduction

Avian cardiology is still in infancy and has not received enough attention in veterinary practice. Based on retrospective pathological studies, it is thought that 10-40 % companion birds have some form of cardiovascular diseases (Bob Doneley,2010). Despite common occurrence of cardiovascular diseases in caged birds accounting for nearly half the deaths yearly in pigeons in United States (Penn et al.,1990), cardiac diseases are often not diagnosed in birds until the heart decompensate because of non-specific clinical signs, and limited diagnostic techniques due to high heart rate and limited size of the patients. It seems that clinical cases of cardiac diseases in parrots have not been diagnosed in India. Therefore, a clinical case of cardiomyopathy associated with ventricular tachycardia in a syncopic parrot is put on record and discussed.

History and Detailed Examinations

A parrot, fallen on the road, was brought at the hospital in semiconscious, condition as an emergency case. Clinical examination revealed semi-conciousness,marked weakness, lethargy, gasping, dyspnoea and syncope.

The parrot was immediately subjected to electrocardiography. Leads were attached on the right wing (RA), left wing (LA) and left leg (LL), and right leg(RL) at gastrocnemius muscle as shown in the Fig 1. Feathers were clipped on the proximal part of the rachis and gel was applied liberally between skin and clips. Lead I, II, III, aVR, aVL and aVF were recorded at 1 mV = 10 mm, with paper speed of 25 mm/s.

A drop of blood, was subjected to semi quantitative test for the estimation of cardiac Troponin –I , (Employing Amicheck-Trop I kit – Zephyr Biomedicals) to detect myocardial damage, if any.

Diagnosis

Based on history of fall on the road, clinical signs of semiconciousness and tachypnoea, electrocardiographic evidence of ventricular tachycardia and positive reaction to semiquantitative cardiac troponin –I test, the

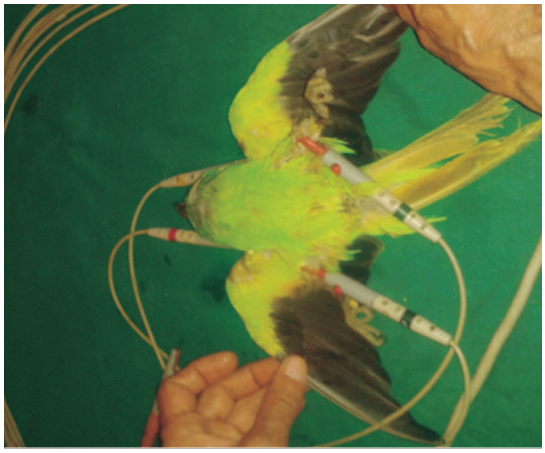

Fig. 1. ECG lead placement in the parrot. Leads were attached on the right wing (RA), left wing (LA) and left leg (LL), and the right leg (RL) at gastrocnemius muscle . Lead I, II, III, aVR, aVL and aVF were recorded at 1 mV = 10 mm, with paper speed of 25 mm/s.

syncope in the parrot was diagnosed due to cardiomyopathy.

Treatment

The parrot was given 0.025 mg digoxin (0.25 mg Digox tablet dissolved in 1.0 ml water and 0.1 ml given orally) , 0.1 mg propranolol (10 mg Ciplar tab. dissolved in 10 ml water and 0.1 ml given orally) and glucose in water orally drop by drop.

Discussion

Clinical signs of semi-conciousness, marked weakness, lethargy, dyspnoea and syncope in the parrot were suggestive of compromised cardiovascular system. The diagnosis of cardiovascular diseases in living birds/ parrots is difficult as there is no palpable pulse and auscultation is difficult to interpret owing to fast heart rate leaving electro and echocardiography as only means to evaluate the heart. Electrocardiogram in the present case revealed aberrant wave forms (VPC) in runs having wide QRS unrelated to 'P' wave (Fig.2) suggesting ventricular tachycardia. Ventricular tachycardia has been recorded in birds during the period of hypoxia and it is one of the most serious and potentially life threatening arrhythmias owing to its association with serious heart disease or metabolic derangements. Semiquatitative cardiac Troponin-I showed a positive reaction with the parrot's blood and thus confirmed that ventricular tachycardia in the syncopic parrot was associated with myocardial injury/cardiomyopathy. Though, much information on cardiac troponins in parrots/birds is not available, on the basis of

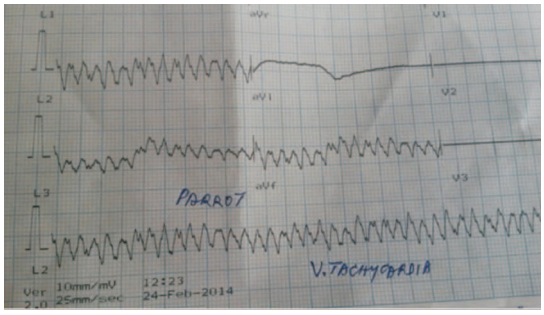

Fig2. Electrocardiogram showing ventricular tachycardia at an average of /min. in a parrot with syncope.

wide diagnostic window of cardiac troponin-I in human, it seems likely that the duration of increased cardiac troponin-I following cardiac injury in other mammals and in birds will be sufficient to provide diagnostic and prognostic indications (O'Brian et al.,1997. Despite initiating treatment with digoxin and propranolol, the parrot succumbed to its illness in the evening (eight hours post therapy). The present report is the first record of a clinical case of cardiomyopathy associated with ventricular tachycardia in a parrot in India.

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Nitrate plant poisoning in animals: Managemental perspectives

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Introduction :

Poisoning in animals is one of the main concerns for veterinarians. Many a times this poisoning is either malicious or accidental which includes poisoning by plants, animals, microorganisms, minerals, industrial wastes, agrochemicals, household products, therapeutic agents, food and feeds etc. causing heavy economic losses to livestock industry. Among these, poisonous plants are the most important and common cause of poisoning affecting the animals in several ways. Animals are poisoned mostly when they are hungry or when other conditions which cause them to graze abnormally or when wrong managemental or agricultural practices are adopted in a farm. Nitrate poisoning is a universal and economically important problem for ruminants due to the ingestion of plants accumulated with toxic levels of nitrate. Therefore, the information about the nitrates plant poisoning and the remedies on it should be available with the livestock farmers as it is very helpful at the time of emergency to save the life of animals.

Table:1 Nitrate accumulating plants

Botanical name	Common name
Amaranthus spp.	*Pigweed*
Chenopodium spp.	*Lamb's-quarter*
Convolvulus arvense	*Field bindweed*
Datura stramonium	*Jimsonweed*
Echinochloa spp.	*Barnyard grass*
Helianthus annuus	*Sunflower*
Kochia scoparia	*Kochia weed*
Malva spp.	*Cheese weed*
Melilotus spp.	*Sweet clover*
Polygonum spp.	*Smart weed*
Rumex spp.	*Sorrel, curly leafed dock*
Solanum spp.	*Nightshades*
Sorghum halapense	*Johnson grass*
Sorghum vulgaer	*Sudan grass*
Zea mays	*Corn*
Glycine max	*Soybean*

Sources of poisoning:

Normally plants absorb nitrates from the soil converting them into plant proteins; however application of organic or inorganic nitrogenous fertilizers can result in excessive accumulation of nitrates in crop plants and common weeds. Livestock consuming these plants in quantity can develop nitrate poisoning. The potential for nitrate poisoning is increased when water sources for livestock also contain high levels of nitrates. Nitrate fertilizers are highly toxic chemicals capable of causing fatal poisoning in ruminants and horses that gain access to them accidentally. Many common weeds, forage crops, and cereal grain plants have the potential for accumulating nitrate under specific growing conditions (Table 1).

Concentration and severity of poisoning:

Nitrate levels in plants vary considerably depending on the plant species, stage of growth, water and organic content of the soil, and application of nitrogen fertilizers. Drought conditions, acidic soils, and soils deficient in sulfur, phosphorous, and molybdenum enhance nitrate accumulation in plants. Cool cloudy days enhance nitrate formation in plants because the light-and warmth-dependent enzyme, nitrate reductase, is inhibited, thus allowing nitrate to accumulate in the plant. Nitrate levels are therefore highest in plants at night and early morning when the nitrate-reducing enzymes are least active. Highest levels of nitrate tend to be found in the stems where nitrate reduction normally occurs, and not in the leaves. Nitrate does not accumulate in the flowers or fruits of plants and therefore nitrate poisoning is unlikely when seeds (corn, oats, and barley) are fed to livestock. Properly prepared silage from forage crops high in nitrates reduces the nitrate content by 60 percent, while there is little reduction of nitrate in dried hay. The application of herbicides such as 2,4-dichlorophenoxyacetic acid (2, 4-D), not only increases the nitrate content of plants, but also the palatability of the plants thereby increasing the potential for poisoning.

Species susceptible:

There is considerable variation between species in their susceptibility to nitrite poisoning. Pigs are the most susceptible, then, in order, cattle, sheep, and horses. Non-ruminants, such as horses and pigs, have no mechanism for converting nitrate to nitrite in their digestive tracts, so they are not susceptible to nitrite poisoning from excessive intake of nitrates. However, they are highly susceptible to poisoning from nitrite intake (for instance in mouldy hay) because they cannot convert the nitrite to ammonia. Sheep are more efficient at converting nitrite to ammonia, so this may be the reason why they are less susceptible to nitrite poisoning than cattle.

Hungry stock :

Hungry stock are at far greater risk than animals receiving regular and good fodder. This is because hungry stock consume more toxic feed, and, in the case of ruminants, their rumen microbes will not have had time to adapt to converting the nitrite to ammonia. For example, it takes about twice as much nitrate to kill a ruminant when the nitrate comes from forages that are eaten over a long period of time, compared to that which is consumed very quickly. Ruminant animals receiving carbohydrate-rich fodders tolerate high nitrate

and nitrite levels better than those that are not. This is because energy carbohydrates (grain) help rumen microbes convert nitrite to ammonia. Animals that are stressed or in poor health or condition will also be more susceptible to nitrate/nitrite poisoning.

Adaptation or acquaintance:

Frequent intake of small amounts of high-nitrate feed increases the total amount of nitrate that can be consumed by ruminant animals without adverse effects. This is because rumen microbes are adapted to deal with the increased nitrate content of the feed.

Mechanism of action:

In general, all animals are susceptible to nitrate poisoning if they consume enough of the chemical. In all animals, the nitrite ion readily reacts with hemoglobin in red blood cells, oxidizing it to form methemoglobin, which cannot transport oxygen. When over 30 to 40 percent of hemoglobin is converted to methemoglobin, clinical signs of poisoning become apparent. Death occurs as methemoglobin levels approach 80 percent. It also produces the signs of disturbed osmotic condition in the body.

Clinical Signs:

Signs develop within 6 to 8 hours of the consumption of a toxic dose of nitrate. Usually sudden death of one or more animals is a first sign of nitrate poisoning. If ruminants observed before death with nitrate poisoning, they exhibit drowsiness and weakness, followed by muscular tremors, increased heart and respiratory rates, staggering gait, and recumbency. Stress, forced exercise will increase the severity of clinical signs and hasten death. When the concentration goes up 20 percent or more, then it results into brownish discoloration of vaginal mucous membranes. This brownish discoloration occurs well before other clinical signs become evident, suggesting vaginal color changes are a good means of detecting nitrate poisoning before severe toxicity develops. Venous blood also has a chocolate brown discoloration. Depending on the quantity and rate of absorption of nitrite from the digestive tract, and the amount of stress to which the animal is subjected, death may occur within 2 to 10 hours. In chronic cases, abortion and fetal death may occur at any stage of gestation. Poisoning also affects metabolism of carotene and vitamin A, followed by reduction in feed intake in cattle.

Materials to be collected:

Forage, water source, rumen contents and tissues (heart, lungs, and kidneys), blood sample, aqueous humor and peritoneal and pericardial fluids, aqueous humor of eye from died animal, ocular fluid from an aborted fetus.

Diagnosis:

Based on

- Clinical signs, lesions, history of exposure to toxic plants and confirmation by demonstration of toxic levels of nitrate in the forage, water source, rumen contents and tissues (heart, lungs, and kidneys) of the animal.
- Methemoglobin analysis (greater than 40 percent of total hemoglobin) in the animal is diagnostic of nitrate poisoning.(Normal levels cattle- 0.1 to 0.2 g/dL)

- If a suspected tissue and plant sample is not possible to analyze immediately, then it should be frozen. In case of blood sample, it should be tested immediately, if not possible diluted with 1 part blood to 20 parts phosphate buffer (pH 6.6) and frozen.
- If the animal has been dead for several hours or more, then aqueous humor from the eyes taken for nitrate analysis as nitrate in the aqueous fluid is protected from autolytic changes that occur rapidly in other parts of the body after death.
- Detection of the serum nitrate levels can be obtained for about 24 hours postmortem remains diagnostically significant for as long as 60 hours.
- The normal level of nitrate in the ocular fluid of healthy cattle is 4 to 5 mg/L. Nitrate levels in aqueous humor of 20 to 40 ppm should be considered suspect, and over 40 mg/L (40 ppm) could be considered diagnostic of nitrate poisoning if corroborating clinical signs are seen and evidence of high nitrate levels is found in the forage and/or water.
- Ocular fluid from an aborted fetus is useful for determining if nitrate is the cause of abortion provided the levels detected are interpreted in light of forage and water nitrate levels to which the dam would have had access.
- As a general rule, levels of nitrate over 0.5 percent in forages and water levels exceeding 200 ppm are potentially hazardous to pregnant animals especially if fed continuously.
- Forages containing in excess of 1 percent nitrate dry matter should be considered toxic. Water levels of 1500 ppm or greater are potentially toxic to ruminants especially if consumed with forages high in nitrate.
- Nitrate and nitrite can be assayed in forage, rumen contents, and water using the diphenylamine test, ion-specific electrodes, and high-performance liquid chromatography (HPLC).
- Presumptive diagnosis of nitrite poisoning can be made in the field using diazotization urine test strips in the aqueous humor and peritoneal and pericardial fluids of animals suspected of nitrate poisoning.

Treatment:

1. Animals showing signs of nitrate poisoning should be handled carefully to avoid stress or excitement that will worsen the animal's respiratory distress. The suspected nitrate food source should be removed.
2. The preferred treatment for nitrate poisoning is methylene blue solution (IV). (reduces methemoglobin to hemoglobin thereby restoring normal oxygen transport by the red blood cells) Dose: methylene blue 4 to 15 mg/kg BW administered as a 2 to 4 percent solution. Cattle: 8 mg/kg BW, IV.

In sheep, half-life of methylene blue is about 2 hours, indicating that small doses of the drug can be repeated as needed every few minutes to reduce methemoglobinemia to the point that the animal is not in severe respiratory distress. Excessive administration of methylene blue to animals (Horses, Dogs, cats) other than ruminants will result in hemolytic anemia due to formation of Heinz bodies.

3. Animals with severe respiratory distress can

be given oxygen where possible to optimize oxygen saturation of remaining hemoglobin.

4. In severe cases, epinephrine (IV) may be administered to counter the acute hypotensive effects of the nitrite.
5. The administration of mineral oil (3 litres for a 500 kg cow) orally via stomach tube will counteract the caustic effect of the nitrates on the gastrointestinal system and will speed up the passage of the nitrates.
6. Several litres of cold water with added oral broad-spectrum antibiotics will further decrease nitrate reduction to nitrite by rumen microorganisms.
7. Similarly vinegar given orally via stomach tube will help prevent nitrate reduction in the rumen.

Prevention:

Avoid the excess application of manure or nitrate fertilizer to plants. It can be prevented if the nitrate levels in forages are predetermined and managed accordingly. Testing of forages such as Sudan grass, sorghum hybrids etc., if heavy nitrogen fertilization has been used or drought has affected the plants. Allow livestock to fresh nitrate free water at all times and mixed high nitrate forages with other forages or feedstuffs which are low in nitrate. Forages containing 1 percent nitrate or more should be fed cautiously to ruminants. There are several strategies that can be implemented if hay and other forages are found to contain high levels of nitrates. Ideally, hay that has more than 1 percent nitrate should be mixed with hay containing no nitrates so that the total nitrate level in the ration is below 1 percent. Feeding low-nitrate forage or hay before turning cattle onto forages containing higher levels of nitrate reduces the amount of nitrate consumed. Feeding high-nitrate forages to non pregnant cattle eliminates the risk of abortion. Products containing nitrate reducing bacteria (Propioni bacteria spp.) are available commercially for feeding to ruminants before exposing them to high-nitrate forages. This enables cattle to tolerate higher nitrate consumption.

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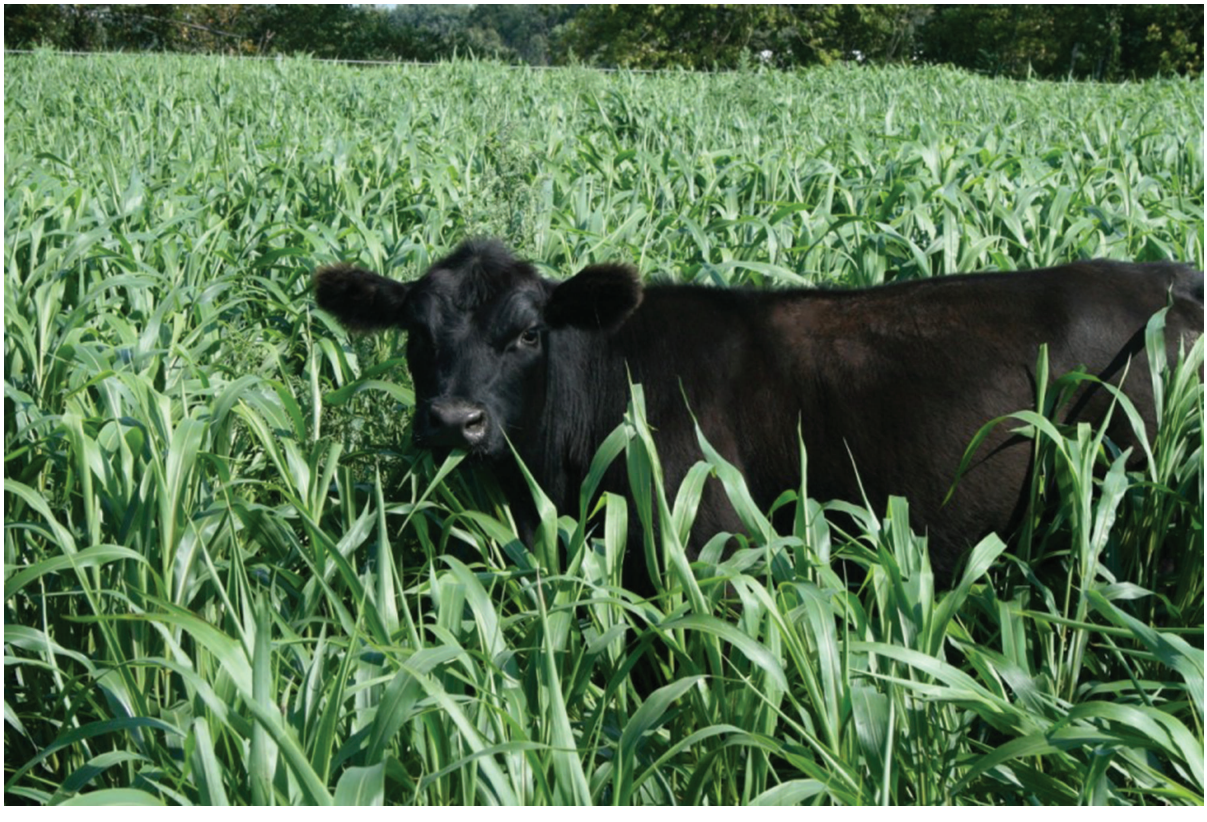

Heavy metals toxicity and their management in animals

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Lead poisoning

In Veterinary Medicine, lead poisoning is most common in dogs and cattle. Lead poisoning in other species is limited due to reduced accessibility, more selective feeding habits, or lower susceptibility. In cattle, many cases are associated with seeding and harvesting activities when used oil and battery disposal from machinery is handled improperly. Other sources of lead include paint, linoleum, grease, lead weights, lead shot, and contaminated foliage growing near smelters or along roadsides. Lead poisoning is also encountered in urban environments. Renovation of old houses that have been painted with lead-based paint has been associated with lead poisoning in small animals and children.

Absorbed lead enters the blood and soft tissues and eventually redistributes to the bone. The degree of absorption and retention is influenced by dietary factors such as calcium or iron levels. In ruminants, particulate lead lodged in the reticulum slowly dissolves and releases significant quantities of lead. Lead has a profound effect on sulfhydryl-containing enzymes, the thiol content of erythrocytes, antioxidant defenses, and tissues rich in mitochondria, which is reflected in the clinical syndrome. In addition to the cerebellar hemorrhage and oedema associated with capillary damage, lead is also irritating, immunosuppressive, gametotoxic, teratogenic, nephrotoxic and toxic to the hematopoietic system.

Clinical Findings:

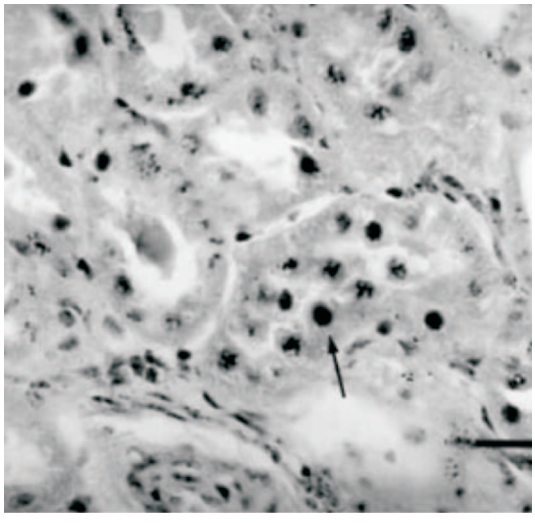
Acute lead poisoning is more common in young animals. The prominent clinical signs are associated with the GI and nervous systems. In cattle, signs that appear within 24-48 hr of exposure include ataxia, blindness, salivation, spastic twitching of eyelids, jaw champing, bruxism, muscle tremors and convulsions.

Subacute lead poisoning, usually seen in sheep or older cattle, is characterized by weight loss, depression, weakness, colic, diarrhoea, laryngeal or pharyngeal paralysis (roaring), and dysphagia that frequently results in aspiration pneumonia. In avian species, anorexia, ataxia, loss of condition, wing and leg weakness, and anemia are the most notable signs.

Lesion:

Animals that die from acute lead poisoning may have few observable gross lesions. Oil or flakes of paint or battery may be evident in the GI tract. The caustic action of lead salts causes gastroenteritis. In the nervous system, edema, congestion of the cerebral cortex, and flattening of the cortical gyri is present. Histologically, endothelial swelling, laminar cortical necrosis, and edema of the white matter may be evident. Tubular necrosis and degeneration and intranuclear acid-fast inclusion bodies may be seen in the kidneys.

Osteoporosis has been described in lambs. Placentitis and accumulation of lead in the fetus may result in abortion.



Section of bovine kidney. Note the acid-fast, intranuclear inclusion bodies (arrow) in the renal convoluted tubules. Ziehl-Neelsen staining. Bar=20 mm.

Diagnosis:

Lead levels in various tissues may be useful to evaluate excessive accumulation and to reflect the level or duration of exposure, severity, and prognosis and the success of treatment. Concentrations of lead in the blood at 0.35 ppm, liver at 10 ppm, or kidney cortex at 10 ppm are consistent with a diagnosis of lead poisoning in most species.

Hematologic abnormalities, which may be indicative but not confirmatory of lead poisoning, include anemia, anisocytosis, poikilocytosis, polychromasia, basophilic stippling, metarubricytosis, and hypochromia. Blood or urinary δ-aminolevulinic acid and free erythrocyte protoporphyrin levels are sensitive indicators of lead exposure but may not be reliable indicators of clinical disease. Radiologic examination may be useful to determine the magnitude of lead exposure.

Lead poisoning may be confused with other diseases that cause nervous or GI abnormalities. In cattle, such diseases may include polioencephalomalacia, nervous coccidiosis, tetanus, hypovitaminosis A, hypomagnesemic tetany, nervous acetonemia, arsenic or mercury poisoning, brain abscess or neoplasia, rabies, listeriosis, and Haemophilus infections. In dogs, rabies, distemper, and hepatitis may appear similar to lead poisoning.

Treatment:

If tissue damage is extensive, particularly to the nervous system, treatment may not be successful.

1. In livestock, calcium disodium EDTA (Ca-EDTA) is given IV or SC (110 mg/kg/day) divided into 2 treatments daily for 3 days; this treatment should be repeated 2 days later. In dogs, a similar dose divided into 4 treatments/day is administered SC in 5% dextrose for 2-5 days. After a 1-wk rest period, an additional 5-day treatment may be required if clinical signs persist. No approved veterinary product containing Ca-EDTA is commercially available at present.

2. Thiamine (2-4 mg/kg/day SC) alleviates clinical manifestations and reduces tissue deposition of lead. Combined Ca-EDTA and thiamine treatment appears to produce the most beneficial response.

3. D-Penicillamine can be administered PO to dogs (110 mg/kg/day) for 2 weeks. However, undesirable side effects such as emesis and anorexia have been associated with this treatment. D-Penicillamine is not recommended for livestock. Succimer (meso 2,3-dimercaptosuccinic acid, DMSA) is a chelating agent that has proven to be effective in dogs (10 mg/kg, PO, TID for 10

days) and is also useful in birds. Fewer side effects have been associated with DMSA as compared with Ca-EDTA.

4. Cathartics such as magnesium sulfate (400 mg/kg, PO) or a rumenotomy may be useful to remove lead from the GI tract. Barbiturates or tranquilizers may be indicated to control convulsions. Chelation therapy, in combination with antioxidant treatment, may limit oxidative damage associated with acute lead poisoning. Antioxidants such as –acetylcysteine (50 mg/kg, PO, SID) have been used in combination with DMSA.

5. Mobilization of lead at parturition, excretion of lead into milk, and lengthy EDTA withdrawal times in food-producing animals raise considerable controversy regarding the rationale for treatment from both public health and animal management perspectives.

Arsenic Poisoning

Arsenic poisoning in animals is caused by several different types of inorganic and organic arsenical compounds. Toxicity varies with factors such as oxidation state of the arsenic, solubility, species of animal involved, and duration of exposure. Therefore, the toxic effects produced by phenylarsonic feed additives and other inorganic and organic compounds must be distinguished.

Inorganic arsenicals poisoning:

These include arsenic trioxide, arsenic pentoxide, sodium and potassium arsenate, sodium and potassium arsenite and lead or calcium arsenate. Trivalent arsenicals, also known as arsenites, are more soluble and therefore more toxic than the pentavalents or

arsenate compounds. The lethal oral dose of sodium arsenite in most species is from 1-25 mg/kg. Cats may be more sensitive. Arsenates (pentavalents) are 5-10 times less toxic than arsenites. Poisoning is now relatively infrequent due to decreased use of these compounds as pesticides, ant baits, and wood preservatives. Arsenites are used to some extent as dips for tick control. Lead arsenate is sometimes used as a taeniacide in sheep.

Toxicokinetics and Mechanism of Action:

Soluble forms of arsenic compounds are well absorbed orally. Following absorption, most of the arsenic is bound to RBC; it distributes to several tissues, with the highest levels found in liver, kidneys, heart, and lungs. In subchronic or chronic exposures, arsenic accumulates in skin, nails, hooves, sweat glands, and hair. The majority of the absorbed arsenic is excreted in the urine as inorganic arsenic or in methylated form.

The mechanism of action of arsenic toxicosis varies with the type of arsenical compound. Generally, tissues that are rich in oxidative enzymes such as the GI tract, liver, kidneys, lungs, endothelium, and epidermis are

considered more vulnerable to arsenic damage. Trivalent inorganic and aliphatic organic arsenic compounds exert their toxicity by interacting with sulfhydryl enzymes, resulting in disruption of cellular metabolism. Arsenate can uncouple oxidation and phosphorylation.

Clinical Findings:

Poisoning is usually acute with major effects on the GI tract and cardiovascular system. Arsenic has a direct effect on the capillaries, causing damage to microvascular integrity, transudation of plasma, loss of blood, and hypovolemic shock. Profuse watery diarrhea, sometimes tinged with blood, is characteristic, as are severe colic, dehydration, weakness, depression, weak pulse, and cardiovascular collapse. The onset is rapid, and signs are usually seen within a few hours (or up to 24 hr). The course may run from hours to several weeks depending on the quantity ingested. In peracute poisoning, animals may simply be found dead.

Lesions:

In peracute toxicosis, no significant lesions may be seen. Inflammation and reddening of GI mucosa (local or diffuse) may be seen followed by edema, rupture of blood vessels, and necrosis of epithelial and subepithelial tissue. Necrosis may progress to perforation of the gastric or intestinal wall. GI contents are often fluid, foul smelling, and blood tinged; they may contain shreds of epithelial tissue. There is diffuse inflammation of the liver, kidneys, and other visceral organs. The liver may have fatty degeneration and necrosis, and the kidneys have tubular damage. In cases of cutaneous exposure, the skin may exhibit necrosis and be dry or leathery.

Diagnosis:

Chemical determination of arsenic in tissues (liver or kidney) or stomach contents provides confirmation. Liver and kidneys of normal animals rarely contain >1 ppm arsenic (wet wt); toxicity is associated with a concentration >3 ppm. The determination of arsenic in stomach contents is of value usually within the first 24-48 hr after ingestion. The concentration of arsenic in urine can be high for several days after ingestion. Drinking water containing >0.25% arsenic is considered potentially toxic, especially for large animals.

Treatment:

In animals with recent exposure and no clinical signs, emesis should be induced (in capable species), followed by activated charcoal with a cathartic (efficacy of charcoal in arsenic toxicosis remains to be determined) and then oral administration of GI protectants (small animals, 1-2 hr after charcoal) such as kaolin-pectin, and fluid therapy as needed. In animals already showing clinical signs, aggressive fluid therapy, blood transfusion (if needed), and administration of dimercaprol (British antilewisite (BAL), 4-7 mg/kg, IM, TID for 2-3 days or until recovery). In large animals, thioctic acid (lipoic acid or α-lipoic acid) may be used alone (50 mg/kg, IM, TID, as a 20% solution) or in combination with dimercaprol (3 mg/kg, IM, every 4 hr for the first 2 days, QID for the third day, and BID for the next 10 days or until recovery). In large animals, the efficacy of dimercaprol alone is questionable. Sodium thiosulfate has also been used, PO, at 20-30 g in 300 mL of water in horses and cattle, one-fourth this dose in sheep and goats, and 0.5-3 g in small animals or as a 20% solution, IV, at 30-40

mg/kg, 2-3 times/day for 3-4 days or until recovery. The water-soluble analogs of dimercaprol, 2, 3-dimercaptopropane-1-sulfonate (DMPS) and dimercaptosuccinic acid (DMSA), are considered to be less toxic and more effective and could be given orally. D-Penicillamine has been reported to be an effective arsenic chelator in humans. It has a wide margin of safety and could be used in animals at 10-50 mg/kg, PO, 3-4 times/day for 3-4 days. Supportive therapy may be of even greater value, particularly when cardiovascular collapse is imminent, and should involve IV fluids to restore blood volume and correct dehydration. Kidney and liver function should be monitored during treatment.

Organic Arsenicals

Phenylarsonic organic arsenicals are relatively less toxic than inorganic compounds or aliphatic and other aromatic organic compounds.

Aliphatic organic arsenicals include cacodylic acid and acetarsonic acid. These are generally used as stimulants in large animals, but their use is no longer common. Some aliphatic arsenicals such as monosodium methanearsonate (MSMA) and disodium methanearsonate (DSMA) are occasionally used as cotton defoliants or crabgrass killers. Persistence of MSMA or DSMA in the soil and their tendency to accumulate in plants creates a potential for arsenic poisoning, especially in grazing animals. Clinical signs, lesions, and treatment of aliphatic organic arsenicals are similar to those of inorganic arsenicals.

Aromatic organic arsenicals include trivalent phenylorganicals such as thiacetarsamide and arsphenamine for the treatment of adult heartworms in dogs and pentavalent compounds such as phenylarsonic acids and their salts. Thiacetarsamide and arsphenamine are no longer used commonly, especially since the recent introduction of melarsomine dihydrochloride.

Phenylarsonic compounds are used as feed additives to improve production in swine and poultry rations and also to treat dysentery in pigs. The 3 major compounds in this class are arsanilic acid, roxarsone (4-hydroxy-3-nitrophenylarsonic acid), and nitarsone (4-nitro-phenylarsonic acid).

Etiology:

Toxicosis results from an excess of arsenic-containing additives in pig or poultry diets. Severity and rapidity of onset are dose-dependent. Signs may be delayed for weeks after incorporation of 2-3 times the recommended (100 ppm) levels or may occur within days when the excess is >10 times the recommended levels. Chickens are tolerant of arsanilic acid; however, roxarsone can produce toxicosis in turkeys at only twice the recommended dose (50 ppm). Roxarsone also has a higher toxicity in pigs as compared with other phenylarsonics.

Clinical Findings and Diagnosis:

The earliest sign in pigs may be a reduction in weight gain, followed by incoordination, posterior paralysis, and eventually quadriplegia. Animals remain alert and maintain good appetite. Blindness is characteristic of arsanilic acid intoxication but not of other organic arsenicals. In ruminants, phenylarsonic toxicosis is similar to inorganic arsenic poisoning. There are usually no specific lesions present in phenylarsonic poisoning. Demyelination and gliosis of peripheral nerves, the optic tract, and

optic nerves are usually seen on histopathology. Analysis of feed for the presence of high levels of phenylarsonics confirms the diagnosis.

Phenylarsonic poisoning in pigs should be differentiated from salt poisoning, insecticide poisoning, and pseudorabies. In cattle, arsenic poisoning should be differentiated from other heavy metal (lead) poisoning, insecticide poisoning, and infectious diseases such as bovine viral diarrhea.

Treatment and Prognosis:

There is no specific treatment, but the neurotoxic effects are usually reversible if the offending feed is withdrawn within 2-3 days of onset of ataxia. Once paralysis occurs, the nerve damage is irreversible. Blindness is also usually irreversible, but animals retain their appetite, and weight gain is good if competition for food is eliminated. Recovery may be doubtful when the exposure is long and the onset of intoxication slow.

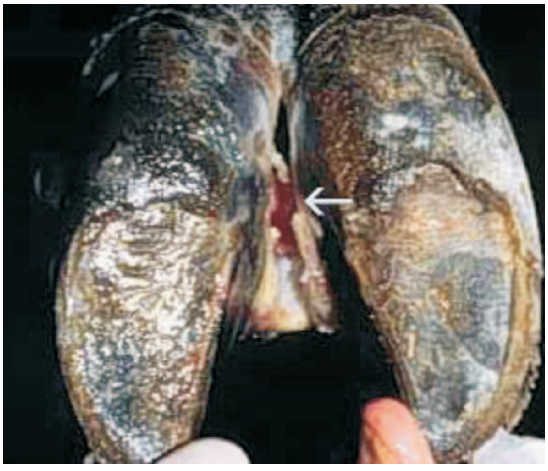
Selenium Toxicity

Selenium is an essential element that has a narrow margin of safety. Feed supplements containing 0.1-0.3 ppm selenium are added to the diet to prevent deficiency diseases such as white muscle disease in cattle and sheep, hepatosis dietetica in pigs, and exudative diathesis in chickens. The maximum tolerable level for selenium in most livestock feed is considered to be 2 ppm or as high as 5 ppm, although some believe that levels as high as 4-5 ppm can inhibit growth.

Selenium is a component of the glutathione peroxidase enzyme that acts as an antioxidant during release of energy. In excess, selenium has 2 general effects: the direct inhibition of cellular oxidation/reduction reactions, and the replacement of sulfur in the body. The inhibition of numerous cellular functions by high levels of selenium results in acute generalized cytotoxicity. The replacement of sulfur by chronic intake of selenium leads to altered structure and function of cellular components. Altered sulfur-containing amino acids (methionine, cystine) affect cell division and growth. Especially susceptible are the cells that form keratin (keratinocytes) and the sulfur-containing keratin molecule. Selenium therefore weakens the hooves and hair, which tend to fracture when subjected to mechanical stress.

Etiology:

All animal species are susceptible to selenium toxicosis. However, poisoning is more common in forage-eating animals such as cattle, sheep, and horses that may graze selenium-containing plants. Plants may accumulate selenium when the element is found at high levels—generally in alkaline soil with little rainfall (<50 cm). Selenium accumulating plants have been categorized. Obligate indicator plants require large amounts of selenium for growth and contain high concentrations (often >1,000 ppm). Facultative indicator plants absorb and tolerate high levels of soil selenium accumulating up to 100 ppm under these conditions, but they do not require selenium. Nonaccumulator plants passively absorb low levels of selenium (1-25 ppm) from the soil. Poisoning may also occur in swine and poultry consuming grain raised on seleniferous soils or, more commonly, due to error in feed formulation. Selenium toxicosis after ingestion of selenium-containing shampoos or excess selenium tablets is rare in pets. Several factors



are known to alter selenium toxicity; however, in general, a single acute oral dose of selenium in the range of 1-5 mg/kg is lethal in most animals. Parenteral selenium products are also quite toxic, especially to young animals, and have caused deaths in baby pigs, calves, and dogs at doses as low as 1.0 mg/kg.

Types of Selenium toxicity-

1. Acute: selenium poisoning due to consumption of plants with levels >50 ppm (dosages 3-20 mg/kg) is rare but has caused large losses in cattle, sheep, and pigs. Animals usually avoid these plants because of their offensive odor; however, when pasture is limited, accumulator plants may be the only food available. Young animals are most susceptible to acute parenteral selenium toxicosis with dosages of 0.2-0.5 mg/kg. Clinical signs are different from those of chronic selenosis and are characterized by abnormal behavior, respiratory difficulty, gastrointestinal upset, and sudden death. Abnormal posture and depression, anorexia, unsteady gait, diarrhea, colic, increased pulse and respiration rates, frothy nasal discharge, moist rales, and cyanosis may be noted.

Death usually follows within a few hours of consumption or injection. The major lesions are lung edema and congestion, and necrosis of multiple organs, including lung, liver, and kidney. Sheep usually do not show these signs, but instead become depressed and die suddenly. Blood selenium concentration in acute poisoning is much higher than in chronic poisoning. In acute cases, blood selenium may reach 25 ppm. Treatment consists of symptomatic and supportive care. Acetylcysteine to boost glutathione levels is beneficial.

2. Subchronic selenium toxicosis: Pigs fed a diet supplemented with selenium >20-50 ppm for >3 days develop a subchronic selenium toxicosis characterized by neurologic abnormalities. Animals are initially ataxic and uncoordinated followed by anterior paresis, then quadriplegia. Pigs continue to eat. The hooves show breaks and impaired growth similar to those seen in cattle; alopecia is observed. In sows, conception rate decreases and number of pigs born dead increases. Lesions of subchronic toxicosis include focal symmetric poliomyelomalacia, which is most prominent in the cervical and thoracic spinal cord. Death may result from complications of permanent paralysis. Hoof and hair damage is similar to but in most cases less severe than that observed in chronic selenium toxicosis. Treatment is similar to that for chronic toxicosis, but spinal lesions are usually permanent.

3. Chronic selenium poisoning: Chronic selenium poisoning usually develops when livestock consume seleniferous forages and grains containing 5-50 ppm of selenium for many weeks or months. Naturally occurring seleno-amino acids in plants are readily absorbed. Until recently, 2 types of chronic

selenium poisoning were recognized—alkali disease and blind staggers. Blind staggers is no longer believed to be caused by selenium but by sulfate toxicity due to consumption of high-sulfate alkali water. Excess sulfate (>2% of diet) leads to polioencephalomalacia and the classical signs of blind staggers. Animals consuming milk vetch (*Astragalus bisulcatus*) have demonstrated clinical signs similar to blind staggers. Although milk vetch contains high levels of selenium, evidence now indicates that the alkaloid swainsonine in milk vetch, responsible for locoism, produces the signs.

Clinical Findings:

Alkali disease has been reported in cattle, sheep, and horses. Affected animals are dull, emaciated, and lack vitality. The most distinctive lesions are those involving the keratin of the hair and hooves. The animal has a rough hair coat and the long hairs of the mane and tail break off at the same level giving a "bob" tail and "roached" mane appearance. Abnormal growth and structure of horns and hooves results in circular ridges and cracking of the hoof wall at the coronary band. Extremely long, deformed hooves that turn upwards at the ends may be seen. Subsequent lameness is compounded by degeneration of joint cartilage and bone. Reduced fertility and reproductive performance occurs especially in sheep.

Reproductive performance may be depressed with a dietary level of selenium lower than that required to produce typical signs of alkali disease. Other lesions may include anemia, liver cirrhosis and ascites, and atrophy of the heart. Birds also may be affected with chronic selenium toxicosis. Eggs with >2.5 ppm selenium from birds in high selenium areas have low hatchability, and the embryos are usually deformed. Teratologic effects include underdeveloped feet and legs, malformed eyes, crooked beaks, and ropy feathers. This has been a problem with waterfowl in southern California, where selenium was leached by agricultural water and concentrated in lakes by runoff.

Blood levels of selenium in chronic cases are usually 1-4 ppm. Other changes in blood include decreased fibrinogen level and prothrombin activity; increased serum alkaline phosphatase, ALT, AST, and succinic dehydrogenase; and reduced glutathione. Hair may have >5 ppm selenium in chronic poisoning. A "garlicky" odor on the animal's breath may be noted.

Treatment and Control:

There is no specific treatment for selenium toxicosis. Eliminating the source and exposure and symptomatic and supportive care of the animal should be started as soon as possible. Addition of substances that antagonize or inhibit the toxic effects of selenium in the diet may help reduce the risk of selenium toxicosis. A high protein diet, linseed oil meal, sulfur, arsenic, silver, copper, cadmium, and mercury have reduced selenium toxicity in laboratory animals, but their use under field conditions is limited. Addition of arsenic salt at 0.00375% to enhance biliary excretion of selenium or use of a high-protein diet to bind free selenium may help reduce incidence of selenium poisoning in cattle. Soil and forages should be tested regularly in high-selenium areas.

Squamous cell carcinoma of eye in cattle

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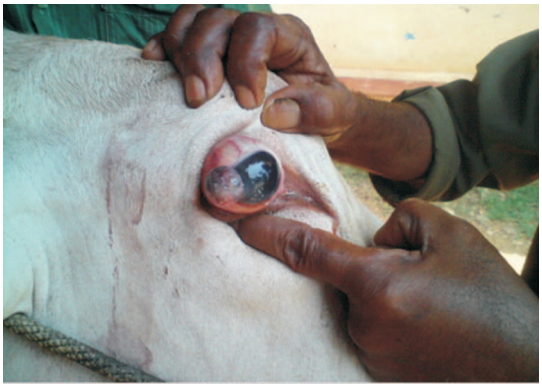

Fig 1: Eyeball exposed after the nerve blocks for examination indicating squamous cell carcinoma of the eye at the corneoscleral junction in a bullock

Introduction :

Squamous cell carcinoma of the eye is the most common neoplasm of cattle. It results in significant economic loss due to condemnation at slaughter and a shortened productive life. It occurs more frequently in the *Bos taurus* than in the *Bos indicus* breeds, and it is seen most often in Herefords, less often in Simmentals and Holstein-Friesians, and rarely in other breeds. In Indian breeds of cattle, Hallikar, Amrithmahal, Deoni and Khilar have been observed to be susceptible. Incidence is reported more in bullocks than in cows. The peak age of incidence is 8 yr and older cattle are more prone. The disease is uncommon in cows under 5 years of age. Males and females are equally susceptible.

Etiology :

The cause is multifactorial, with heritability, sunlight, nutrition, eyelid pigmentation, and perhaps viral involvement playing roles. The medial and lateral limbal regions (corneoscleral junction) are affected most frequently, but the eyelids, conjunctivae, and nictitating membrane may also be affected. Bilateral involvement varies, but can be as high as 35 percent. Ultraviolet radiation and a high plane of nutrition are contributing factors. The viruses of infectious bovine rhinotracheitis and papilloma have been isolated from the neoplasms, but their significance in etiology is unknown.

The lesions usually begin as benign, smooth, white plaques on the conjunctival surfaces; they may progress to a papilloma and then to a squamous cell carcinoma or go directly to the malignant stage. Diagnosis usually is made by the typical clinical appearance but can be confirmed rapidly by cytologic examination of impression smears.

Factors affecting incidence of squamous cell carcinoma of eye

Breed

The disease occurs in cattle that have areas of unpigmented skin or conjunctiva. Breeds such as Hereford, Poll Hereford and white-faced Friesian are most susceptible and Hereford crosses are less susceptible than purebreds. This disease occurs in the unpigmented areas of the

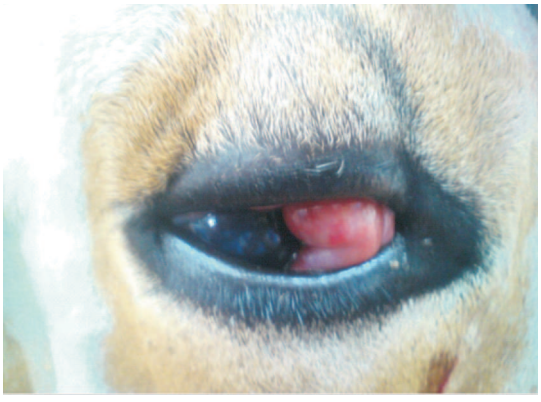

Fig 2: Squamous cell carcinoma of the third eyelid in a HF cross bred cattle.

body surface. Lesser incidence of the eye cancer is reported in breeds with fully pigmented skin and conjunctiva.

Strain and sire

There is a variation in the susceptibility to the disease with strain and sire and the heritability of eye cancer has been estimated as moderate at 0.4.

Virus

Papillomavirus is isolated from some cases of squamous cell carcinoma of eye in bullocks. But, there is no specific research to reveal any definite association between papillomavirus and the development of these tumours.

Pregnancy

Squamous cell carcinoma of eye usually develops or progresses more rapidly in the latter half of pregnancy. This may be due to increased stress or the suppressed immunity associated with pregnancy.

Nutrition

It has been reported that a high level of nutrition and increased growth rate increases the risk of eye cancer. Some reports suggest that iodine deficiency may be associated with an increased risk of cancer eye. Squamous cell carcinoma of eye is reported more common during and after droughts. It is because of longer hours of grazing under the sun and stress on the animal reducing the immunity.

Ultraviolet solar radiation

Increased ultraviolet (UV) radiation is thought to predispose cattle to squamous cell carcinoma of eye. Animals that are located at hilly areas and that are exposed to the sun for longer hours in a day are supposed to be more susceptible to this disease.

Pigmentation

Cattle that are having unpigmented skin around the eyes are more susceptible to squamous cell carcinoma of eye. Cancers usually start on unpigmented skin, but can then spread to pigmented areas. Eyelid pigment is present at birth and is easily assessed. Eyeball pigment usually takes more than 5 years to develop fully. Pigmentation of the eyelids and pigmentation of the eyeball are highly heritable and appear to be genetically related.

Location of eyeball

It is likely that protruding eyeballs are more susceptible than 'hooded' eyes, which are more protected from sunlight.

Progression of carcinoma

All eye cancers develop from precursor lesions,

but less than half of these lesions develop into cancer. The precursor lesion can be a plaque (slightly elevated, flat, opaque area on the eyeball) or a papilloma (a wart-like growth protruding from the eyeball).

As the disease progresses from the precursor stage to the cancer stage, the tumour becomes ulcerated. Bleeding and weeping are common. Bacteria invade the lesion, which usually develops externally into a festering, foul-smelling growth. The cancerous tissue also grows inwards, invading the deeper tissues behind the eye. It can progress to the lymph nodes of the head and then to body organs such as the lungs and liver. Cancers that begin in the third eyelid or outer eyelids usually invade the deeper tissues more quickly than do those that start on the eyeball. Untreated cattle may live for 2–5 years after the first appearance of a cancerous lesion, although such cattle may become weak and emaciated within 6 months, indicating the involvement of internal organs. It is more common for only one eye to be affected, but occasionally the condition may occur in both the eyes.

Diagnosis :

Differentiation from similar lesions can only be achieved by proper laboratory examination of tissues. The squamous cell carcinoma must be differentiated clinically from pink eye and its complications which results in excessive lacrimation and purulent material, so also from Lymphoma of the periorbital tissues which usually manifests itself as exophalthmos.

Treatment

Surgical excision is indicated for small lesions or for debulking the larger lesions before cryotherapy or hyperthermia. Superficial keratectomy can be used to excise the limbal plaques, papillomas, and squamous cell carcinomas. After superficial keratectomy and tumor removal, the site is rubbed with silver nitrate crystals or with liquid nitrogen. Recurrence of the condition is rare after the above treatment. For advanced lesions confined to the globe, enucleation is recommended. When adjacent tissues are affected, removal of the globe and all orbital contents (exenteration) should be performed. Immunotherapy is still experimental, and the resulting tumor regression may be temporary. Radiation therapy is not practical in the field but may be an option for valuable animals.

Vaccination:

An experimental vaccine made from cancer tissue to elicit an immune response has been reported to cause regression and sometimes complete disappearance of existing eye tumours, even in some moderately advanced cases. However, the vaccine is quite difficult to make, and requires large amounts of tumour material to be collected from other animals. Development of a vaccine for treatment purposes is therefore not considered to be a commercially viable proposition. Since it is undesirable to keep animals that are predisposed to eye cancer, vaccination is not recommended as a management tool.

Control

Squamous cell carcinoma of eye can be controlled by selective breeding. Selection for pigmented eyelids is most effective and can

result in rapid improvement in the herd. This selection is recommended for all Hereford herds. Eyeball pigmentation develops slowly, but apparently is genetically linked to eyelid pigmentation, so selection for one, in effect, selects for both. Lower-lid pigmentation is more important than pigmentation of the upper lid. The intensity of pigmentation is not important as long as it is darker than just a pale brown.

Unlike eyeball pigment, third-eyelid pigment is not affected by selection for external eyelid pigment. Third-eyelid cancers account for about 10% of cancers in Herefords. They are easily removed by surgery in the early stages. Treated cows and their progeny should be culled as soon as practicable to select against this form of cancer eye.

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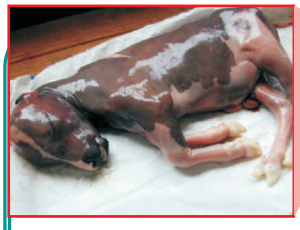

Brucellosis : A hurdle in Farm Livestock Development

Prevalence and Economic Losses

Prevalence of brucellosis in India with respect to different species of Brucella and their biotypes in various species of livestock (cattle, buffaloes, goats, sheep, pigs) in different geographical zones of India under varying sociocultural and economic conditions, so also under different managemental (organized / unorganized) practices is yet to be worked out systematically.

However, available prevalence studies and epidemiological evidences suggest that Brucellosis is prevalent in India, in all domestic livestock, in all the States with wide variation in prevalence, varying from as low as 0.13% to as high as 44%.

The economic losses due to Brucellosis are attributed mainly to loss of milk production subsequent to abortion, loss of milk and meat producing animals, prolonged inter-calving periods, temporary or permanent loss of fertility in females and males, so also, loss in man - days, if there are incidences of human brucellosis on livestock farms.

The estimated losses due to brucellosis cost India at-least Rs. 350 millions every year

Management of Colic in Equines

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Colic is a symptom, not a disease. The term colic can encompass all forms of gastrointestinal conditions which cause pain as well as other causes of abdominal pain not involving the gastrointestinal tract. The most common forms of colic are gastrointestinal in nature and are most often related to colonic disturbance. Among domesticated horses, colic is the leading cause of death.

Classification of colic:

Displacement colic – The small intestine is suspended in the abdominal cavity by the mesentery and is free floating in the gut. This mobility can predispose the small intestine to become twisted. In addition, both the small and large intestine can become displaced in the abdominal cavity, causing both pain and restricted blood flow. Displacement colic needs immediate surgical treatment.

Impaction colic – The flexures and diameter shifts can be the sites for impactions, where a firm mass of feed or foreign material blocks the intestine (including the cecum). Impactions can be induced by coarse feed stuff, dehydration or accumulation of foreign material like sand.

Gas colic – Gas can accumulate in the stomach as well as the intestines. As gas builds up, the gut distends, causing abdominal pain. Excessive gas can be produced by bacteria in the gut after ingestion of large amounts of grain or moldy feeds.

Spasmodic colic - Defined as painful contractions of the smooth muscle in the intestines. Spasmodic colic has been compared to indigestion in people and over excitement can trigger spasmodic colic.

Stomach distention – The small capacity of the horse's stomach makes it susceptible to distension when large amounts of grain are ingested in a single meal. There is the potential for the stomach to rupture which is fatal.

Enteritis – Inflammation of the intestine possibly due to bacteria, grain overload or tainted feed. Horses with enteritis may also have diarrhea. Enteritis is often hard to diagnose and may present itself similar to displacement or impaction colic.

Pelvic flexure impaction - This is caused by an impaction of food material (Water, Grass Hay, Grain) at a part of the large bowel known as the pelvic flexure of the left colon where the intestine takes a 180 degree turn. This condition could be diagnosed on rectal examination by a Veterinarian.

Ileal impaction - The ileum is the last part of the small intestine that ends in the cecum. Ileal impaction can be caused by obstruction of

ingesta. Other causes can be obstruction by ascarids (*Parascaris equorum*) or tapeworm (*Anoplocephala perfliata*).

Sand impaction - This is most likely to occur in horses that graze on sandy or heavily grazed pastures, leaving only dirt to ingest. The ingested sand or dirt accumulates in the pelvic flexure, right dorsal colon and the cecum of the large intestines. Horses should not be fed from the ground in areas where sand, dirt and silt are prevalent, although small amounts of sand or dirt will still be ingested by grazing.

Enterolith - Enteroliths in horses are round balls of mineral deposits often formed around piece of ingested foreign material, such as sand or grave. Once a horse is diagnosed with colic due to enterolith, it usually requires surgery to correct the condition.

Large roundworms - Occasionally large number of roundworms can cause obstruction. This is most commonly seen in young horses as a result of a very heavy infestation of *Parascaris equorum* that can subsequently cause a blockage and rupture of the small intestine.

Tapeworms - The most common species of tapeworm causing colic in the equine is *Anoplocephala perfoliata*. However, a 2008 study in Canada indicated that there is no connection between tapeworms and colic.

Torsion - Torsion is common after foaling, but can also occur spontaneously. It is most likely to be either small intestine or part of the colon. Occlusion of the blood supply means that it is a painful condition causing rapid deterioration and requiring emergency surgery.

Intussusception - Intussusception is a form of colic in which a piece of intestine "telescopes" within a portion of itself. It most commonly happens in the small intestine of young horses and requires urgent surgery. The intestine becomes trapped and surgery is the only available treatment.

Strangulating lipoma (Peduculated lipoma)- Mostly seen in old horses, benign fatty tumors known as lipomas can form on the mesentery. The tumor forms a button that latches onto the stalk of the tumor, locking it on place, and requiring surgery for resolution.

Mesenteric rent entrapment - Occasionally, a small rent (hole) can form in the mesentery, through which a segment of bowel can occasionally enter. As the bowel enlarges, it becomes less and less likely to be able to exit the site of entrapment. This problem also requires surgical correction.

Gastric ulceration - Gastric ulceration is fairly common in young horses. Risk factors include confinement, infrequent feedings, a high proportion of concentrate feeds, excessive non-steroidal anti-inflammatory drug use, and the stress of shipping and showing. Dietary management is critical. Bleeding ulcers leading to stomach rupture are rare.

Pathophysiology of equine colic

The primary pathophysiological abnormality caused by obstruction is related to the trapping of fluid within the intestine proximal to the obstruction. This is due to the large amount of fluid produced in the upper gastro-intestinal tract and the fact that this is primarily re-

absorbed in parts of the intestine downstream from the obstruction. The first problem with this degree of fluid loss from circulation is one of decreased plasma volume, leading to a reduced cardiac output, and acid-base disturbances. There also occur effects on the intestine itself, which becomes, distended due to the trapped fluid, and by gas production from bacteria. It is this distension, and subsequent activation of stretch receptors within the intestinal wall, that leads to the associated pain. With progressive distension of the intestinal wall, occlusion of blood vessels occurs, firstly veins, then arteries. The difference in time to onset of occlusion is due to the relatively more rigid walls of arteries compared to veins. This impairment of blood supply leads firstly to hyperaemia and congestion, and ultimately to ischemic necrosis and cellular death. The poor blood supply also has effects on the vascular endothelium, leading to an increased permeability. This results initially to leakage of plasma, and eventually blood into the intestinal lumen.

Diagnosis of colic

A diagnosis can be made and appropriate treatment begun only after thoroughly examining the horse, considering the history of any previous problems or treatments, determining which part of the intestinal tract is involved, and identifying the cause of the particular episode of colic.

The history of the present colic episode and previous episodes, if any, must be ascertained to determine if the horse has had repeated or similar problems, or if this episode is an isolated event. It is also critical to determine the horse's deworming history (schedule, treatment dates, drugs used), when the teeth were floated last, if any changes in feed or water supply or amount have occurred.

The physical examination should include assessment of the cardiopulmonary and GI systems. The oral mucous membranes should be evaluated for colour, moistness, and capillary refill time. The mucous membranes may become cyanotic or pale in acute cardiovascular compromise and eventually hyperemic or muddy as peripheral vasodilation develops later in shock. The capillary refill time (normal ~1.5 sec) may be shortened early but usually becomes prolonged as vascular stasis (venous pooling) develops. The heart rate increases due to pain, hemoconcentration, and hypotension; therefore, higher heart rates have been associated with more severe intestinal problems (strangulating obstruction). An important aspect of the physical examination is passing a nasogastric tube. Passing a stomach tube may, therefore, save the horse's life and assist in diagnosis of these conditions.

The abdomen and thorax should be auscultated, and the abdomen percussed. Gas sounds may indicate ileus or distention of a viscus. Fluid sounds may indicate impending diarrhea associated with colitis. A complete lack of sounds is usually associated with adynamic ileus or ischemia. Percussion will assist in identifying a grossly distended segment of intestine (cecum on right, colon on left) that may need to be trocarized.

The most definitive part of the examination is the rectal examination. The intestines should be palpated for size, consistency of contents (gas, fluid, or impacted ingesta), distention, edematous walls, and pain on palpation.

A sample of peritoneal fluid (obtained via paracentesis performed aseptically on midline) often reflects the degree of intestinal damage. Normal peritoneal fluid is clear to yellow, contains <5,000 WBC/μL, most of which are mononuclear cells, and <2.5 g of protein/dL.

The age of the horse is important because a number of age-related conditions cause colic. Ultrasonographic evaluation of the abdomen may help clinicians differentiate between diseases that can be treated medically and those that require surgery.

Clinical signs

The common clinical signs of colic include

- Kicking at the belly. Unless the horse is being bothered by flies, this is a fairly specific symptom.
- Turning to look at and/or biting at the belly or flank. The horse may rest quietly, or may alternate between lying flat and lying on his sternum.
- Restlessness. Horse may lie down and get up repeatedly
- Nosing at water but not drinking.
- Grunting or groaning. Some horses may lift their lip or grind their teeth.
- Pawing at the ground. Indicate more severe pain
- Rolling and/or thrashing when down. This indicates severe pain.
- Change in faeces. This includes no/less quantity, diarrhea, change in size of the fecal balls, faeces covered with mucus
- Excessive gas production, abnormal abdominal sounds, or a complete lack of sound.
- Abnormal postures. Standing stretched out or a horse sitting on his haunches like a dog.
- Changes in mouth and gum color. Abnormally pale, or abnormally red or dark
- Nonspecific signs that tell the horse is distressed. These include depression, poor appetite, sweating, increased pulse rate, breathing more rapidly.

Treatment of colic

Almost all the cases require some form of medical treatment, but only those with certain mechanical obstructions of the intestine need surgery. The type of medical treatment is determined by the cause of colic and the severity of the disease. In some instances, the horse may be treated medically first and the response evaluated; this is particularly appropriate if the horse is mildly painful and the cardiovascular system is functioning normally. Pain relief, fluid therapy, protection against bacterial endotoxin, intestinal lubricants and laxatives, larvicidal deworming are the main things to be taken in considerations.

Pain Relief

An analgesic that has the fewest adverse effects and causes the least alteration in the horse's attitude should be selected. Flunixin meglumine may mask the early signs of conditions that require surgery and, therefore, must be used carefully in horses with colic. The most commonly used sedative for colic is xylazine, an α2-agonist. Detomidine, a more potent α2-agonist that is much longer acting, is used successfully under similar circumstances. Butorphanol is frequently combined with an α2-agonist to produce a more prolonged period of analgesia.

Fluid Therapy

When IV fluids are needed but the clinical signs are mild to moderate, the horse is usually given 8–10 L of a sterile replacement fluid that contains electrolytes in concentrations that normally exist in the blood. This volume is administered over 1–2 hr, and the horse is reevaluated to determine if additional fluids are needed. Horses in circulatory shock require much larger volumes of IV fluids, given as rapidly as possible; up to 20 L in 1 hr may be needed to reestablish tissue perfusion. In severe cases, hypertonic saline (7% NaCl) may be given to rapidly increase plasma volume. Depending on the cause of colic, IV fluids may be needed for several days until intestinal function has returned, electrolyte concentrations are balanced, and the horse can maintain its fluid needs by drinking. Under such circumstances, the daily IV fluid requirements may range from 30 to 100 L.

Protection Against Bacterial Endotoxin

Minimizing the inflammatory responses to endotoxemia is a vital part of colic therapy. Flunixin meglumine reduces the cellular production of prostaglandins and can help prevent some of their effects. Flunixin @ 0.25 mg/kg can be administered without masking clinical signs associated with conditions that require surgery. Polymyxin B has been evaluated in several experimental studies of endotoxemia and is being used in clinical cases at 1,000–5,000 U/kg, twice or thrice a day.

Intestinal Lubricants and Laxatives

Mineral oil is administered through a nasogastric tube, up to 4 L, once or twice a day, until the impaction is resolved. Dioctyl sodium sulfosuccinate (DSS) is more effective than mineral oil in softening impactions; however, it may interfere with the normal fluid absorptive functions of the colon and can be toxic. Thus, DSS can be given safely only in small quantities 2 times 48 hr apart. Horses that live in a sandy environment may be given psyllium powder, 400 g/500 kg/day in their feed for 7 days. Horses with extremely hard impactions can be treated with magnesium sulphate but it may increase the risk of dehydration and diarrhoea.

Larvicidal Deworming

Ivermectin and moxidectin, can be used against migrating *Strongyles vulgaris* larvae. Fenbendazole kills migrating strongyles if given at twice the recommended dosage daily for 5 days.

Surgical Management

Requirements: Equine surgical table, Facility of large animal anaesthesia machine with ventilator, suction apparatus, colon tray, surgical instruments, sleeves etc.

Anaesthesia: General anaesthesia induced with intravenous agents and maintained with halothane/isofluoran etc is indicated. Where muscle relaxants are used, positive pressure ventilation is mandatory.

Positioning: Dorsal recumbency with adequate support for head and limbs to avoid post operative complications.

Surgical approach: Ventral celiotomy through midventral incision is standard surgical approach. Other approaches such as flank approach are also used for some conditions. After opening of abdomen, the gas from the

intestine must be removed by suction needle making a valve at the band or the mucosal surface. Systematically the intestine should be explored to locate the obstruction. It is preferred to open the cecum and drain the contents. The obstructing mass or fecolith can be removed. The intestine may be sutured with inversion sutures with synthetic absorbable suture material. The small intestine can be resected and end to end anastomosis or side to side anastomosis to give more lumen for passage of intestine contents may be preferred. The LDDLC or RDDLC can be corrected by bringing the colon to its normal anatomical position. The abdomen can be closed by continuous sutures or interrupted sutures. Intestine should be thoroughly washed with saline and abdomen lavaged adequately before closure of abdomen. The horse should be placed in a well padded stall with soft bedding during recovery and monitored for smooth recovery. Antibiotic cover and NSAIDs should be administered. Abdominal drains may be provided to drain peritoneal fluid.

Post operative management: Post operative management includes regular monitoring of vital parameters, administration of fluids (balanced solution without lactate), antibiotics, NSAIDs, adequate wound protection, padded bedding. Concentrates may be avoided during recovery, however, greens can be given. Major complications of equine colic surgery include peritonitis, recurrence, laminitis and complications related with wound and recovery of animal from anaesthesia.

Prognosis

A large retrospective study in the USA documented an overall survival rate of 60% for horses with colic, and a survival rate of 50% for those horses undergoing abdominal surgery. Survival rates for horses with strangulating obstruction and inflammatory diseases were only 24% and 42%, respectively. There have been reports documenting survival rates of 70% for horses requiring resection of strangulated small intestine or correction of large colon volvulus.

Prevention

- The horse should be fed on a regular schedule even on the weekends.
- Sudden changes in the horse's diet should not be made.
- A clean fresh water supply should always be available.
- The feed boxes and hay racks as well as the feedstuffs be kept clean and free of mold and dust.
- The teeth be checked frequently for dental problems that may cause chewing issues.
- Adequate exercise be provided.
- The appropriate amount of forage (at least 50% of the total diet) be fed.
- The feed be kept off the ground to avoid sand ingestion.
- An effective parasite control program be followed.

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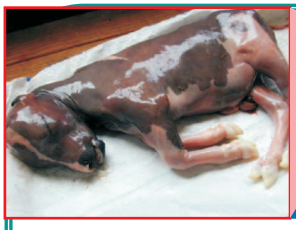

Brucellosis : A hurdle in Farm Livestock Development

Cumulative seroprevalence, PD-ADMAS study, 2011-13

No.	State	No. of samples received		% of positive samples	
		Cattle	Buffalo	Cattle	Buffalo
1	Andhra Pradesh	474	134	11	13.4
2	Assam	72	-	18	-
3	Gujarat	351	240	16.3	18
4	J & K	1437	-	6.7	-
5	Kerala	566	45	14.3	9
6	Madhya Pradesh	523	256	15.4	7
7	Maharashtra	334	75	28.4	12
8	Manipur	412	41	3.8	19.5
9	Meghalaya	23	-	0	-
10	Odisa	528	32	11.3	-
11	Punjab	140	25	43	48
12	Rajasthan	119	121	17.6	10
13	Tamilnadu	152	-	11.1	-
14	Uttarakhand	120	-	6	-
15	Karnataka	380	52	14	2
		5631 (Positive 717)	1021 (Positive 125)	Av. 12.73%	12.24%

Feline Immunodeficiency Virus

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Introduction

Feline immunodeficiency virus (FIV) was first reported in California in 1987. Feline immunodeficiency virus (FIV) is a lentivirus of cats related structurally, biochemically and genetically to human immunodeficiency virus (HIV), the cause of AIDS in humans. Most isolates have come from domestic cats; however, related but distinguishable isolates have also been made from lions, puma, pallas, cat, and bobcat; Antibodies have been found in sera of more than 18 different feline species.

Etiology - Group- group VI (ssRNA-RT)

Family - Retroviridae

Subfamily - Orthoretrovirinae

Genus - lentivirus

Species - feline immunodeficiency virus

Epidemiology

Feline immunodeficiency virus is found worldwide and the infections are acquired by horizontal transmission among adult cats. Vertical transmission of FIV appears to be unlikely. Feline immunodeficiency virus is mainly passed from cat to cat through deep bite wounds, the kind that usually occurs outdoors during aggressive fights. Infectious virus is found in the saliva of FIV positive cat, and the fighting and biting behavior of this group of cat is believed to be the main source of transmission. Transfusion of blood from and infected cat is also highly effective means of transmitting the disease. Another, less common mode of transmission is from an FIV infected mother cat to her kitten. FIV is highly variable dependent on age, sex, lifestyle, physical condition, and geographic location.

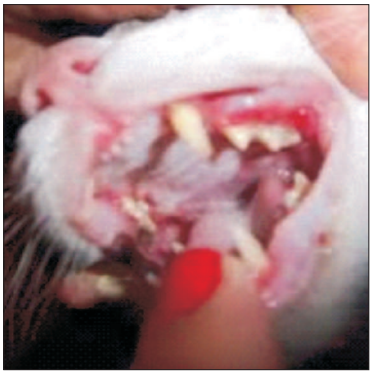
Pathogenesis

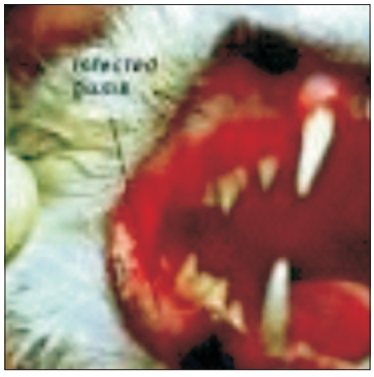
Inoculation with FIV generally results in seroconversion by 3-4 weeks and the development of lymphadenopathy at around 6 weeks after infection. Some hematological changes may develop, especially lymphocytosis. This primary stage appears to be essentially self-limiting and lasts several months. Cats may have intermittent pyrexia and secondary infections e.g. bacterial skin infections and vaccination against other pathogens may not be effective at this stage of infection. After a few months, the lymphadenopathy subsides and virus may become increasingly difficult to isolate with time.

Clinical feature

FIV attacks the cat's immune system which makes it vulnerable to secondary bacterial, viral, fungal and protozoal infections. It can be developed in the following steps-

Stage 1: Once inside the body, FIV is carried to the regional lymph nodes, where it replicates in the white blood cells known as T lymphocytes (CD4+ lymphocyte). It then spreads to other lymph nodes throughout the body. At this time, there may be an acute illness which is characterized by fever, leukopenia, anemia, malaise and swollen lymph nodes, lasting a few weeks. During this initial stage, it may go unnoticed that the cat is unwell.





Gingivities and Stomatitis as seen in FIV infection

Stage 2: This is the asymptomatic phase which can last for many years. During this stage cat appears healthy and is able to lead a normal life.

Stage 3: FIV destroys the T lymphocytes and these cells are required for the proper functioning of the immune system. Eventually when enough T lymphocytes have been destroyed, the immune system loses its ability to fight off opportunistic infections and signs of immunodeficiency develop. Cats show a range of symptoms in this stage; these symptoms may vary from cat to cat, including enlarged lymph nodes, weight loss, poor coat condition, anemia, gastroenteritis, gingivitis and stomatitis, diarrhea, chronic or recurrent infections of the skin, eyes, urinary tract and respiratory tract.

Diagnosis

Diagnosis of FIV is based on history, clinical signs, antibody test and virus isolation. ELISA is commercially available for the detection of antibody and some kits can be bought as combined FIV and feline leukaemia virus kit for practice laboratories. Some laboratories use in-house ELISAs or immunofluorescence assay and some also use western blots or radioimmune precipitation assays (RIPA) to test for antibody.

Virus isolation is very expensive. It involves the collection of 1-5 ml of heparinized blood into special media and culture of lymphocytes from the sample. It may take a month or more before the presence of virus can be confirmed. Future developments, however, may include antigen capture ELISA which work directly on the saliva or blood and more routine use of molecular techniques such as the polymerase chain reaction (PCR) for rapid virus detection.

Treatment

Treatment is based on nursing and treatment for secondary pathogens. Antibiotics can be used to help control secondary and opportunist bacterial infection. Corticosteroids or megoestrol acetate may also help moderate systemic but probably have little long term beneficial effect.

Reference:

The matter is compiled from -

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Fading Puppy Syndrome in Dogs

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Introduction

Fading Puppy Syndrome is a term used to describe puppies that are apparently normal at birth but gradually "fade" and die within the first two weeks of life. Normal pre-weaning losses in dogs, including stillborn puppies, can be up to thirty percent (30%), with about half of these deaths occurring within the first week of life. During the first two weeks of life, puppies are very vulnerable to illness and environmental stress since they are unable to regulate their body temperature independently. Very young puppies also have poor ability to regulate fluid and energy balance. In addition, their immune systems are not fully functioning and they have limited protection from infections. This makes puppies susceptible to dying from a variety of causes.

Clinical signs

The clinical signs are vague and insidious. It is often too late to save a puppy once clinical signs are apparent. The common findings are a low birth weight or failure to gain weight at the same rate as their siblings (the "runt" of the litter), decreased activity and inability to suckle. These puppies have a tendency to remain separate from the mother and the rest of the litter. They are often reported to cry weakly in a high-pitched tone. Some people refer to this as "seagulling" due to its similarity to the cry of seagulls. These puppies often quickly progress to severe lethargy, loss of muscle tone and death.

Causes

There are many factors that contribute to fading puppy syndrome. Some of the more common factors include:

1. Lack of adequate care from the mother
2. Lack of milk production or poor quality milk
3. Inadequate nursing or milk consumption
4. Congenital defects in the puppy, which may not be immediately apparent
5. Low birth weight
6. Infectious causes

One or more of these factors can contribute to fading puppy syndrome. For example, a lack of mothering instinct coupled with poor hygiene can often result in neonatal septicemia (systemic infection) in a very short time. Although some maternal immunity is conferred to the puppy while it is developing in the mother's uterus, the majority of this immunity is acquired via the colostrum or "first milk." If the puppy does not drink an adequate amount of this first milk, it is more vulnerable to infection. It is important that

the mother be examined immediately after giving birth for abnormal teat discharge, mastitis, metritis or other illness.

Many common bacteria can cause overwhelming septicemia and death in a vulnerable puppy in a very short amount of time. Because of the weakness and poor immune response, death often occurs quickly and with few, if any, clinical signs. Viral infections can cause fading puppy syndrome. If the mother is carrying a virus or isn't properly vaccinated, the puppies are more likely to contract an infection from the mother or have an even weaker immune system. Canine parvovirus, adenovirus and canine distemper have all been implicated as causes of fading puppy syndrome.

Intestinal parasites (especially hookworms), fleas and ticks can also weaken a puppy by taking vital blood and nutrients away from the developing puppy. Infested puppies often fail to gain weight and slowly "fade" and die.

Prevention

It is important to ensure that the puppy receives adequate fluid and is kept warm. Puppies should not be allowed to become chilled. During the first four days of life, the environmental temperature where the puppies are kept should be maintained at 85 -90°F (29.5-32°C). The temperature may then be gradually decreased to approximately 80°F (26.7°C) by the seventh to tenth day. It is not necessary to heat the whole room to these temperatures. Heating over the whelping box with the aid of a heat lamp is usually all that is necessary.

If bacterial septicemia develops, antibiotics may benefit the puppy, but strict hygiene and good management procedures are also critical.

A necropsy (autopsy) should be performed to determine the cause of death. This may help to prevent other puppies from dying from the same cause.

Tularemia a Biological Weapon - a Threat

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Introduction –

Tularemia is a serious infectious disease caused by bacterium *Francisella tularensis*, a gram negative, non motile coccobacillus. The bacterium has several subtypes, the most important of these is *F. tularensis tularensis* which is found in rabbits and it is highly virulent in human and domestic rabbit. *F. tularesis palaearctica* occurs maily in aquatic rodents and it is less virulent. The primary vectors are ticks, deer flies and some arthropods. The disease is named after Tulare country, California.

Epidimiology-

The disease is endemic in Asia and some parts of Europe. The most common mode of transmission is via arthropod vectors and the ticks involved are *Amblyomma*, Dermacentor, Haemaphysalis and Ixodes. Rodents, rabbits and hares often serve as reservoir hosts. Tularemia can also be transmitted by biting flies, particularly the deer fly *Chrysops discalis*. Individual flies can remain infective for 14 days and ticks for over two years .Tularemia may also be spread by direct contact with contaminated animals or materials, by ingestion of poorly cooked flesh of infected animals or contaminated water or by inhalation.

Signs And Symptoms –

Depending on the site of infection , tularaemia has six characteristic clinical symptoms- ulceroglandular (75 %), glandular, oropharyngeal, pneumonic , oculoglandular and typhidal.

The incubation period for tularaemia is 1 to 14 days , most human infection becomes apparent after three to five days. In most susceptible mammals, the clinical sign include fever, lethargy, anorexia, sign of septicaemia and possibly death. Nonhuman mammals rarely develop the skin lesions seen in humans. Sub clinical infections are common, and animals often develop specific antibodies to the organism . Fever is moderate or very high and tularaemia bacilli can be isolated from blood culture at this stage. The face and eyes redden and become inflamed. Inflammation spreads to

the lymph nodes, which enlarge and may suppurate. Death occurs in less than 1%, if therapy is initiated promptly.

The microbiologist must be informed when tularaemia is suspected because F. tularensis requires special media for cultivation such as buffered charcoal and yeast extract . Molecular method such as PCR are available in reference laboratories.

The bacteria can penetrate into the body through damaged skin and mucous membrane, or through inhalation. Humans are most often infected by tick bite or through handling an infected animal. Ingesting infected water, soil, or food can also cause infection. Tularemia can also be acquired by inhalation. Hunters are at a higher risk for this disease because of the potential of inhaling the bacteria during the skinning process. It has been contracted from inhaling particles from an infected rabbit ground up in a lawnmower. Tularemia is not spread directly from person to person. The exact cause of death is unclear, but it is thought to be a combination of multiple organ system failures.

Treatment and Prevention –

The drug of choice is streptomycin. Tularemia may also be treated with gentamicin for 10 days, Tetracycline drugs such as doxycycline for 2-3 weeks, chloramphenicol or fluoroquinolones. An attenuated, live vaccine is available, but its use is only for high risk groups.

Tularemia as a Biological Weapon-

The Center for Disease Control and Prevention (CDC) regards F. tularensis as a viable biological warfare agent , and it has been included in the biological warfare programs of the United States, Soviet Union and Japan at various times.

In the US, practical research into using rabbit fever as a biological warfare agent took place in 1954 at Pine Bluff Arkansas, an extension of the Camp Detrick problem.

It was viewed as an attractive agent because:

- It is easy to aerosolize.
- It is highly infective, between 10 and 15 bacteria are sufficient to infect victims.
- It is non persistent and easy to decontaminate (unlike anthrax).
- It is highly incapacitating to infected persons.
- It has comparatively low lethality, which is useful where enemy soldier are in proximity to noncombatants , e.g. civilians

The Schu S4 strain was standardized as " Agent UL" for use in the United States M143 bursting spherical bomblet. It was a lethal biological warfare agent with an anticipated fatality rate of 40-60 percent. When the 425 strain was standardized as "agent JT" (an incapacitant rather than lethal agent), the S4 strain's symbol was changed again to SR.

Both wet and dry types of F. tularensis (identified by the codes TT and ZZ) were examined during the "Red Cloud" tests, which took place from November 1966 to February 1967 in the Tanana valley, Alaska.

No vaccine is available to the general public. The best way to prevent tularemia infection is to wear rubber gloves when handling or skinning wild lagomorphs and rodents, avoid ingesting uncooked wild game and untreated water sources, wear long-sleeved clothes, and use an insect repellent to prevent tick bites.

Hybrid animals- an interesting update

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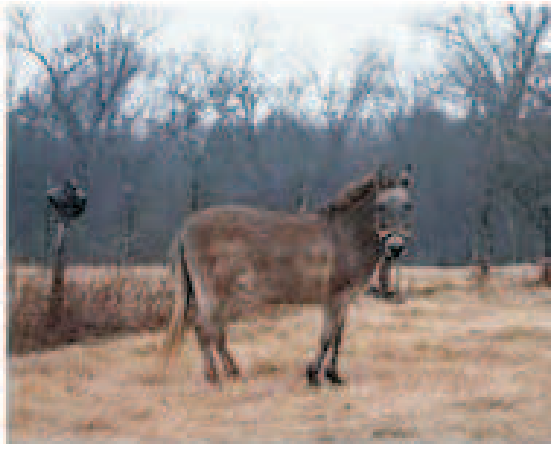
Zorse

Hybrid in animals refers to the offspring resulting by sexual reproduction from genetically dissimilar parents or animals of different varieties, species, or races. Various kind of animal hybrids are highlighted in this article.

Mule and Hinny :

Mule is the offspring of a male donkey and a female horse, whereas, **Hinny** is the offspring of a male horse and a female donkey. Horses and donkeys are different species, with different chromosome numbers. The somatic cells of horse and donkey have 64 and 62 chromosomes respectively. The hybrid mule/hinny have 63 chromosomes (Benirschke et al., 1962). Mules and Hinnies are mostly sterile. Very exceptionally (1 out 1 billion cases) hybrids have produced fertile offspring. In China (1981), when hinny was bred to donkey, they produced "Dragon Foal" resembling to the mule. Hinnies are slightly smaller than mules. Similarly when mule was bred to donkey in Morocco (2003), they produced male offspring genetically similar to Dragon foal.

Mule



Hinny

Various combinations of horse and donkey hybrids

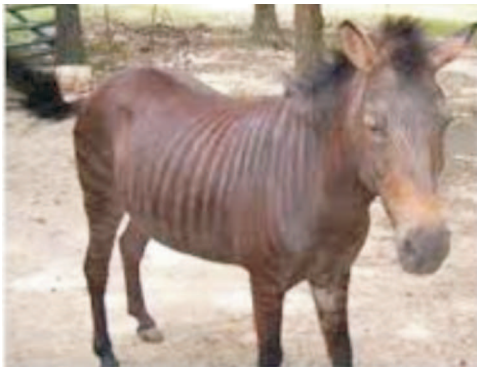
Sire	Dam	Offspring	Male offspring	Female offspring
Donkey (Jack) 2n= 62,XY	Horse (Mare) 2n= 64XX	Mule (sterile) 2n= 63,XY or XX	Horse mule or John mule,	Mere mule or Molly
Horse (Stallion), 2n= 64,XY	Donkey (Jenny), 2n=62XX	Hinny (sterile) 2n=63,XX or XY	Horse hinny	Mare hinny

Zebroid:

Zebroid is a hybrid of Zebra and donkey and horse, also known as zedonk, zorse, zebra mule, zonkey, and zebrule. Zorse is mostly sterile. In most cases, the sire is a zebra. Offspring of a donkey sire and zebra dam, called a zebra hinny, or donkra. Similarly the offspring of Zebra sire and horse dam are called Zorse, Zebrula, zebrule or zebra mule. Zebroid is used for all type of Zebra hybrid. Zebra has 32 to 46 chromosomes depending on species (Hansen, 1975).

Various combinations of zebra, horse and donkey hybrids

Sire	Dam	Offspring
Donkey (Jack) (2n=62,XY)	Zebra (2n=32 to 46,XX)	Zonkey, Donkra, Zebra hinny (2n=47 to 54)
Zebra stallion (2n=32 to 46XY)	Horse (mare) 2n=64,XX	Zorse, Zebrula, Zebrule, Zebra mule
Zebra Stallion	Pony mare	Zony



Jony

Jonkey

Dzo :

Dzo is a hybrid of yak and domestic cattle. Dzo refers to male hybrid where as female hybrid is called as dzomo or zhom. Females are fertile whereas males are sterile. They are larger and stronger than yak or cattle (Madsen et al., 2007). The diploid chromosome number in Yak is 60 (2n =60) (Das et al., 2004). All autosomes are acrocentric, X-is submetacentric similar to cattle but Y is metacentric whereas it is acrocentric and submetacentric in Bos taurus (exotic) and Bos indicus (zebu) respectively

Dzo

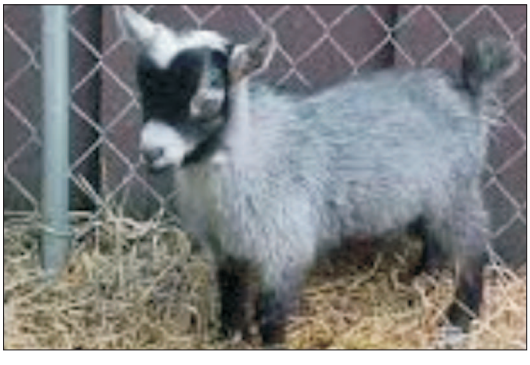
Beefalo

Beefalo

Beefalo is a fertile hybrid offspring of domestic cattle (Bos taurus) and the American bison (Bison bison) usually called buffalo in the US. The hybrid was created to produce beef. Beefalo are primarily cattle in genetics and appearance (Polzhiehn et al.,1995). The Karyotype of Bison exhibits 60 chromosomes (2n=60). All the autosomes and the X-chromosome were similar to corresponding chromosomes of exotic cattle (Bos Taurus); the Y chromosome was acrocentric (Grafodatskii, et al., 1990) similar to the zebu cattle (Bos indicus).

Sheep and goat hybrid:

Sheep and goat hybrid is generally still born due to their chromosome numbers as goat and sheep possesses 60 and 54 diploid number of chromosomes respectively. The goat and sheep belong to different genus; Capra and Ovis respectively. A live birth was obtained by crossing male sheep with female goat in Botswana. Although infertile, the hybrid had a very active libido, mounting both ewes and does even when they were not in heat (Jonathan, 2000; Mine et al., 2000). A male sheep impregnated a female goat in New Zealand, resulting in a mixed litter of kids and a female sheep-goat hybrid which was with 57 chromosomes (Stewart-Scott et al., 1990). The hybrid was subsequently shown to be fertile when mated with a ram (Tucker et al., 1989).



Sheep and goat hybrid

Liger:

Liger is a hybrid cross between a male lion and a female tiger. A similar hybrid, the offspring of a male tiger and a female lion is called a tigon. A liger resembles a tiger with diffused stripes. Male tigons and ligers are sterile, but female hybrids can produce cubs (Moscow times and BBC news 11th and 18th September, 2012). Lion and tiger both have 38 chromosomes in their somatic cells. A liger comprise of a total of 38 chromosomes, 19 of these chromosomes

Liger

come from the lion (father) whereas 19 come from tiger (mother). For tiger (2n=38), its autosomes are divided into three groups: the first group (1 to 11th pairs) is metacentric; the second group (12th-16th pairs) is submetacentric; and the third group (17th-18th pairs) is acocentric. The chromosome X is metacentric and chromosome Y is so small that its centromere cannot easily be identified (Kefen and Xingyi, 1986). The chromosome number of lion was found to be 38. The chromosomes can be divided into six groups, which consist of submetacentric, metacentric and acrocentric chromosomes (Geldenhuys, 1989). Ligers are fertile as Russian Zoo announced the birth of a liliger, which is the offspring of a liger mother and a lion father (Andreassi, 2012). Various combinations of crosses between Tiger and Lion are as under:

Sire	Dam	Offspring
Lion	Tiger	Liger
Tiger	Lion	Tigon
Lion	Liger	Liliger
Lion	Tigon	Litigon
Tiger	Liger	Tiliger
Tiger	Tigon	Titigon

Leopon

Leopon :

Leopon is the result of breeding a male leopard with a female lion. The male offspring is fertile. Female liguar, offspring of a female jaguar and male lion is fertile. The head of the animal is similar to that of a lion while the rest of the body carries similarities to leopards. The leopon has the size and strength of a lion. But, unlike the lion, they have extraordinary climbing abilities like the leopard. The karyotype (2n=38) of leopard consist of 6 groups; A-type has 4 large and 2 medium submetacentric, B-type has 6 large and 2 medium size acrocentric, C-type has 4 large metacentric, D-type 8 small submetacentric, E-type has 8 small metacentric, F-type has 2 telocenric chromosomes. The X-chromosome is medium submetacentric whereas Y- is the smallest submetacentric (Tanomtong et al., 2008).

There are many hybrids between canines, domestic and wild cats, camels etc.

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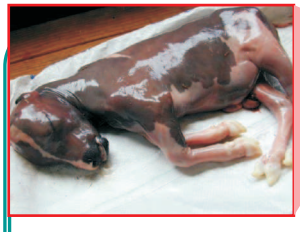

Brucellosis : A hurdle in Farm Livestock Development

Brucellosis - Overall Prevalence in farm livestock

During the period 2012-13, a total of 6701 random serum samples (Cattle-4366, Buffalo-689, Sheep-960, Goats-427, Swine-259) received from AICRP Centres were tested for brucellosis. Among 5 livestock species screened, highest seroprevalence was found in cattle and sheep (12%), followed by buffaloes (11.7%), goats (7.7%) and swine (5.7%).

The State wise disease prevalence among all the species studied showed highest prevalence in Punjab (43.6%), followed by Maharashtra (32.4%), Assam (18.9%) and Kerala (14.5%). Taminadu and Andhra Pradesh have almost same prevalence rate (11%), whereas J & K, Manipur and Uttarakhand have the lowest prevalence (less than 5%).

Plague: An Overview

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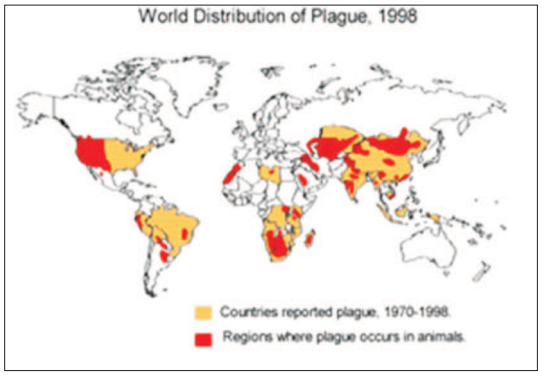

Introduction and Epidemiology

Plague is an infectious bacterial zoonotic disease of animals and humans caused by *Yersinia pestis,* a gram negative, rod shaped, non motile and non sporulating bacterium. Disease circulates mainly among small animals and their fleas. It is transmitted between animals and humans by the bite of infected fleas (*Xenopsylla cheopis*), direct contact, inhalation and rarely, ingestion of infective materials. Plague can be a very severe disease in human population, with a case-fatality ratio of 30-60% if left untreated (Pal, 1997).

People usually get plague when bitten by a rodent flea that is carrying the plague bacterium or by handling an infected animal. Millions of people in Europe died from plague during the middle Ages, when human homes and places of work were inhabited by flea-infested rats. Today, modern antibiotics are effective against plague, but if an infected person is not treated promptly, the disease is likely to cause illness or death (Gage, 1998).

Plague is endemic in many countries in Africa, in the former Soviet Union, the Americas and Asia. In 2003, nine countries reported 2118 cases and 182 deaths, 98.7% of the cases and 98.9% of the deaths were reported from Africa. Today the distribution of plague coincides with the geographical distribution of its natural foci (Bahmanyar and Cavanaugh, 1976).

Wild rodents in certain areas around the world are infected with plague. Outbreaks in human population still occur in rural communities or in cities. They are usually associated with infected rats and rat fleas that live in the home. In the United States, the last urban plague epidemic occurred in Los Angeles in 1924-25. Since then, human plague in the United States has occurred as mostly the scattered cases in rural areas (an average of 10 to 15 persons each year). Globally, the World Health Organization reports 1,000 to 3,000 cases of plague every year. In North America, plague is found in certain animals and their fleas from the Pacific Coast to the Great Plains, and from Southwestern Canada to Mexico. Most human cases in the United States occur in two regions: 1) northern New Mexico, northern Arizona, and southern Colorado; and 2) California, southern Oregon, and far western Nevada. Plague also exists in Africa, Asia, and South America (Campbell and Dennis, 1998). India also experienced outbreak of plague in

Male Xenopsylla cheopis (oriental rat flea) engorged with blood

September 1994 with a total of 693 suspected bubonic or pneumonic cases which were diagnosed to be due to *Yersinia pestis*. Initially unusually large number of deaths of domestic rats were reported in Mamla village of Maharashtra, following severe earth quack in Latur, 150 Km southeast to Surat city of Gujarat. Within 6 weeks heavy morbidity was recorded in human beings of Surat city with 56 deaths. This triggered the biggest post-independence migration of people in India, around 3,00,000 people leaving Surat city in just 2 days (Source: Wikipedia).

Clinical Symptoms

Infection usually starts with "flu-like" symptoms after an incubation period of 3-7 days. Patients typically experience the sudden onset of fever, chills, headache and body ache, weakness, vomiting and nausea. Clinically, plague manifests itself in three forms depending on the route of infection: bubonic, septicaemic and pneumonic form.

Bubonic form is the most common form of plague, resulting from the bite of an infective flea. Plague bacillus enters the skin from the site of the bite and travels through the lymphatic system to the nearest lymph node. The lymph node becomes inflamed as the plague bacteria, *Yersinia pestis*, replicate here in large number. The swollen lymph node is called a "bubo" which is very painful and can become suppurated as an open sore in advanced stage of infection.

Septicaemic form of plague occurs when infection spreads directly through the bloodstream without evidence of a "bubo". More commonly, advanced stages of bubonic plague result in the presence of *Y. pestis* in the blood. Septicaemic plague may result from flea bites and from direct contact with infective materials through cracks in the skin.

Pneumonic form of plague is the most virulent and least common form of plague. Typically, pneumonic form is due to a secondary spread from advanced infection of an initial bubonic form. Primary pneumonic plague results from inhalation of aerosolized infective droplets and can be transmitted from human to human without involvement of fleas or animals. Untreated pneumonic plague has a very high case-fatality ratio (Pal, 1997).

Diagnosis

Diagnosis is usually based on sudden outbreak, clinical symptoms and laboratory investigations of bubo aspirates, blood/serum, and sputum samples and detection of vectors and pathogen in the area/patient.

Recovery and identification of *Y. pestis* culture from a patient sample is optimum for confirmation. Depending upon the presentation of the form of plague bubo aspirates, blood, and sputum are the most appropriate specimens for rapid testing and culture. Serum taken during

the early and late stages of infection can be examined to confirm infection. Blood agar is ideal medium for the isolation of bacteria. Rapid dipstick tests have been validated for field use to quickly screen for Y. pestis antigen in patients. Specimens should be collected and forwarded to laboratories for plague testing (Perry and Fetherston, 1997).

Treatment

Rapid diagnosis and treatment is essential to reduce complications and fatality. Effective treatment methods enable almost all plague patients to be cured if diagnosed in time. These methods include the administration of antibiotics and supportive therapy. Chloramphenicol, doxycycline, streptomycin and tetracycline are effective drugs for the treatment of plague (Pal, 1997).

Prevention and Control

The objective of preventive measures is to inform people to be aware of the areas where zoonotic plague is active and to take precautions against flea bites and handling carcass while in plague-endemic areas. People should avoid having direct contact with infective tissues, or from being exposed to patients with pneumonic plague. Rodent control measures such as rodenticide (sodium fluoroacetate and zinc phosphide) and environmental sanitation should be considered in addition to health education of people about the environmental hygiene.

Vaccination

Plague vaccines at one time were widely used but have not proven to be an approach that could prevent plague effectively. Vaccines are not recommended for immediate protection in outbreak situations. Vaccination is only recommended as a prophylactic measure for high-risk groups, e.g. laboratory personnel who are constantly exposed to the risk of contamination (Gage, 1998).

Surveillance and control

Identification of animals and flea species that are implicated in the plague enzootic cycle in the region should be investigated to develop a programme on environmental management to limit its potential spread. Active long-term surveillance of zoonotic foci and rapid response to reduce exposure during epizootic outbreaks have been successful in reducing human plague (Butler, 1983).

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News... National...

The new scheme for Dairy Development

New Delhi - July 14, 2014 - The Cabinet Committee on Economical Affairs has approved the implementation of "The National Programme for Bovine Breeding and Dairy Development (NPBBDD)", with an allocation of Rs. 1800 Crores during the 12th Five Year Plan. The Scheme will be implemented during the 12th Plan Period (2013-14 to 2016-17). The spill over activities of the Project will be extended to 13th Plan.

The NPBBDD has been formulated by merging four on-going Schemes in Dairy Sector of the Department of Animal Husbandry, Dairying and Fisheries of Govt. of India. They are, National Project for Cattle and Buffalo Breeding (NPCBB), Intensive Dairy Development Programme (IDDP), Strengthening Infrastructure for Quality and Clean Milk Production (SIQ and CMP) and Assistant to Co-operative. This integration has been done to integrate milk production and dairying activities in a scientific and holistic manner for attaining higher levels of milk production to meet the increasing demand for milk in the country.

The NPBB component of the Scheme will focus on extension of the field artificial insemination (AI) network, monitoring of AI programme, development and conservation of indigenous breeds and established meant of Breeder's Associations and Societies.

The NPDD component has been designed to develop infrastructure from the grass root level by providing financial and technical assistance for dairy development. The infrastructure related to production, procurement, processing and marketing by milk unions / federations shall be focused. Extension activities related to training of farmers shall be introduced.

By the end of NPBBDD Scheme, 5000 MAITRI centers will be established on self sustainable basis to deliver breeding inputs at the farmer's doorstep. 36418 dairy co-operatives will be organized with membership of 2 million farmers. The programme will also create milk chilling capacity of 2.8 million liters of milk per day and processing capacity of 3.01 million liters of milk per day.

Know the prestigious institute

Indian Veterinary Research Institute - Mukteswar campus
The glorious 125 years of service to the nation

The establishment of a Veterinary Research Laboratory in India was recommended in 1885. The College of Science at Pune, under Principal Dr. Theodore Cook was chosen as its site. The investigation on cattle diseases and the appointment of an Imperial Bacteriologist was entrusted to him. Dr. Cook recommended the appointment of Dr. Alfred Lingard, a distinguished medical bacteriologist, for the post and this recommendation was accepted.

Consequently, the foundation stone of Imperial Bacteriological Laboratory was laid by the Governor of Bombay on 9th December, 1889 at Pune on 5.5 acres of land adjoining the College of Science. Dr. Alfred Lingard, was appointed in 1891 as Imperial Bacteriologist and his terms of reference were as follows:

"To investigate diseases of domesticated animals in all provinces in India and to ascertain, as far as possible, by biological research both in the laboratory and, when necessary, at the place of outbreak, the means for preventing and curing such diseases"

The laboratory at Pune first devoted its attention to the making of Pasterur's anthrax vaccine. Later, when it was realized that anthrax, as a disease of cattle in India, is of secondary consideration compared to rinderpest, the Government thought it advisable to proceed with the investigation and manufacture of a serum for the protection of bovines against rinderpest. For this purpose, Pune was regarded as an unsuitable headquarters, its temperature having been found too high for the cultivation of micro-organisms in certain media, while the situation of the buildings precluded the working on any highly infectious cattle disease. Consequently, it was decided to shift the laboratory to the Himalayas with a view to obtaining, amongst other requisites, a large area of ground for isolation purposes,

a high elevation and a moderate temperature. For the above mentioned reasons, in August 1893, Mukteswar was selected as the new site and 3000 acres of estate purchased there. It fulfilled, what were then regarded as the necessary requirements for a laboratory designed for research into animal diseases. It is situated at a height of 7,620 ft. and is cool enough round the year for the preservation of vaccines and sera, a necessary qualification the days before the introduction of modern methods of refrigeration. In addition, it is sufficiently remote from populous areas for there to be little fear of disease spreading from its experimental herds to those of neighboring places.

Historical Visit of Robert Koch to Mukteswar

Robert Koch (1843-1910), the famous German bacteriologist was invited by the Govt. of India to control rinderpest pandemic. He arrived at Mukteswar in May 1897 with his scientific workers George Gaffky (1850-1918) and Richard Pfeiffer (1858-1926). They confirmed that the clinical picture of rinderpest in India and South African countries were identical and proposed the production of anti-rinderpest serum.

Production of anti-rinderpest serum

As Anti-Rinderpest serum inoculation has become very popular soon after 1900, the large scale production of Rinderpest anti-sera started at Imperial Bacteriological Laboratories at Mukteswar. The passive immunization was found to protect animals up to 14 days.

Goat Tissue Rinderpest Virus (GTRV) vaccine

In 1926, J.T. Edwards, Director of the Imperial Bacteriological Laboratories at Mukteswar attempted to "fix" a Hill Bull virus in goats by growing it serially in goats. After 600 serial passages, the virus was sufficiently attenuated and did not cause the disease, but conferred the immunity which resulted into the development of Goat Tissue Rinderpest Virus (GTRV) vaccine. This paved the way for new era in the fight to control the Rinderpest in India and beyond.

In late 30's when the techniques of lyophilization for the freeze drying of veterinary biologicals became available, it was rapidly applied to produce freeze dried GTRV. In 1931, it became apparent that GTRV vaccine could be used amongst Indian plain cattle without serum simultaneous method of immunization after the successful experimental trials conducted in Bengal, Mysore and Central Province. The desiccated spleen powder as a source of vaccine became available in 1936 and this live attenuated GTRV vaccine method for production and use were updated and the vaccine was available from IVRI.

Tissue culture Rinderpest Vaccine (TCRP)

The development of tissue culture Rinderpest virus (TCRP) strain by Walter Plowright and Ferris (1959) in Kenya provided a definite edge over then existing traditional vaccine strains, because of its uniformity in the production and acceptability to the international community engaged in combating the disease world over. In 1966, Kabate "O" strain of TCRP virus for vaccine use was introduced at IVRI Mukteswar with the assistance of FAO, which was procured from Cairo, Egypt. The initial experimental batches of TCRP vaccine were produced satisfactorily in 1967 and 1968 in calf kidney monolayer. In 1969, 20 lakh doses and in 1970, 11 lakh doses were produced at IVRI Mukteswar and were supplied to the field.

Rinderpest diagnostic assays developed for field use

A battery of rapid diagnostic tests for detection of rinderpest antigen and antibody viz., CIE, RIA, ELISA, IPT, FAT, SRH, RPHA, TLI, SPACE and LA were developed during the eighties. The rinderpest laboratory of this

campus served as a Reference laboratory in the country for differential (rinderpest and Peste des Petits Ruminants) and confirmatory diagnosis and supply of diagnostic reagents and various seed viruses. The MAb based c-ELISA kit developed here was approved by O.I.E and validated by IAH, Pirbright, U.K. and the technology was used for sero-surveillance of rinderpest during the last phase of Rinderpest surveillance.

Renaming of the Institute:

In 1925, the name of the Imperial Bacteriological Laboratory was changed as Imperial Institute of Veterinary Research and the name of the Imperial Bacteriologist as the Director. Dr. J.D.E. Holmes expanded the dimension of research paving the way for the commercial manufacture of sera, to be carried out in the sub-station on the plains with the advantage of cheaper costs and more readily available transport to the field. Accordingly, a sub-station was established at Izatnagar in 1913 for large scale manufacture of sera and vaccines, and also for undertaking investigations which could best be conducted in the plains. With the passage of time and resultant expansion of activities, the daughter (Izatnagar) outgrew the mother (Mukteswsar). Dr. Ware divided the work at Mukteswar into three sections, viz, Pathology, Serology and Protozoology, each headed by a Veterinary Research Officer. In 1936, the Imperial Veterinary Serum Institute, Izatnagar, became the Biological Products Section and the Institute was renamed as Imperial Veterinary Research Institute. Dr. Frank Ware planned the addition of new sections at Izatnagar, namely, Animal Nutrition (1936), Poultry Research (1938) and Animal Genetics (1945). Soon after independence, the Institute was re-designated as the Indian Veterinary Research Institute.

Historical contributions on other diseases:

The first batch of anti-rinderpest serum was produced in 1899. During the years 1901-1906, the Institute's products included antisera against haemorrhagic septicaemia, anthrax and tetanus. During this period, a vaccine against black quarter and a diagnostic agent against equine glanders were also prepared. Some notable contributions in the field of veterinary biologicals include, fractionation of immune bodies from rinderpest serum (Hartley, 1911-13), new oxalate method of collecting blood to increase yield of sera (Shilston, 1914-15), first time reporting of buffalo malaria and attention to Johne's disease (Sheather), serum simultaneous method of rinderpest inoculation and bacteriological studies on equine abortion (W.A. Pool, 1920-21). The works of Edwards and his associates in diverse directions, viz. efforts to implant and fix rinderpest virus in hosts other than cattle, such as rabbits (1924) and goats (1927), therapeutic studies on piroplasmosis, theileriosis, etc., first description of Ranikhet disease (Cooper) and development of a vaccine (Haddow, 1939), production of crystal violet inactivated vaccine against FMD (1943).

Investigation on diagnosis of South African Horse Sickness (1960), identification and typing of the virus, development of diagnostic tests, production of potent vaccine and designing strategic control programme resulted in achieving zero status for the disease within 5 years. The Centre, presently under the Project Directorate on FMD has standardized the technique for the preparation of FMD virus infection associated antigen, its purification and characterization and its use in screening of FMD susceptible animals. In addition newer diagnostic tests viz., micro CFT, micro-neutralization, ELISA, IPT, FAT, radial immuno-haemolysis and radial immuno-diffusion were standardized for rapid typing and sub typing of FMD virus. The Centre had also lead on the study of bacterial diseases like haemorrhagic septicaemia, blackquarter, anthrax, etc. especially those of widespread origin. It had developed tuberculin, johnin and mallein for diagnosis of tuberculosis, Johne's disease

and glanders. Equine encephalomyelitis and contagious caprine ecthyma of goats were recorded for the first time in India and pathological investigation on acute theileriosis in exotic and crossbred cattle led to indication regarding the identity of the haemoprotozoan. The development of a vaccine against contagious caprine pleuropneumonia and multicomponent clostridial vaccines are the other notable contributions in post-independence era.

Current activities of the Campus

Presently, the centre has two major Divisions, Veterinary Virology and Temperate Animal Husbandry including cattle, goats, poultry and lab animals, besides some related sections like Animal Extension, Animal Biotechnology, Farm & Forestry to support research on animal production and health activities at Temperate regions as well as investigation on animal viral diseases of the country.

The Division of Virology

The Division of Virology started as a serology section in 1931, which was later upgraded as Pathology and Bacteriology section in 1939 and latter as Bacteriology and Virology section in 1952. A full-fledged Division of Bacteriology and Virology was created in 1963 and a specialized Division of Virology was subsequently established in 1975. The division has the following objectives:

- Research on animal viruses and viral disease of livestock and poultry.
- Development of diagnostic reagents and vaccines against animal viral diseases.
- Epizootiological and pathological studies on viral diseases.
- Monitoring, surveillance and diagnosis of economically important diseases.
- Post-graduate teaching, research and training.

Major achievements: (1990-present)

The live attenuated PPR Vaccine (Sungri/96): A live attenuated PPR vaccine using an indigenous isolate of PPRV (PPRV/Sungri/96) isolated from a goat in 1996 from Sungri village in Himachal Pradesh was successfully developed at this campus.

PPR and capripox combined vaccines : A Vero cell-based live attenuated goat pox and PPR combined vaccine has been developed. This vaccine is a combination of the Uttarkashi isolate of goat pox at passage level 60 (P-60) and PPR Sungri/96 at passage level 60 (P-60) in Vero cells.

The development of thermostable PPRV vaccines : The application of deuterium to enhance the thermostability of PPR vaccines has been evaluated using heavy water to reconstitute lyophilised vaccine. When a solution of deuterated water (D_2O) and $MgCl_2$ was used as the reconstituting diluent, the deuterated vaccine maintained titers of greater than $10^{2.5}$ $TCID_{50}$/ml for a period of 28 days at both 37°C and 40°C. These vaccines have undergone successful in-house trials in both sheep and goats.

Live attenuated Goat Pox vaccine: An Indian isolate of Goatpox virus (GTPV) was adapted and propagated in Vero cells for development of an attenuated virus. The technology has been transferred to VBRI Hyderabad, IIL Hyderabad, IAH &VB, Bengaluru and Hester bioscience Gujarat.

Vaccines for other diseases: Vaccines are under development for control of other viral diseases like,

buffalopox, sheeppox and orf. In house trials of these vaccines have been tested successfully and field evaluation is in progress.

Diagnostic Kits developed:

- For PPR: Competitive ELISA (c-ELISA) kit, Sandwich ELISA kit and RT-LAMP kit.
- Other diagnostic tests like RT-LAMP assay, indirect ELISA and PCR-ELISA were also developed.
- For Bluetongue: recombinant antigen based Indirect ELISA (i-ELISA) and sandwich ELISA (s-ELISA) kits, RT-LAMP assay and combined indirect ELISA for PPR and bluetongue.
- For Pox viruses: Real time PCR, LAMP assay and duplex PCRs.

Other activities

- Molecular epidemiological studies on PPR, sheeppox, goatpox, orf, buffalopox, camelpox, bluetongue, rota and classical swine fever are being carried out routinely.
- Sero-epidemiological studies on PPR, sheeppox, goatpox, orf, buffalopox, camelpox, bluetongue, and classical swine fever are being carried out routinely using serum samples from field.
- National Repository of animal viruses: The animal viruses in the repository include clinical samples (positive/negative); cell culture adapted viruses; attenuated vaccine viruses viz. PPR, sheeppox, goatpox, buffalopox, orf, camelpox; naturally recombinant goatpox virus isolated from buffalo and classical swine fever virus.
- Serum Bank: The serum bank consists of more than 20,000 serum samples from cattle, buffalo, sheep, goat, pig and deer against various animal viral diseases.

Temperate Animal Husbandry Division

The Division comprises of experimental cattle, goat and poultry farms and laboratory animal resource and has the following objectives.

- Research on various health aspects of animals of temperate region.
- Physiology of animals of temperate region and to study the nutritional requirement for optimum health and production.
- The reproductive behavior of animals of temperate region.
- To conserve genetic resources/ germ plasm of animals and to improve their genetic potential.

Present Activities:

- Maintenance and enhancement of productivity of the experimental cattle herd.
- Epidemiology of parasitic infestations of different livestock species.
- Health management of experimental cattle herd.
- Modulation of ovarian response function using insulin in anoestrus cross-breds.
- Understanding the reproductive behavior of animals of temperate region.
- Participation in different extension activities organized by the Campus.

Extension Education Section

The Section provides extension services besides participating in extension education programmes and other service activities for the socio-economic up-liftment of the farmers in the area.

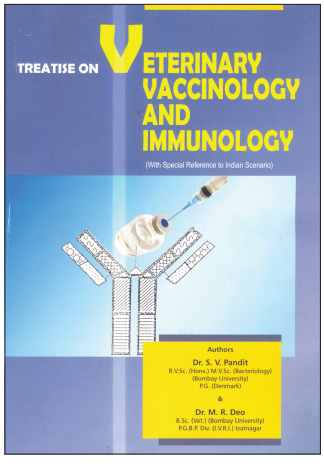
Veterinarian's Book Shelf

"Treatise on Veterinary Vaccinology and Immunology"

Dr. S. V. Pandit and Dr. M. R. Deo

Biospace; Division of Avians Trade Pvt. Ltd 36/1, Dynanesh Society, Warge Malwadi; Pune - 411052

With the advent of newer technologies in vaccine production, an era of genetically engineered Veterinary vaccines has emerged during the recent past. There are now live vector vaccines, non-replicating recombinant antigen vaccines, genetically engineered plant-vaccines, gene deleted vaccines, nucleic acid mediated vaccines and so on. This newer technology of Veterinary vaccine development, supported by biotechnology and bioengineering has developed into a new branch of science, known as "Veterinary Vaccinology".



Though the vaccination was in practice from a century ago, Immunology as a science of the defense mechanism of the body against a foreign intrusion is a recent scientific advancement. Knowing the fact now that awakening of immune system is the sole purpose of vaccine administration, studying vaccinology and immunology, *inter-alia,* becomes imperative for Veterinarians (or physicians) to exploit the vaccination effects.

This book describes the role of different body cells like T and B cells, WBCs, macrophages, natural killer cells and more specifically immunoglobulins and cytokines in mammalian and avian immune response. The book further emphasizes, not only the utility of vaccines in light of the beneficial immunological response they evoke, but also, the undesirable and side effects like anaphylaxis, amyloidosis, hypo-immune response, immune tolerance and autoimmune disorders, they produce. This book also explains the factors responsible for vaccine failure at various levels and also the measures to overcome such failures.

The vaccine production technology, including the cultivation of organisms, industrial scale production, testing procedures, use of adjuvants, freeze drying of vaccines, quality control testing and good manufacturing practices have been dealt in sufficient details. The vaccination schedule for livestock and poultry given in the book shall certainly be useful for field level officials.

In India, Veterinary vaccines shall certainly continue to be an important tool to protect not only the livestock health, but also the human health, food security and food safety. More number of vaccines against more number of existing and emerging diseases need to be developed, and there lies the importance of this book for the students, academicians, researchers, field Veterinarians and vaccine manufacturing industries alike.

This is the FIRST book of its kind published in India; holistically covering the technologies of vaccination. The authors of the book, Dr. Pandit and Dr. Deo, both of them the ardent Veterinarians, deserve the special appreciation and compliments for bringing out this finely blended product of thoroughly understood theory and the practical experience gained by them during their lifetime involvement in investigation, diagnosis, prevention, control and vaccine production in almost every livestock disease of microbial origin - old and recently emerged - prevalent in India.

Guidelines To Contributors

The contributions to the journal are accepted in the form of review articles, research articles (clinical / field studies), case reports, other information pertaining to animal health and production. The decision of the Editorial Board members will be final regarding acceptance of the article for publication. The manuscript should be typed on one side of the paper with double spacing except for footnotes and references for which single spacing be used. The style of reference citing should be followed as shown below.

The manuscript should be arranged in the following order:

Title:

Name/s of author/s:

Place of work :

Introduction :

Material and Methods : In details

Results and Discussions :

Summary / Conclusions :

Acknowledgment : If necessary

References :

Periodical/s : Surname/s and initial/s of author/s, year of publication in parenthesis, title, abbreviated name of journal (italics), volume number, (Bold), Issue number first and last page number/s.

Books : Name/s of author/s., year of publication in parenthesis, title of the book, edition (Bold), name of publishers (Italics) and place.

Tables and Figures: Tables are to be numbered in Roman numbers (1 II and so on). Each table should have a clear title. Figures should be of good quality and numbered in Arabic numbers (1,2,3 and so on).

Clinical articles and short communications: Not exceeding 3 to 4 typed pages. In case reports, history, observation, tentative and confirmatory diagnosis, line of treatment and follow up on the case should be given. Trade names of drugs should be given in the Material & Methods and their details like composition, manufacturer etc. as a footnote.

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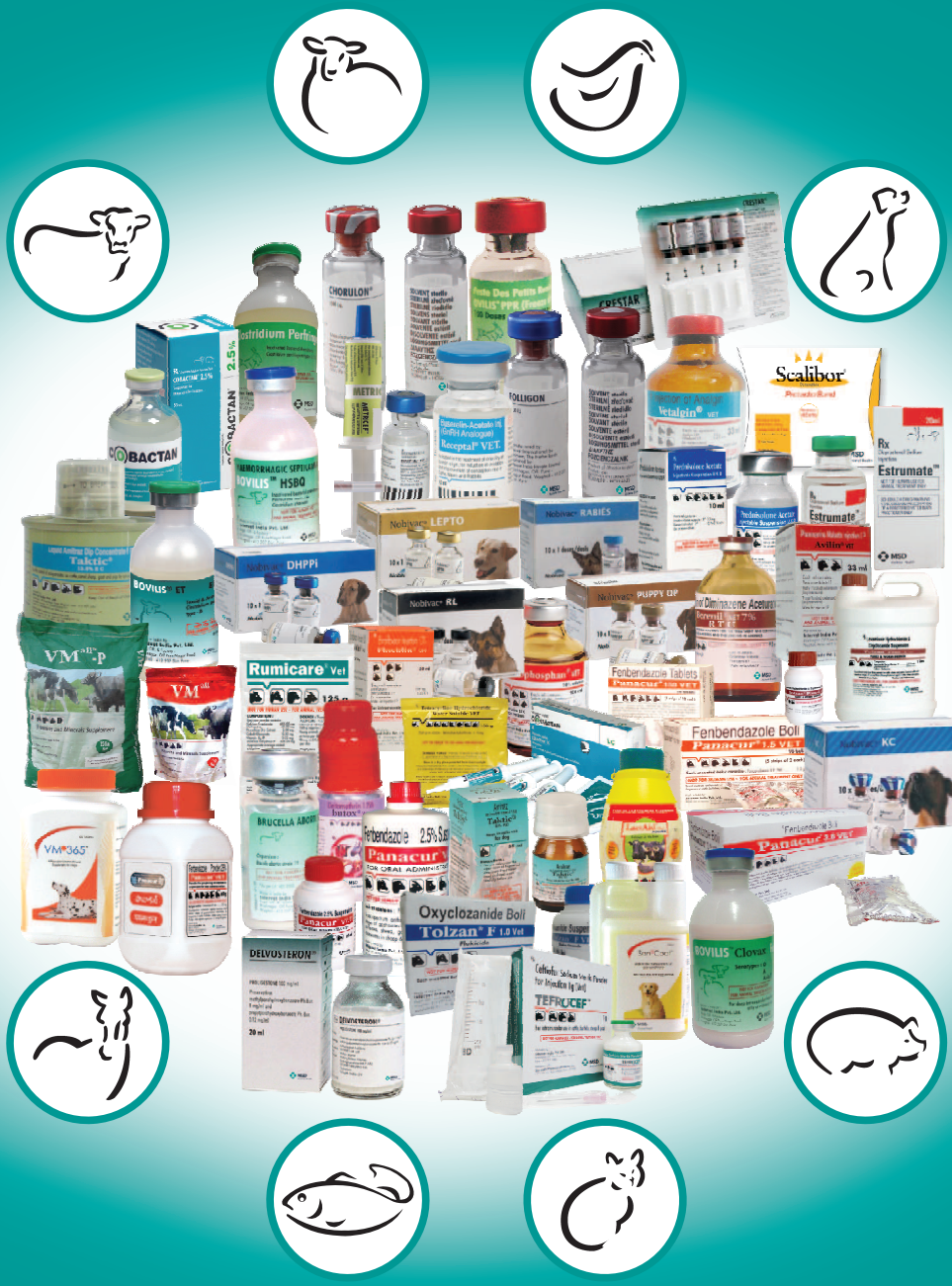
MSD
Animal Health

The Science of Healthier Animals™



For more information,
Please visit www.msd-animal-health.co.in

NEW INTRODUCTION

KNZ.™ FREE CHOICE SALT & MINERAL LICKS

AkzoNobel
Tomorrow's Answers Today

INTRODUCING

MSD Animal Health
The science of healthier animals™

KNZ™

GROW YOUR BUSINESS

HIGH QUALITY LICKS, HIGH ADDED VALUE

NEW INTRODUCTION

DEDICATED LICKS FOR INDIA

KNZ™ Tradition Universal Multi
Insurance for a daily balanced intake by a mix of essential minerals and trace elements

UNIVERSAL
MULTI
Daily Support

Ensuring your cattle
get all their daily
nutrients plus

TRADITION UNIVERSAL MULTI

The ideal supplement to daily feed and grazing for mixed cattle. Universal Multi contains essential trace elements and is enriched with iron. It is suitable for a wide variety of animals that suffer from anaemia, have a low intake of iron from basic forage or need more iron due to their workload. Iron helps to prevent animals from anaemia. This lick does not contain copper or selenium.

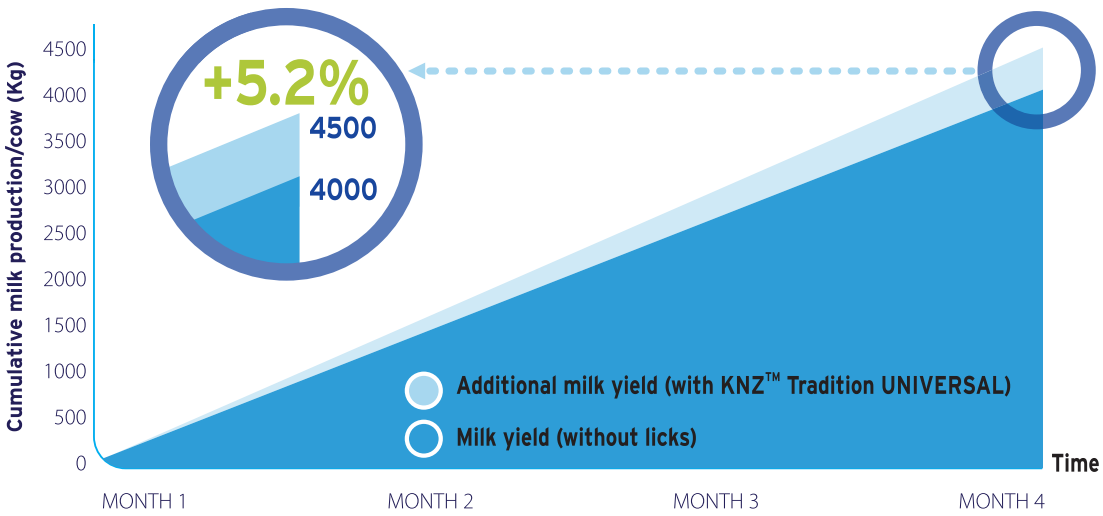
KNZ™ Tradition Fertility
Stimulates fertility with higher level of selenium, iodine and vitamin E-plus yeast selenium with a higher effectiveness

FERTILITY
Stimulates Fertility

Increasing
fertility
and improving

TRADITION FERTILITY

The perfect supplement when the fertility of your cattle is important. The lick has been specially developed to increase fertility and improve reproduction of dairy cattle. It contains a higher level of selenium, iodine and vitamin E plus yeast selenium for higher effectiveness.



According to a practical study conducted by KNZ it has been proven that using KNZ Salt & Mineral licks increased the milk yield of dairy cattle with 5.2%. Special recipes of licks to suit the Indian environment have been developed.

NEW INTRODUCTION

Floxidin™ LA (Vet) (Single Dose Formulation)

(Enrofloxacin 10%)

Presentation: 50 ml

Composition:

Each ml contains:

Enrofloxacin IP (Vet)	100 mg
Benzyl Alcohol	2% v/v

Indications:

- Mastitis
- Calf Scours, Enteritis
- Metritis Pneumonic
- Pasteurellosis (HS)
- Enzootic Pneumonia
- Wound Infections

Dosage and Administration:

Administer at the dose rate of 7.5 – 12.5 mg per Kg BW (1ml per 8-13 Kg BW) IM or SC as a single dose. If required repeat after 48 – 72 hrs.

Target Species: Cattle, Buffaloes, Sheep and Goats

Withdrawal Period: Milk – 84 Hours, Meat- 14 days

Taktic® 12.5% EC (25ml) The Brand Advantage

Features	Advantages
Brand Advantage	• Quality , consistency and reliability
Amitraz (12.5%) Advantage (amitraz-125 mg)	• Effective even after prolonged application and against all life cycle stages of ectoparasite. • Ease of using specially in large and medium sized dogs. Dose Rate- ▪ Demodectic mange-4ml/ltr of water ▪ Sarcoptic mange- 2ml/ltr of water ▪ Tick and lice: 2ml/ltr of water ▪ Weekly bath or dip to the dog till clinical symptoms subside.
Broad Spectrum	• Effective against ticks , mange and lice
Safety	• Not a organophosphorous compund, very safe . Rarely toxic and recovery is spontaneous

MSD Animal Health

RECENT INTRODUCTION

Estrumate™ *The Global Brand ...*

Available in 20 ml vial

WITHDRAWAL PERIOD
Milk : 0 (Zero) days
Meat : 0 (Zero) days

COMPOSITION

Each ml of Estrumate contains 263 mcg of cloprostenol sodium, equivalent to 250 mcg of cloprostenol.

INDICATIONS

Induction of luteolysis in dairy cattle and horses-

- Anestrous,
- Subestrous,
- Luteal Cyst,
- Pyometra,
- Persistant Corpus Luteum (PCL),
- Chronic Endometritis,
- Expulsion of Mummified Foetus,
- Termination of Pregnancy,
- Induction of parturition
- Synchronization of Estrous

DOSAGE AND ADMINSTRATION

Cattle - 2.0 ml by IM route
Ponies - 0.5-1.0 ml by IM route
Thoroughbreds, hunters and heavy horses 1.0-2.0 ml by IM route

TEFROCEF *A Solution to Many Problems...*

1g vial with 20ml sterile water for injection, disposable syringe and needle.

WITHDRAWAL PERIOD
Milk : 0 (Zero) days
Meat : 4 days

COMPOSITION

Ceftiofur Sodium sterile powder equivalent to Ceftiofur.....1g
One ampoule of sterile water for Inj. IP 20ml

INDICATIONS

- Genital infections of bovine (acute metritis, cervicitis, vaginitis, prolapse related to ROP cases ets) associated with *Arcanobacterium pyogens, Fusobacterium necrophorum and Bacteroides spp.*
- Respiratory diseases of cattle, buffalo, sheep and goat (shipping fever, pneumonia) associated with *Pasteurella haemolytica, Pasteurella multocida* and *Haemophilussomnus*.
- Acute interdigital necrobacillosis (Foot rot, Pododermatitis) caused by *Fusobacterium* & *Bacteroides.*

DOSAGE AND ADMINSTRATION

Cattle, Buffalo, Sheep & Goat : 1.1 to 2.2 mg ceftiofur per kg body weight by IM route for 3 to 5 days.

RECENT INTRODUCTION

LactAid™ Oral POWER

The Powerful Health Tonic...

COMPOSITION

Each 100 ml oral solution contains ...

Calcium ... 3500mg Phosphorus...1750 mg Zinc...100 mg

Vitamin D3...15000... Vitamin B12...200 mcg Chromium...4 mg

Carbohydrate...40,000 mg

INDICATIONS

- Improved milk production
- Improvement in growth and performance
- Stronger bones and resistance to diseases

DOSAGE AND ADMINSTRATION

25-40 ml twice daily by Oral Route

PRESENTATION

1 ltr & 5 ltr jars

Ovilis® PPR

COMPOSITION

Live attenuated PPR virus with NLT $10^{2.5}$ $TCID_{50}$ per dose with suitable freeze drying stabilizer.

INDICATIONS

For the active immunization of sheep and goats in the control of PPR infection.

DOSAGE AND ADMINSTRATION

1 ml per animal by subcutaneous route.

PRESENTATION

Vials of 100/50/25 doses.

RECENT INTRODUCTION

Nobivac® KC

COMPOSITION

Contains both Brodetella bronchiseptica (Bb) and canine parainfluenza virus (CPiV)

INDICATIONS

Vaccination against "Kennel Cough"

DOSAGE AND ADMINISTRATION

Nobivac KC aims to make administration as easy as possible:

- Low 0.4 ml dose
- Single nostril only
- Can be used with or without applicator

PRESENTATION

One box contains 5 vials of dose and 5 vials of diluent along with one applicator

Scalibor®

Deltamethrin

COMPOSITION

Scalibor P B 65 cm contains 1 gm of deltamethrin

Scalibor P B 48 cm contains 0.76 gm of deltamethrin

INDICATIONS

- Anti tick, anti flea, anti sandfly and anti sandfly.

DOSAGE AND ADMINISTRATION

One collar for six months. 65 cm (medium to large dogs) and 48 cm (smaller dogs.

PRESENTATION

6 x 65 cm and 6 x 48 cm.

HORMONES

Receptal® VET.

COMPOSITION	INDICATIONS	DOSAGE	PRESENTATION
Each ml contains : Buserelin acetate 0.0042 mg equivalent to 0.004 mg buserelin.	• True anoestrus	5 ml, IM	Vial of 10 ml and 2.5 ml
	• Improvement of conception rate (at the time of AI)	2.5 ml, IM	
	• Ovarian cyst (Follicular), Irregular oestrus, Nymphomania	5 ml, IM	
	• Delayed ovulation & Anovulation	2.5 ml, IM	
	• Improvement of pregnancy rate (11-12 days post AI)	2.5 ml, IM	WITHDRAWAL PERIOD Milk : 0 (Zero) days Meat : 0 (Zero) days
	• Improvement of post partum fertility (10-15 days post-calving)	5ml, IM	

CHORULON®

COMPOSITION	INDICATIONS	DOSAGE	PRESENTATION
Each vial contains human Chorionic Gonadotrophin (hCG) as a white freeze-dried crystalline powder (1500 IU)	• Improvement of conception rate (cows/buffaloes)	1500 IU at AI or mating, IM or IV	Box containing 5 vials (1500 IU each) with 5 vials of solvent
	• Enhancement of luteal function post AI	1500 IU, 4-6 days post AI, IM	
	• Cystic Ovarian Disease (anoestrus, prolonged estrus, nymphomania)	3000 IU, IV	
	• Induction of ovulation (mares)	1500-3000 IU, IM or IV, 24 hours before AI/mating	WITHDRAWAL PERIOD Milk : 0 (Zero) days Meat : 0 (Zero) days

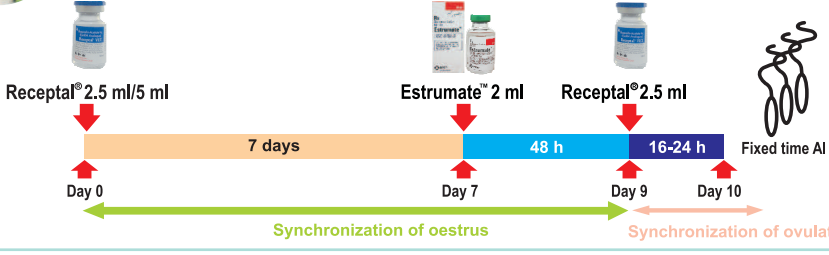
FOLLIGON®

COMPOSITION	INDICATIONS	DOSAGE	PRESENTATION
Each vial contains Pregnant Mare Serum Gonadotrophin (PMSG) as a white freeze-dried crystalline powder (1000 IU)	Females: • Anoestrus	Cow/Buffalo Anoestrus : 500 - 1000 IU IM	Box containing 5 vials (1000 IU each) with 5 vials of solvent
	• Super ovulation	Super ovulation: 1,500-3,000 IU, IM between day 8-13 of cycle	
	• Increase of fertility rate after progestagen pre-treatment	300-750 IU, IM, at the end of a progestagen treatment	WITHDRAWAL PERIOD Milk : 0 (Zero) days Meat : 0 (Zero) days

Making calf a year a reality

Oestrus Management in Dairy Cattle

Goal: One Calf per Cow per Year



MSD Animal Health

ANTI-INFECTIVE

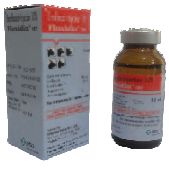
COBACTAN® 2.5% ACHIEVE MORE

COMPOSITION	INDICATIONS	DOSAGE	PRESENTATION
Each ml of suspension contains 29.04 mg Cefquinome Sulphate (equivalent to 25 mg Cefquinome).	Cattle • Respiratory disease caused by *Pasteurella multocida* and *Mannheimia haemolytica* • Digital dermatitis, infectious bulbar necrosis and acute interdigital necrobacillosis (foul in the foot) • Acute *E. coli* mastitis with signs of systemic involvement Calf • *E. coli* septicaemia	1 mg cefquinome/kg bw MI (2ml/50 kg bw) 1 mg cefquinome/kg bw MI (2ml/50 kg bw) 1 mg cefquinome/kg bw MI (2ml/50 kg bw) 2 mg cefquinome/kg bw MI (4ml/50 kg bw)	50 ml multidose vial. WITHDRAWAL PERIOD Milk : 1 day Meat : 5 days

COBACTAN® LC ACHIEVE MORE

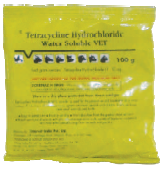
COMPOSITION	INDICATIONS	DOSAGE	PRESENTATION
Each syringe contains 75 mg Cefquinome sulphate as active ingredient.	For the treatment of clinical mastitis in lactating cows caused by *Staphylococcus aureus, Streptococcus uberis, Streptococcus dysgalactiae, Escherichia coli* & other entero-bacteria susceptible to cefquinome.	Gently infuse the contents of one syringe into the teat canal of the infected quarter every 12 hours after each of 3 successive milkings. Milk out the affected quarter (s). After thoroughly cleaning & disinfecting the teat & teat orifice, gently infuse the contents of one syringe into affected quarter. Disperse the product by gently massaging the teat & udder of the affected animal.	Box of 3 injectors with 3 isopropyl alcohol soaked towels WITHDRAWAL PERIOD Milk : 84 hours Meat : 2 days

Floxidin™ VET



COMPOSITION	INDICATIONS	DOSAGE	PRESENTATION
Floxidin 10% injection : Each ml contains - Enrofloxacin 100 mg	- Alimentary canal e.g. Enteritis, calf scours. - Respiratory tract e.g. Pneumonia - Urogenital system e.g. Metritis, cystitis - Skin e.g. Bacterial dermatitis, pyodemra. - Mastitis, & Haemorrhagic Septicaemia.	Floxidin can be given once daily, for 3-5 days. Cattle, Sheep & Goat 2.5-5 mg/kg body weight IM Dog/Cat (adult) 5 mg/kg body weight IM Camel 2.5 mg/kg body weight IM	15 ml, 50 ml Now also available 100 ml WITHDRAWAL PERIOD Milk : 3.5 days Meat : 14 days

Tetracycline WSP VET



COMPOSITION	INDICATIONS	DOSAGE	PRESENTATION
Each gm contains Tetracycline Hydrochloride WS I.P. 50 mg	In Sheep & Goat : Pneumonia, Joint ill, Anthrax, Septicaemia, Contagious Caprine Pleuro-Pneumonia, Scours, Acute Mastitis, Acute Metritis, In Cattle : Infectious diseases like Haemorrhagic septicaemia, Anthrax, Black Quarter, Leptospirosis, Foot Rot & Contagious Bovine Pleuro-Pneumonia, Calf Scours, Calf Diphtheria, Pneumonia, Septicaemia, Acute Metritis, Acute Mastitis.	Sheep & Goat : 1 gm/kg body weight Cattle : 2.5-5 gm/15kg body weight for 5 days	Sachet of 100 grams WITHDRAWAL PERIOD Milk : 7 days Meat : Cattle-15-22 days, Poultry-5 Days

METRICEF™

COMPOSITION	INDICATIONS	DOSAGE	PRESENTATION
Each single dose syringe of 19 g contains: Cephapirin - 500 mg (as benzathine) Excipient to - 19 g	• Subacute/chronic endometritis in cows over 14 days postpartum • Repeat breeders (3 or more unsuccessful inseminations).	Single dose syringe to be administered intra-uterinely	Single dose (19 g) syringe provided with a separate disposable catheter and a glove.

PARASITE CONTROL

butox® Vet

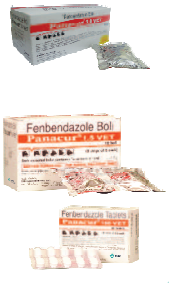
Highly effective & safe ectoparasiticide only for external use.
Ideally suited for control of ticks, mites, lice & flies of livestock, poultry, dogs & farm houses.

COMPOSITION	INDICATIONS	DOSAGE	PRESENTATION
Each ml contains : Deltamethrin BP 12.5mg	To control the ectoparasites in cattle, sheep, goats, horses, camels, dogs & farm houses.	Spray or dip : Ticks : 2 ml/lit Mites : 4 ml/lit Flies : 2 ml/lit Lice : 1 ml/lit	Aluminium container of 5 ml, 15ml, 50 ml, 250 ml and 1 lit with plastic measuring cup WITHDRAWAL PERIOD Milk : 0 (Zero) day Meat : 20 days

Taktic® 12.5% EC

Broad spectrum ectoparasiticide against ticks, mites, lice & keds

COMPOSITION	INDICATIONS	DOSAGE	PRESENTATION
Each ml contains : Amitraz I.P. (Vet) 125 mg	1. For prevention & control of ectoparasitic infestation like ticks, mites, lice & keds in cattle, sheep, goat, camel & pig. 2. Taktic kills tick, mite and lice. 3. Taktic kills organochlorine, organophosphate & pyrethroid resistant strains of ectoparasites.	Taktic 12.5%/lit of water for ticks : Cattle/Buffaloes/Camel: 2.0 ml Sheep/Goat : 4.0 ml Pigs : 4.0 ml Taktic 12.5%/L of water for mites and keds : Cattle / Camel : 2.0 ml Sheep/Goat : 4.0 ml Pigs : 4.0 ml	Tin Container of 15 ml, 50 ml & 250 ml with plastic measuring cup WITHDRAWAL PERIOD Milk : 7 hrs after applications Meat : 1 day for Cattle & Goats & 7 days for Pigs & Sheep



Panacur® VET

COMPOSITION	INDICATIONS	DOSAGE	PRESENTATION
The active ingredient of Panacur is Fenbendazole which is the research product of Intervet/Schering-Plough Animal Health. Each 1.5 g Bolus contains 1.5 g of active Fenbendazole. Each 150 mg tablet contains 150 mg of active Fenbendazole.	Infestation of cattle, buffaloes, sheep, goat & horses with gastro-intestinal nematodes, lungworms & tapeworms such as *Haemonchus spp., Ostertagia spp., Trichostrongylus spp., Cooperia spp.* and *Nematodirus spp.*	Recommended for cattle, sheep, goat, horses & pigs. Panacur 150 mg table per 30 kg body weight & Panacur 1.5 gm bolus per 300 kg body weight (5 mg Fenbendazole per kg body weight). Dose for horses : 7.5mg/kg bw	Box of 5 x 2'- 1.5 gm bolus Box of 5 x 2'- 3 gm bolus Box of 5 x 10'- 150 mg tablets. WITHDRAWAL PERIOD Milk : 0 (Zero) day Meat : 8 days

Panacur® VET Powder

COMPOSITION	INDICATIONS	DOSAGE	PRESENTATION
Each gram contains Fenbendazole I.P (Vet) 250 mg	Infestations of cattle, buffaloes, Sheep & goats with gastro-intestinal nematodes, lungworms & tapeworms such as *Haemonchus spp., Ostertagia spp., Trichostrongylus spp., Cooperia spp., Nematodirus spp., Neoascaris vitulorum, Oesophagostomum spp., Chabertia spp., Bunostomum spp., Gaigeria pachyscelis, Capillaria, Trichuris spp., Strongyloides spp., Dictyocaulus filaria, Dictyocaulus viviparus, Moniezia spp., Infestation of dogs with Ancylostoma spp., Infestation of horses with strongyles, Ascarids, Ascarids (Parascaris), Oxyuris & Strongyloides Infestation of pigs with Hyostrongylus rubidus, Oesophagostomum spp., Ascaris suum, Trichuris suis & Metastrongylus spp.*	Recommended for cattle, sheep, goat & pigs. Infestation with gastrointestinal nematodes & lungworms : (5 mg Fenbendazole per kg body weight) Suspension to be made by mixing clean water as: 6 g with 100 ml 60 g with 1 lit. 120 g with 2 lit.	6 g sachet, 60 g & 120 g container WITHDRAWAL PERIOD Milk : 0 (Zero) day Meat : 8 days

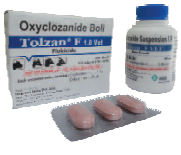
PARASITE CONTROL

Panacur® VET Suspension

COMPOSITION	INDICATIONS	DOSAGE	PRESENTATION
Each ml of Panacur 2.5% suspension contains 25 mg Fenbendazole in 90 ml 450 ml and 1 lit pack.	Infestation of cattle, buffaloes, sheep & goats with gastrointestinal nematodes lungworms & tape worms such as *Hoemonchus spp., Ostertagia spp., Trichostrongylus spp., Cooperia spp., Nematodyrus spp.,*	Dose recommended for cattle, buffaloes, sheep, goats & pigs' infestation with gastrointestinal nematodes & lungworms: (5 mg Fenbendazole per kg body weight)	90 ml 450 ml and 1 lit HDPE bottle pack of Panacur 2.5% suspension. WITHDRAWAL PERIOD Milk : 0 (Zero) day Meat : 8 days

Tolzan® Plus -L

COMPOSITION	INDICATIONS	DOSAGE	PRESENTATION
Oxyclozanide .....3.4% Levamisole Hydrochloride.....2.5%	• Tolzan Plus-L treats the round worms and liver flukes in cattle, sheep and goats • Tolzan Plus-L controls adult and immature stages of conical flukes also (Paramphistomum spp.) • Tolzan Plus-L can be used safely in pregnant animals during all stages of pregnancy. • Tolzan Plus-L can safely be given to all cattle, sheep and goats without any pre-dosing, starving or change of diet.	Cattle: 90 ml for 300 kg live mass PO Sheep and goats: 9 ml for 30 kg live mass PO	120 ml HDPE bottle, 1 Ltr can WITHDRAWAL PERIOD Milk : 0 (Zero) days Meat : 14 days



Tolzan® F VET

COMPOSITION	INDICATIONS	DOSAGE	PRESENTATION
Each ml of suspension contains Oxyclozanide I.P (Vet) suspension of 34 mg	1) Tolzan -F is used in the treatment of acute & chronic Fascioliasis in cattle, buffaloes, sheep & goats. The important species are : a) *Fasciola hepatica* b) *Fasciola gigantica* 2) Tolzan -F is also used to treat paramphistomiasis. The species involved are : *P. microbrothriodes, P. microbrothridium, P. gotal, P. orthocoelium* 3) Tolzan -F also acts on *Monezia* tapeworm in sheep.	Cattle & Buffalo : Orally 10-15 mg/kg body weight Sheep & Goat: Orally 15 mg/kg body weight	90 ml HDPE bottle & 1 ltr jerry can. Also available as 1 gm bolus 1x3x10 strip pack. WITHDRAWAL PERIOD Milk : 0 (Zero) days Meat : 14 days

Berenil® VET 7% RTU

As treatment & control therapy of Babesiosis, Trypanosomiasis and Theileriosis

COMPOSITION	INDICATIONS	DOSAGE	PRESENTATION
Each ml contains : Diminazine Aceturate 70 mg Phenazone B. P. 375 mg	Babesiosis & Trypanosomiasis, Tenacious Trypanosomiasis, Theileriosis & mixed infections, Pyrexia of Unknown Origin	Babesiosis and Trypanosomiasis at 5-10 ml per 100 kg b.w. Resistant strains of Trypanosomiasis at 10 ml per 100 kg b.w. Theileriosis & Mixed infections at 5 -10 per ml 100 kg b.w. along with antibiotic (3-4 antibiotic injections on alternate days)	Amber coloured vials of 20 ml, 30 ml and 90 ml WITHDRAWAL PERIOD Milk : 3 days Meat : 20 days

SUPPORTIVES

Tonophosphan® VET

Injectable phosphorus preparation for improving metabolism, milk production & fertility in livestock. Its content of organically bound phosphorus is 20%.

COMPOSITION	INDICATIONS	DOSAGE	PRESENTATION
Each ml contains : Sodium salt of 4-dimethylamine, 2-methylphenyl-phosphinic acid 0.2 g	As a tonic in general metabolic disorders, debility, exhaustion, repeat breeding & infertility due to phosphorus deficiency. For disorders of bone formation as in rickets & osteomalacia. To promote callus formation in fractures in combination with calcium & vitamin D. For treatment of tetany & paresis resulting from calcium, magnesium & phosphorus imbalance (as in milk fever).	In acute conditions- Large Animals : 5-20 ml. Small Animals : 1-3 ml. In chronic conditions- Large Animals : 2.5-5 ml Small Animals : 1-2 ml.	Vial of 10 ml and 30 ml Now also available 100 ml vial

VM^{all}™

COMPOSITION	INDICATIONS	DOSAGE	PRESENTATION
Each Kg contains a nutritional value of : Cobalt 120mg, Copper 1000mg, Magnesium 5000mg, Iron 2500mg, Potassium 100mg, Manganese 2000mg, Flourine 60mg, Calcium 150g, Selenium 10mg, Vit A 1200000 IU, Vit D3 120000 IU, Sulphur 0.70%, Vit E 1200 IU, Iodine 300mg, Zinc 5000mg, Phosphorus 60g, Niacinamide 4g, Vit K 200mg, Sodium 8mg.	To improve on fertility. To safeguard health and growth. To optimize milk yield and fat.	Ruminants Mix 100-200 g per 10 kg of feed depending on the availability of other fodder/feed. For direct feeding, Cow and Buffalo: 25-30 g/head/day Calf, Sheep and Goat: 15-20 g/head/day Aqua: Mix 100g to 10 kg of fish feed.	1 kg Zip-Locked pouch with measuring spoon. 5 Kg & 25 Kg bag

VM^{all}™ - P

COMPOSITION				INDICATIONS	DOSAGE	PRESENTATION
Each KG contains a nutritional value of (When packed):				• To improve on fertility • To safeguard health and growth. • To optimize milk yield and fat.	Ruminants Mix 100-200 g per 10 kg of feed depending on the availability of other fodder/feed. For direct feeding, Cow and Buffalo: 25-30 g/head/day Calf, Sheep and Goat: 15-20 g/head/day Aqua: Mix 100g to 10 kg of fish feed.	25 kg Sealed bag Now also available 5 Kg bag
Cobalt	150 mg	Vit A	1200000 IU			
Copper	2200 mg	Vit D3	120000 IU			
Iodine	325 mg	Vit K	200 mg			
Iron	2500 mg	Vit E	500 IU			
Magnesium	2500 mg	Calcium	225 g			
Manganese	2200 mg	Phosphorus	90 g			
Potassium	100 mg	Niacinamide	1000 mg			
Sodium	8 mg	Biotin 2%	500 mg			
Sulphur	1%	Bioactive				
Zinc	9000 mg	chromium	65 mg			

SUPPORTIVES

Rumicare® Vet

Normalises milk production by restoring ruminal activity.

COMPOSITION	INDICATIONS	DOSAGE	PRESENTATION
Each gm powder contains : Calcium Propionate 480.00 mg Methionine 40.00 mg Picrorhiza Dry Extract 2.00 mg Cobalt Gluconate 0.32 mg Vitamin B6 IP 0.32 mg Dextrose Anhydrous IP 428.00 mg	Bloat, digestive disorders caused by decreased activity of reticulum & rumen or sudden dietary changes &/ or intoxication. As a supportive therapy in diseases caused by foreign bodies & hypo-glycaemic conditions in cattle, calves, sheep & goats.	Adult Cattle : 125 gm sachet twice daily, (once in 12 hours Young Animals : 65 gm (approx) once or twice daily Sheep & Goat : 32 gm once or twice daily	125 g & 500 g sachet

Avilin® VET

For quick relief from allergic manifestations.

COMPOSITION	INDICATIONS	DOSAGE	PRESENTATION
Each ml contains: Pheniramine maleate IP 22.75 mg.	Itching due to eczema, dermatitis, urticaria, skin oedema, insect bites, photo-dermatitis, rhinitis, tail eczema in horses, stomatitis & inflammation of the hooves of cattle, serum sickness, paresis during pregnancy, toxaemia & retention of placenta, pulmonary oedema in cattle, pulmonary emphysema in horses.	Large animals : 5-10 ml. Small animals : 0.5-1 ml. or more. By IM or IV route	Amber coloured vial of Avil 10 ml and 33 ml WITHDRAWAL PERIOD Milk : 2 days Meat : 7 days

Prednisolone Acetate Injection

For quick relief from ketosis.

COMPOSITION	INDICATIONS	DOSAGE	PRESENTATION
Each ml contains : Prednisolone acetate I.P. 10 mg	Prednisolone is indicated in ketosis in dairy cattle, shock, inflammations (especially rheumatic arthritis, dermatitis, bursitis) and allergic conditions of livestock	Cattle, horses : 5-20 ml. Calves, pigs : 2.5-5ml. Piglets, dogs, cats :1-3 ml. or as recommended by Veterinarian.	Vial of 10 ml WITHDRAWAL PERIOD Milk : 3 days Meat : 5 days

Vetalgin® VET

Highly effective analgesic, antispasmodic, antirheumatic & antipyretic agent.

COMPOSITION	INDICATIONS	DOSAGE	PRESENTATION
Each ml contains : Analgin I.P. 0.5 g Chlorbutal (as bacteriostat) 0.4% w/v	For relief from pain, fever, labour, spastic condition of cervix during parturition, rheumatic conditions, neuritis, neuralgia, retention of placenta, dysentry, bloat & gastritis in domestic animals.	Preferably intravenous, otherwise intramuscular or combination of IV/IM injection. Horse : 20-60 ml Cattle : 20-40 ml Foal, Calf : 5-15 ml Sheep, Goat : 2-8 ml Pig : 10-30 ml Dog : 1-5 ml	Vial of 33 ml WITHDRAWAL PERIOD Milk : 2 days Meat : Cattle 12 days/Pig 3 days & Horse IV 5 days

RUMINANT BIOLOGICALS

BOVILIS™ Clovax

COMPOSITION	INDICATIONS	DOSAGE	PRESENTATION
Trivalent FMD vaccine contains inactivated and concentrated antigens of Foot and Mouth Disease virus serotypes O, A and Asia 1, adjuvanted with mineral oil sufficient to elicit > 3 PD_{50} as per Indian Pharmacopoeia regulations.	For the active immunization of cattle, buffalo, sheep and goats against Foot and Mouth Disease.	Cattle, Buffalo & Calves: 2 ml, Sheep & Goat: 1 ml by deep intramuscular route	Vials of 25 doses (50 ml).

BOVILIS™ HSBQ

COMPOSITION	INDICATIONS	DOSAGE	PRESENTATION
Each vaccine dose contains inactivated anacultures of Pasteurella multocida and Clostridium chauvoei as water in oil emulsion sufficient to induce protective levels of antibodies against HS and BQ diseases	For the prophylaxis against Haemorrhagic septicaemia and Black quarter disease in cattle and buffaloes	2 ml of vaccine per animal by deep intra-muscular route	Vials of 100 ml(50 dose)

BRUCELLA ABORTUS STRAIN 19 VACCINE LIVE IP

COMPOSITION	INDICATIONS	DOSAGE	PRESENTATION
Each vaccine dose contains 40 X 10^9 of live attenuated Brucella abortus strain 19 organisms in freeze dried form	For the active immunization of female calves of cattle and buffaloes against Brucella abortus infection	2 ml of reconstituted vaccine per animal by subcutaneous route only	Vials of 5 doses with sterile diluent

BOVILIS™ ET

COMPOSITION	INDICATIONS	DOSAGE	PRESENTATION
The vaccine contains highly immunogenic toxoids of *Clostridium perfringens* type D adsorbed on aluminium hydroxide gel as an adjuvant sufficient to induced protective levels of epsilon antitoxin titres in vaccinated animals.	For active immunization of sheep and goats against Pulpy kidney disease (Enterotoxaemia) caused by *Clostridium perfringens* type D	Sheep/Goats - 2 ml by subcutaneous injection only.	Vial of 50 doses (100 ml)

Clostridium Perfringens Vaccine Inactivated IP

COMPOSITION	INDICATIONS	DOSAGE	PRESENTATION
Each vaccine dose contains inactivated anacultures of *Clostridium perfringens* types-B,C & D adsorbed on aluminium hydroxide gel sufficient to induce protective levels of beta and epsilon antitoxin titres in vaccinated animals.	For active immunization of sheep and goats against infections due to *Clostridium perfringens* types-B, C & D.	2 ml per animal by subcutaneous route	Vials of 25 doses (50 ml).

COMPANION ANIMAL

Nobivac®: Puppy DP

COMPOSITION	INDICATIONS	DOSAGE	PRESENTATION
Each dose contains live attenuated strains of : Canine Parvo virus (strain CPV 154) $\geq 10^{7}$ $TCID_{50}$ Canine Distemper virus (strain Onderstepoort) $\geq 10^{5}$ $TCID_{50}$	Vaccination against CDV and CPV. Efficacious in puppies with maternal antibodies.	Reconstitute one vial of Nobivac Puppy DP in one vial of Nobivac Solvent & inject subcutaneously.	One box contains 10 vials of 1 dose.

Nobivac®: DHPPi

COMPOSITION	INDICATIONS	DOSAGE	PRESENTATION
Each dose contains live attenuated strains of : Canine Parvo virus (strain CPV 154) $\geq 10^{7}$ $TCID_{50}$ Canine Distemper virus (strain Onderstepoort) $\geq 10^{4}$ $TCID_{50}$ Canine Adeno virus type 2 (strain Manhattan LPV3) $\geq 10^{4}$ $TCID_{50}$ Canine Para-influenza virus (strain Cornell) $\geq 10^{5.5}$ $TCID_{50}$	Vaccination against CDV, CAV2, CPV & CPi. Besides providing protection against CAV2 disease entities such as respiratory tract infections, the vaccine also protects against infectious canine hepatitis (ICH) caused by CAV1.	Reconstitute the contents of one vial of Nobivac DHPPi in one vial of Nobivac Solvent, Nobivac Lepto, Nobivac Rabies or Nobivac RL immediately prior to use & inject subcutaneously.	One box contains 10 vials of 1 dose.

Nobivac®: Lepto

COMPOSITION	INDICATIONS	DOSAGE	PRESENTATION
Each dose contains inactivated strains of : *Leptospira canicola* (strain Ca-12-000) $\geq$40 hamster PD_{80} *Leptospira icterohaemorrhagiae* (strain 820k) $\geq$40 hamster PD_{80}	Active immunisation against Leptospirosis caused by *L.icterohaemorrhagiae* & *L.canicola of Leptospira interrogans.* Animals are protected against clinical disease, & also against becoming renal carriers after challenge.	Inject 1 ml of Nobivac Lepto subcutaneously. Nobivac Lepto can also be used to reconstitute Intervet's freeze dried vaccines Nobivac Puppy DP & Nobivac DHPPi.	One box contains 10 vials of 1 dose

Nobivac®: Rabies

COMPOSITION	INDICATIONS	DOSAGE	PRESENTATION
Each 1 ml contains inactivated Rabies strain Pasteur RIVM with potency $\geq$ 2 IU. The virus is grown on the BHK-21 clone CT cell line inactivated with β-propiolactone, and adsorbed on aluminium phosphate.	For the active immunisation of healthy dogs, cats, cattle, sheep, goats, horses and in principle all healthy mammals against Rabies.	1 ml by subcutaneous or intramuscular injection. Shake well before use.	One box contains 1 ml x 10 vials or one box contains 10 ml x 10 vials

Nobivac®: RL

COMPOSITION	INDICATIONS	DOSAGE	PRESENTATION
Each dose contains Rabies strain Pasteur RIV inducing more than 3 IU in the potency test, and inactivated strains of *Leptospira canicola* (strain Ca-12-000) $\geq$40 hamster PD_{80} and *Leptospira icterohaemorrhagiae* (strain 820k) $\geq$40 hamster PD_{80}	For the active immunisation of dogs against rabies, and canine leptospirosis caused by *L.interrogans* serogroups *canicola* and *icterohaemorrhagiae*.	1 ml by subcutaneous injection. Can be used to reconstitute Nobivac DHPPi. Intended for dogs from 8 weeks of age onwards.	One box contains 1 ml x 10 vials.

COMPANION ANIMAL

Taktic® 5% EC

COMPOSITION	INDICATIONS	DOSAGE	PRESENTATION
Each ml contains : Amitraz I.P. (Vet) 50 mg	It is indicated for the topical treatment of Demodectic & Sarcoptic Mange, ticks & lice in dogs.	Mixing Rate / lit of water: Ticks & lice - 6 ml Mites - 10 ml 3-5 applications for mange and 2 applications for ticks and lice at weekly intervals. Taktic to be used as dip or spray	Glass bottle of 25 ml with plastic measuring cup

SanCoat®

COMPOSITION	INDICATIONS	DOSAGE	PRESENTATION
Essential Fatty Acids (Linoleic Acid, Alpha Linolenic Acid, Gamma Linolenic Acid, Eicosapentaenoic Acid and Docosahexaenoic Acid) Vitamins (Vitamin A and E, Biotin and Pyridoxine) Zinc and Inositol Omega 6 and Omega 3 fatty acids in 6:1 ratio	San Coat is indicated as an aid in the management of allergic and inflammatory skin conditions like alopecia, dull and dry hair coat, pruritis, atopic dermatitis, *Malassezia pachydermatis*, pyoderma, mange etc. in dogs.	Pour measured dose on food once daily according to the following schedule. 0.3 to 1.0 ml per kg body weight. Under 7 kg - 3.75 ml 7 - 23 kg - 7.5 ml Over 23 kg - 15.0 ml	Container of 150 ml (bettix shape)

VM365®

COMPOSITION	INDICATIONS	DOSAGE	PRESENTATION
Vitamins and minerals	Beneficial for all dogs as a daily vitamin-mineral supplement, and especially during periods of stress, convalescence, growth, pregnancy and lactation.	For oral administration to dogs. Puppies and dogs under 10 lbs/4.54 kg – ½ tablet daily Dogs over 10 lbs/4.54 kg – 1 tablet daily	Container of 60 tablets

DELVOSTERON™

COMPOSITION	INDICATIONS	DOSAGE	PRESENTATION
Each ml contains 100 mg proligestone	Suppression & postponement of oestrus in the bitch, treatment of pseudo pregnancy in the bitch, suppression and postponement of oestrus in the queen and suppression and postponement of oestrus in the ferret.	Dogs Body weight — Dosage < 3 kg — 1.0 ml 3-5 kg — 1.0-1.5 ml 5-10 kg — 1.5-2.5 ml 10-20 kg — 2.5-3.5 ml 20-30 kg — 3.5-4.5 ml 30-45 kg — 4.5-5.5 ml 45-60 kg — 5.5-6.0 ml > 60 kg — 1 ml/ 10 kg	20 ml Vials

4CYTE™

COMPOSITION	INDICATIONS	DOSAGE	PRESENTATION
Marine concentrates(NZ Green tipped mussel, Abalone, Marine cartilage), Epiitalis, Binders, Antioxidants.	Can be used in all dogs for joint management . It's a nutraceutical which works as an adjunct to therapy for early recovery.	4 gm per 5 kg of weight will be loading dose which will be given for 4 to 6 weeks. Maintenance dose will be half of it.	10 gm and 50 gm sachet.

POULTRY PRODUCTS

Live Vaccine

Nobilis® Gumboro 228E

COMPOSITION	INDICATIONS	DOSE & ROUTE	PRESENTATION
The vaccine contains : Live I.B.D. virus strain 228E: >=2.0 $\log^{10}$ EID_{50}	The vaccine is recommended for active immunization of chickens against Gumboro Disease (IBD)	One dose per bird through drinking water	1000 ds 2500 ds

Nobilis® Gumboro D78

COMPOSITION	INDICATIONS	DOSE & ROUTE	PRESENTATION
The vaccine contains : Live I.B.D. virus strain D78: >=4.0 $\log^{10}$ $TCID_{50}$	The vaccine is recommended for active immunization of chickens against Gumboro Disease (IBD)	One dose per bird through drinking water	1000 ds 2500 ds

Nobilis® ND Clone 30

COMPOSITION	INDICATIONS	DOSE & ROUTE	PRESENTATION
The vaccine contains : Live ND strain Clone 30: > = 6.0 $\log^{10}$ ELD_{50}	The vaccine is recommended for active immunization of chickens against Newcastle Disease	One dose per bird through drinking water, spray, intranasal/intra ocular	1000 ds 2500 ds 5000 ds

Nobilis® IB H120

COMPOSITION	INDICATIONS	DOSE & ROUTE	PRESENTATION
The vaccine contains : Live IB strain H120: > = 3.0 $\log^{10}$ EID_{50}	The vaccine is recommended for active Immunization of chickens against Infectious Bronchitis	One dose per bird through drinking water, spray, intra-nasal / intra-ocular	1000 ds 2500 ds 5000 ds

Nobilis® MG 6/85

COMPOSITION	INDICATIONS	DOSE & ROUTE	PRESENTATION
The vaccine contains : Live M gallisepticum strain 6/85: > = $10^{6.9}$ CFU	The vaccine is recommended for active immunization of chickens to reduce the clinical signs of Mycoplasma gallisepticum infection.	One dose per bird through intraocular	1000 ds

Cell Associated Vaccine

innovax™ ND-SB1

COMPOSITION	INDICATIONS	DOSE & ROUTE	PRESENTATION
Each ampoule contains per dose at least 1534 PFU of live HVT strain/NDV-F and 1514 PFU of live chicken Herpes virus strain SB-1 in the cell associated form	The vaccine is recommended for active immunization of chickens against Marek's Disease (MD) and Newcastle Disease (ND)	0.2 ml injection subcutaneously per chick in the neck	2000 ds 4000 ds

Inactivated Vaccine

Nobilis® MG inac

COMPOSITION	INDICATIONS	DOSE & ROUTE	PRESENTATION
The vaccine contains: Inactivated Mycoplasma gallisepticum cells	The vaccine is recommended for active immunization of chickens against infections caused by Mycoplasma gallisepticum.	0.5 ml S/C	500 ml (1000 ds)

Nobilis® E. coli inac

COMPOSITION	INDICATIONS	DOSE & ROUTE	PRESENTATION
The vaccine contains: E. coli fimbrial antigen (F11) E. coli flagellar antigen (FT)	The vaccine is recommended for passive immunization of broilers against colibacillosis by vaccination of broiler breeders	0.5 ml S/C or I/M	500 ml (1000 ds)

Nobilis® Salenvac T

COMPOSITION	INDICATIONS	DOSE & ROUTE	PRESENTATION
The vaccine contains: Inactivated Salmonella enteritidis PT4 and Inactivated Salmonella typhimurium DT104	The vaccine is recommended for active immunization of chickens against S. enteritidis and S. typhimurium and to give passive immunity against these agents in the progeny	0.1 ml for day-old chicks and 0.5 ml for older birds I/M	500 ml (1000 ds)

Nobilis® Newcavac

COMPOSITION	INDICATIONS	DOSE & ROUTE	PRESENTATION
The vaccine contains: Inactivated ND Clone 30 virus	The vaccine is recommended for booster vaccination of layers and breeding stock for protection against Newcastle Disease throughout the laying period	0.5 ml S/C or I/M	500 ml (1000 ds)

Nobilis® ND Broiler

COMPOSITION	INDICATIONS	DOSE & ROUTE	PRESENTATION
The vaccine contains: ND virus Clone 30	The vaccine is recommended for the vaccination of Newcastle Disease in day-old chicks in areas where ND is endemic	0.1 ml S/C or I/M	200 ml (2000 ds)

Nobilis® Corvac

COMPOSITION	INDICATIONS	DOSE & ROUTE	PRESENTATION
The vaccine contains: Inactivated Avibacterium paragallinarum strain 083 (serotype A), strain Spross (serotype B), strain H-18 (serotype C) in oil base adjuvant	The vaccine is recommended for protection against Avibacterium paragallinarum infections in chicken	0.5 ml S/C	500 ml (1000 ds)

Nobilis® Coryza

COMPOSITION	INDICATIONS	DOSE & ROUTE	PRESENTATION
The vaccine contains : Inactivated Avibacterium paragallinarum strain 083 (serotype A), strain Spross (serotype B), strain H-18 (serotype C) in saponin base adjuvant	The vaccine is recommended for protection against Avibacterium paragallinarum infections in chicken.	0.25 ml I/M or S/C	250 ml (1000 ds)

Nobilis® Reo inac

COMPOSITION	INDICATIONS	DOSE & ROUTE	PRESENTATION
The vaccine contains : Inactivated Reovirus strains 1733 and 2408	The vaccine is recommended for booster vaccination of breeding stock against Avian Reovirus to protect their offspring against Avian Reovirus infections	0.5 ml S/C or I/M	500 ml (1000 ds)

Nobilis® G + ND

COMPOSITION	INDICATIONS	DOSE & ROUTE	PRESENTATION
The vaccine contains : Inactivated ND virus Clone 30 Inactivated Gumboro virus strain D78	The vaccine is recommended for booster vaccination of future breeders to protect against Newcastle Disease throughout the laying period, and to induce high maternal antibody levels against infectious Bursal Disease in their offspring.	0.5 ml S/C or I/M	500 ml (1000 ds)

Nobilis® IB multi + ND

COMPOSITION	INDICATIONS	DOSE & ROUTE	PRESENTATION
The vaccine contains : Inactivated IB strain M41 Inactivated IB strain D274 Inactivated ND Clone 30	The vaccine is recommended for booster vaccination of layers and breeding stock for protection against the Massachusetts and D207/D274 (and related nephropathic) serotype of Infectious Bronchitis and Newcastle Disease.	0.5 ml S/C or I/M	500 ml (1000 ds)

Nobilis® IB + G + ND

COMPOSITION	INDICATIONS	DOSE & ROUTE	PRESENTATION
The vaccine contains : Inactivated IB strain M41 Inactivated Gumboro strain D78 Inactivated ND Clone 30	The vaccine is recommended for breeding stock: as a booster vaccination to protect against Newcastle Disease and the Massachusetts serotype of Infectious Bronchitis, and to induce high maternal antibody levels against Infectious Bursal Disease in their offspring	0.5 ml S/C or I/M	500 ml (1000 ds)

Nobilis® Reo + IB + G + ND

COMPOSITION	INDICATIONS	DOSE & ROUTE	PRESENTATION
The vaccine contains : Inactivated IBV strain M41 Inactivated NDV virus Clone 30 Inactivated IBDV strain D78 Inactivated Reo virus strains 1733 and 2408	The vaccine is recommended for booster vaccination of breeding stock for protection against the Massachusetts serotype of Infectious Bronchitis and for protection against Newcastle Disease; and for immunization against Reovirus infection and Infectious Bursal Disease virus, in order to protect the offspring of the vaccinated birds against Reovirus infections and Gumboro Disease by maternal antibodies for at least the first weeks of life	0.5 ml S/C or I/M	500 ml (1000 ds)

Feed Supplement

Enradin®

COMPOSITION	BENEFITS	INCLUSION RATE	PRESENTATION
Each 1 Kg of Enradin contains 80 gm of Enramycine HCL	Helps in reducing incidence of sub-clinical necrotic entritis in chicken	5-10 ppm (63-125 gm) per ton of feed	20 Kg Withdrawal period - 7 days Avoid use in laying hens

Amnovit®

COMPOSITION	BENEFITS	INCLUSION RATE	PRESENTATION
Scientifically balanced formulation of vitamins and amino acids	Helps in relieving the stress conditions by supporting vitamins and minerals	Through water 1gm/lit for 5-7 days Through feed 500gm/ton for 5-7 days	1 Kg

Pharma Product

Floxidin™

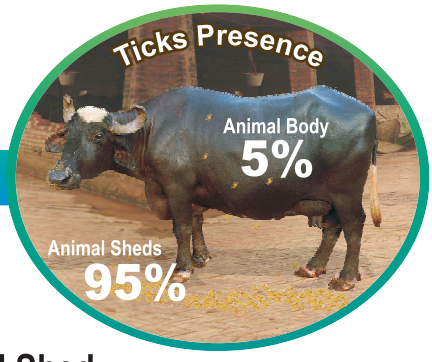
COMPOSITION	INDICATIONS	DOSE & ROUTE	PRESENTATION
Enrofloxacin 10% oral solution	The product is recommended for treatment of the common infections caused by gram-positive, gram-negative, anaerobes and mycoplasma species	10 mg per kg BW for 3-5 days	5 Lt Withdrawal period - Meat - 8 days Eggs - Stop using 14 days before laying

VAC-SAFE®

COMPOSITION	BENEFITS	INCLUSION RATE	PRESENTATION
An effervescent tablet that dilutes easily and neutralizes the chlorine in the water	Helps in improving the quality of drinking water during vaccination	1 tablet /100 Lt water	Box of 30 tablet

Tick Eradication Program

Do You Know ?



For Application on Animal Shed

butox® Vet Power

WITHDRAWAL PERIOD

Milk : 0 (Zero) day
Meat : 20 days

For Application on Animal Body

Taktic® 12.5% EC

WITHDRAWAL PERIOD

Milk : 7 hrs after applications
Meat : 1 day for Cattle & Goats & Goats & 7 days for Pigs & Sheep

Advantages

- Reduced tick load in animal shed
- Low incidences of tick born diseases
- Increased interval between two consecutive spray on animal body
- Better herd health

A step forward in the treatment of Mastitis

COBACTAN® LC

(Intramammary)

Control Measures for Mastitis

- Wash the hands with soap and water before hand milking.
- Clean the udder with antiseptic solution before & after milking.
- Use full-hand milking instead of knuckling.
- Allow animals to stand for 30 minutes after milking by providing feed or grass.
- Identify the chronic mastitic cow and milk them at last.

Advantages of Using Cobactan LC in Early Stages:

- Stops the Progression of Mastitis.
- Faster Recovery.
- Symptoms disappear quickly.
- Quick return to normal Milk production.

Withdrawal Period:
Milk- 84 hrs. (7 milking)
Meat- 2 days

Administration of Cobactan LC

Infuse **COBACTAN® LC**
(Intramammary)

At 0 hr.	At 12 hr.	At 24 hr.
1st tube	2nd tube	3rd tube



MSD Animal Health

A trusted source for comprehensive animal health solutions

Today's Merck is a global healthcare leader working to help the world be well. MSD Animal Health, known as Merck Animal Health in the United States and Canada, is the global animal Health business unit of Merck. MSD Animal Health offers veterinarians, farmers, pet owners and Governments the widest range of veterinary pharmaceuticals, vaccines, health management solutions and services. MSD Animal Health is dedicated to preserving and improving the health, well being and performance of animals. It invests extensively in dynamic and comprehensive R & D resources and a modern, global supply chain. MSD Animal Health is present in more than 50 countries, while its products are available in some 150 markets.

For more information,

visit www.msd-animal-health.co.in

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