

# RAKSHA TECHNICAL REVIEW

Publication of Indian Immunologicals Limited

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**Methicillin-Resistant  
Staphylococcus aureus:**  
A Livestock Concern

**Diagnosis and Treatment of  
Cushing's syndrome in the  
Companion Dog: An Update**

**Dystocia Due to Breech  
Presented Foetal Anasarca  
in a Marwari Doe**



INDIAN IMMUNOLOGICALS LIMITED

# Reader's Desk



It reflects through the shared content that editorial board of RTR journal is doing great. What I liked the most is quality of research stuff, case reports and articles that are beneficial not only for students but also for field vets as well. February 2019 issue was worth reading and articles related to Glanders, Marwari goat, Thelaziasis and Equine Lameness made it very special. Wish you all the best and I hope that future editions of RTR will mesmerize the veterinary fraternity.

**Dr Yogesh Arya**  
Bhilwara, Rajasthan.



I am interested in your publication. The Raksha Technical Review journal is good.

**Dr T Ramesh Babu**  
Hyderabad, Telangana.



RTR, a comprehensive publication for veterinary profession to case studies in various kinds of animals. It is not only needful column for researchers, scientists but also for students & grassroot level of field. So a lot of thanks to RTR again for better clinical issues to include and to new heights up.

**Dr Bhanwar Singh Sewada**  
Churu, Rajasthan

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## From the Editor's desk

Anti-Microbial Resistance (AMR) is a cause of serious concern for the veterinary fraternity the world over. This issue of RTR brings before you two articles relevant to AMR. One discusses about microbial biofilm formation and the other about Methicillin-Resistant *Staphylococcus aureus*.

Bovine mastitis is a serious problem caused by various microorganisms. *Microbial Biofilm Formation: A Threat to Treatment of Bovine Mastitis*, reviews the general biofilm forming pathogens that cause bovine mastitis and various methods of biofilm formation.

MRSA, (Methicillin-Resistant *Staphylococcus aureus*), is a form of contagious bacterial infection that is resistant to numerous antibiotics including methicillin, amoxicillin, penicillin, and oxacillin. The article *Methicillin-Resistant Staphylococcus aureus: A Livestock Concern* gives a brief history on the types of Methicillin-Resistant *Staphylococcus aureus*, its zoonotic risks and implications of microbial resistance.

Dystocia refers to abnormal or difficult birth. In cattle, the most common cause is foeto-maternal disproportion. This issue of RTR presents two articles on dystocia, one for HF cow and how fetotomy had helped in handling lateral deviation of neck. The second article relates to a surgical intervention in case of a foetal Anasarca in a Marwari Doe.

We wish to draw the attention of readers towards various disease management aspects. Articles on, *Buffalo Calves Mortality*, *Theileriosis*, *Brucella Vaccines*, *Swine Dysentery in Pig Production* and *Traumatic Hyphema in Kathiawari Foal* provide a detailed overview of diseases.

Under Grazers and Browsers, this issue presents to you the *Sirohi Goat*, and *Surgical Management of Traumatic Proptosis in an Assam Hill Goat*.

*Binjharपुरi Cow* and *Rampur Hound* are introduced to you under the livestock and companion animal sections respectively.

Companion Animal Section also throws light on diagnosis, treatment and management of various cases in canine practice for e.g., *Diagnosis and Treatment of Cushing's Syndrome in the Companion Dog - An Update*, *Periodontal Disease in a Female Shih Tzu Dog*, *Diagnosis and Treatment of Dilated Cardiomyopathy in Dogs*, *Surgical Management of Cutaneous Squamous Cell Carcinoma in Scrotum of A Dog* and *Surgical Management of Inguinal Nongravid Hysterocoele in a Bitch*.

*Health Care and Training Information for A New Puppy Dog Owner* is a detailed article on how to care and train a puppy for the first time pet owner.

The College of Veterinary Science and Animal Husbandry, Mhow, Madhya Pradesh is a well-known and age-old veterinary institute of high repute covered in Pioneers in Veterinary Education section.

Season's greetings to you all ahead of the festival season. We hope this issue will be interesting and enlighten the veterinary fraternity and practitioners as well to their entire satisfaction and helps in the application of the inputs in day to day practice as well as academics.

As always, we look forward to receiving your feedback and comments.

Regards,

**Dr Prasanna A Deshpande**



## Managing Director's Message

Dear Patrons,

Greetings!

Ahead of the festival season, IIL has concluded another successful business year. IIL's business is on a growth trajectory with good demand for all principal products e.g., FMD, Anti-rabies and Pentavalent vaccines.

Animal Husbandry, Dairy and Fisheries are the most important elements of India's rural economy. Major announcements made by the Union Government include a massive outlay of Rs.25000 crores for fisheries and Rs.12650 crores for National Animal Disease Control Program for eradicating the Foot and Mouth Disease and Brucellosis in livestock. These expenses are expected to be incurred during the next 5year period.

Identification of priority areas with respect to diseases and infrastructure building are among the top-10 action points of One Health Roadmap of India. Above mentioned initiatives of the Union Government are certainly good steps in this direction. This commitment of the Government encourages IIL to work even more vigorously for the future of Animal Health.

IIL believes that it is the collective responsibility of Public, Industry and the Government to combat the issues that bother the health environment of the country. It is our constant endeavor to work closely with all the stake-holders i.e., the farmers, Medical and Veterinary fraternity, business partners and the Government of India in meeting the unmet medical needs towards improving and extending lives.

I wish you all a very healthy and happy festive season ahead!

Warm Regards

**Dr K Anand Kumar**

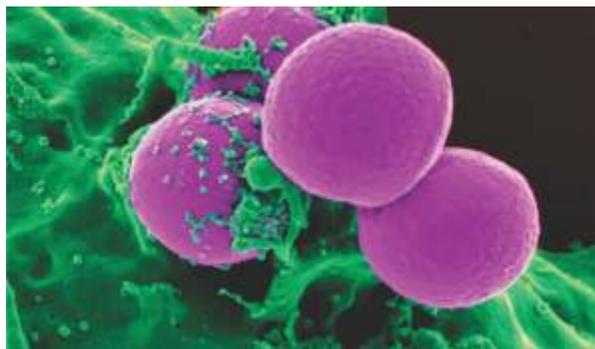


## Methicillin-Resistant *Staphylococcus aureus*: A Livestock Concern

Prateek Mishra, Vidhi Gautam, R.K. Sharma, Sachin Jain and Anushri Tiwari  
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Animal Husbandry, Jabalpur, Madhya Pradesh.

### Introduction

MRSA (Methicillin-Resistant *Staphylococcus aureus*) refers to a group of gram positive bacteria that are genetically distinct from other strains of *Staphylococcus aureus*. *Staphylococci* are Gram-positive cocci about 0.5-1.0  $\mu\text{m}$  in diameter. They grow in clusters, pairs and occasionally in short chains. The clusters arise because *staphylococci* divide in two planes. The configuration of the cocci helps to distinguish *micrococci* and *staphylococci* from *streptococci*, which usually grow in chains (1).



*Staphylococcus aureus* is a bacterium commonly found on skin of healthy people where it usually remains harmless. However, in those people who are immune-compromised, it can lead to infections ranging from the trivial to serious. Before the introduction of antibiotics in the 1940s, staphylococci were responsible for most hospital infections, primarily pneumonias and were initially susceptible to penicillin (2). However, as early as the 1950s resistant strains of *S. aureus* had emerged, Methicillin synthetic penicillin was released into the market in March 1960 and was



used to treat penicillin resistant *S. aureus*. However, by the end of the 1960s strains of MRSA had emerged and by 1980 they had spread throughout the world. The emergence of MRSA together with other related antibiotic resistant strains of bacteria such as vancomycin resistant *Enterococcus*, threatens to reverse the gains made by western bio-medicine, ushering in a return to a pre-antibiotic era in terms of the control of bacterial disease. MRSA can result serious illness, disability and death.

### **MRSA: Methicillin-Resistant *Staphylococcus aureus***

Methicillin-Resistant *Staphylococcus aureus* (MRSA) is a cause for significant concern. It is a bacterium that causes infections in different parts of the body. It is more difficult to treat than most strains of *Staphylococcus aureus* as it has become resistant to some commonly used antibiotics (3). MRSA is sometimes called a super bug. *Staphylococcus aureus* or Staph is found on the skin or nasal lining in up to 30% of healthy individuals. In this setting of colonization, it usually does not cause symptoms. However, when the skin is damaged in some way and the bacterium enters the skin it can cause a wide range of problems from a mild pimple to severe illness. Initially, most Staph infections were sensitive to penicillin. However, in the early 1960's, the term MRSA was derived as it was found that many infections from *S. aureus* had become resistant to penicillin and methicillin. Alternate antibiotics can be used but it is becoming more difficult to treat as time goes on.

The proportion of MRSA among *S. aureus* from nosocomial infections increased considerably from the end of the 1980s until 2000, in nearly all of Europe and worldwide. There are a few countries, such as the Netherlands and the Scandinavian countries, where the consequent implementation of appropriate infection control measures prevented this development. During the past five years the rising trend was halted and even reverted in several European countries, which is likely due to the introduction of mandatory surveillance for MRSA bacteremia. *S. aureus* isolates from humans and a variety of animal species, including livestock, by means of phenotypical characterization, had led to the discrimination of different ecovars (biotypes) of *S. aureus*.

MRSA was detected in domestic animals a long time ago. In early studies the genetic background and antimicrobial resistance of *S. aureus* and MRSA have been associated with host specificity in livestock. MRSA CC1 (PVL negative) was first reported from a cluster of subclinical mastitis in cattle from Hungary. Later on it was identified from infections in hospitalized horses, as a frequent colonizer of farmed pigs in Italy, and less frequently in pigs from other European countries. MRSA attributed to CC130, which contains the homologue *mecC* instead of *mecA*, gained attention. Host specificity for this clonal complex also is limited. Isolates attributed to CC130 have been reported from domestic animals, especially cattle, sheep, goats, dogs, and cats, as well as from wildlife such as roe deer, chamois (MSSA), brown rats, seals, hares (captive Mara) etc. Interestingly, MRSA CC130 was observed in wildlife as well as in domestic ruminants sharing the same habitat, suggesting mutual exchange. Although MRSA CC130 is also able to cause infections in humans.

### **MDR-MRSA (Multidrug-Resistant Methicillin-Resistant *Staphylococcus aureus*)**

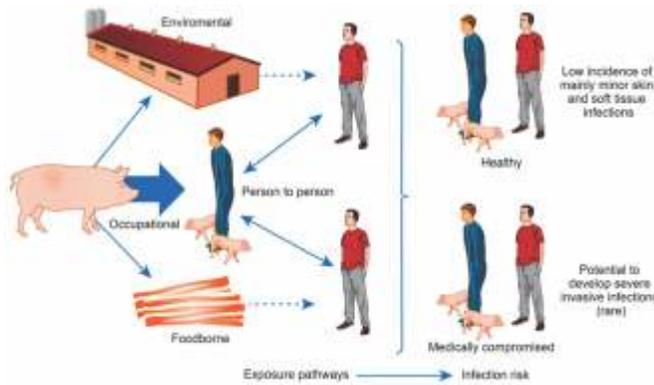
Multidrug-Resistant (MDR) *Staphylococci* pose a growing problem for human health. The rise of drug-resistant virulent strains of *Staphylococcus aureus*, particularly Methicillin-Resistant *S. aureus* (MRSA) is a serious problem in the treatment and control of Staphylococcal infections. Methicillin-Resistant *Staphylococci* (MRS) cause hard-to-treat infections. The most striking situation is that MRSA strains have emerged with concomitant resistance to many commonly used antibiotics of groups, aminoglycosides, macrolides, fluoroquinolones, chloramphenicol, and tetracycline.

A special rule has been applied in defining antimicrobial resistance in *S. aureus*. Once a *S. aureus* isolate is characterized as an MRSA, it is instantly classified as MDR, because resistance to oxacillin or ceftiofur infers non susceptibility to all categories of  $\beta$ -lactam antimicrobials (i.e., all categories of penicillins, cephalosporins,  $\beta$ -lactamase inhibitors, and carbapenems). Thus, MDR-MRSA is a new or rather a continually evolving paradigmatic pathogen (4).

### **LA-MRSA (Livestock Associated - Methicillin-Resistant *Staphylococcus aureus*)**

One of the most prominent changes in the MRSA epidemiology is the emergence of livestock associated MRSA (LA-MRSA) strains in the human population. MRSA was detected in domestic animals a long time ago. In earlier studies the genetic background and antimicrobial resistance of *S. aureus* and MRSA have been associated with host specificity in livestock. This opinion has changed due to studies based on comparative genome analysis. Furthermore, MRSA with low host specificity attributed to CC130 and CC398 emerged. Clonal complexes CC97, CC133, CC522, and clonal lineage ST151 are mainly represented by isolates from

ruminants, whereas clonal lineage ST385 is mainly represented by isolates from poultry. The first communication on LA-MRSA CC398 colonizing in conventionally raised pigs was reported from European countries. The emergence of LA-MRSA in livestock seems to correlate with farm size, farming systems, usage of disinfectants, and in-feed zinc. The spread of LA-MRSA between farms is often mediated by animal trading, mainly of piglets that are sold by specialized producers. The finding of LA-MRSA CC398 in tank milk suggests udder colonization and possibly cases of subclinical mastitis in dairy cattle. LA-MRSA represents about 13% of MRSA-linked severe skin and soft tissue infections (5).



LA-MRSA can enter hospitals either via patients who suffer from infections caused by these bacteria who need appropriate treatment or by patients with nasal colonization. The latter can lead to nosocomial infections such as surgical site infections, infections after joint arthroplasty, ventilator associated pneumonia or septicemia.

**CA-MRSA (Community Associated - Methicillin-Resistant *Staphylococcus***

**CA-MRSA (Community-Associated Methicillin - Resistant *Staphylococcus aureus*)**

**Chromosomal Genetic Elements**

**ACME**  
Uniquely carried by MRSA USA300

**SCCmec IV or V**  
Contain *mecA*, conferring  $\beta$ -lactam resistance

**Efflux Pump**

**NorB**  
May provide a fitness advantage to USA400

**Secreted Toxins and Factors**

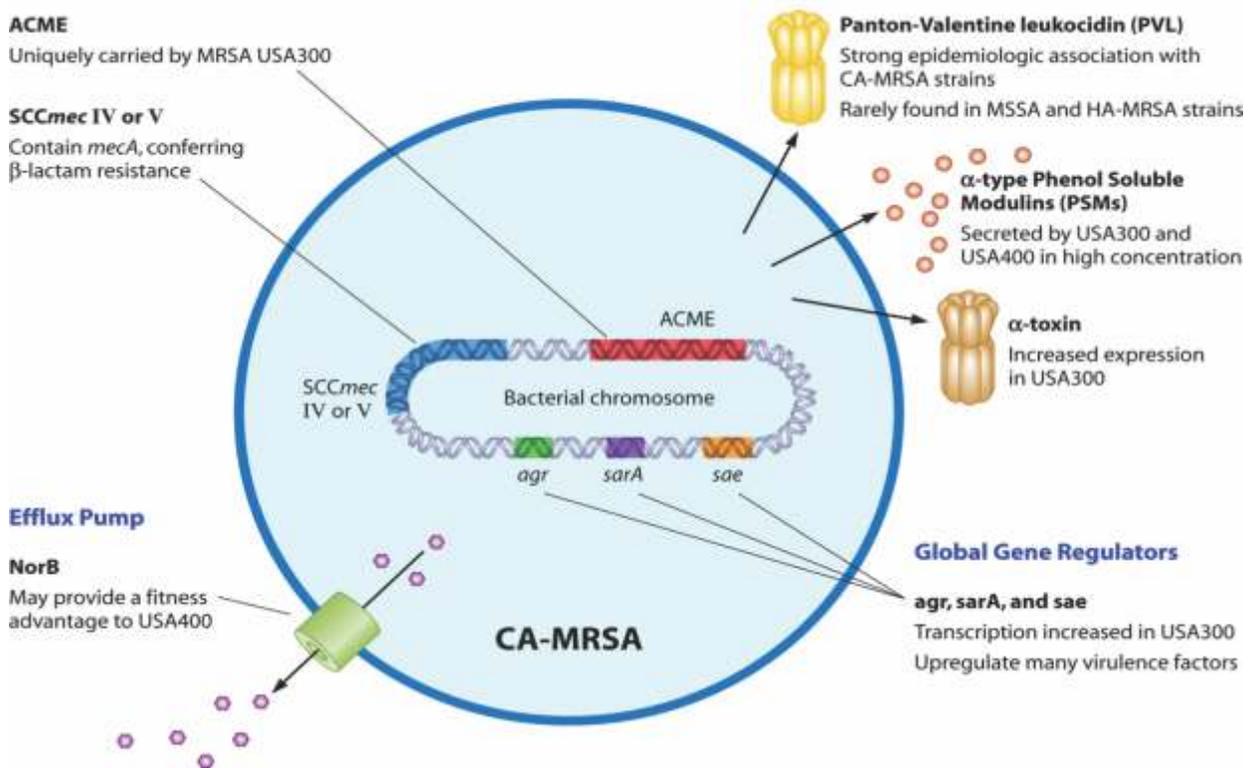
**Panton-Valentine leukocidin (PVL)**  
Strong epidemiologic association with CA-MRSA strains  
Rarely found in MSSA and HA-MRSA strains

**$\alpha$ -type Phenol Soluble Modulins (PSMs)**  
Secreted by USA300 and USA400 in high concentration

**$\alpha$ -toxin**  
Increased expression in USA300

**Global Gene Regulators**

***agr*, *sarA*, and *sae***  
Transcription increased in USA300  
Upregulate many virulence factors



**aureus)**

The first well documented CA-MRSA cases appeared in the western United States between 1997 and 1999 in children. These infections, which were fatal cases of sepsis and severe pneumonia, were caused by strain MW2 (pulsed-field type USA400). In the meantime, strains belonging to pulsed-field type strain USA300 have replaced USA400 strains in the U.S. but USA400 CA-MRSA infections are still observed in Alaska. CA-MRSA is also a global problem as Global strains of CA-MRSA belong to a series of different lineages and specific strains are predominant in different countries. For example, infections with CA-MRSA strains belonging to sequence type (ST) 80 are common in Europe and ST30 CA-MRSA infections occur predominantly in Australia. By far, the most frequent disease manifestation associated with CA-MRSA is infection of the skin and soft tissues. Skin and soft tissue infections (SSTI) account for at least 90% of CA-MRSA infections. CA-MRSA SSTI is usually moderately severe to severe, often very painful and is treated by simple incision and drainage. However, rare cases of very dramatic skin infections, such as necrotizing fasciitis, have been reported with CA- in contrast to HA-MRSA (host associated). CA-MRSA strains also cause infections of the bones and joints such as osteomyelitis and respiratory infections such as pneumonia, sepsis and urinary tract infections (6).

**Antimicrobial agents used against MRSA**

Methicillin-Resistant *Staphylococcus aureus* (MRSA) has a unique cell wall structure consisting of peptidoglycan and wall teichoic acid. In recent years, new anti-infectious agents (spirohexaline, tripropeptin C, DMPI, CDFI, cyclabdan, 1835F03, and BPH-652) targeting MRSA cell wall biosynthesis have been discovered using unique screening methods (7). These agents were found to inhibit important enzymes involved in cell wall biosynthesis such as undecaprenyl pyrophosphate (UPP) synthase, FemA, flippase, or UPP phosphatase. Viridicatumtoxin and spirohexaline, produced by *Penicillium sp.* FKI-3368, were isolated as inhibitors of undecaprenyl pyrophosphate (UPP) synthase of *Staphylococcus aureus*, which was involved in cell wall synthesis. Viridicatumtoxin and spirohexaline with a pentacyclic spiro

skeleton inhibited UPP synthase activity with an IC (50) value of 4.0 and 9.0  $\mu\text{M}$ , respectively. Actually, the growth of Gram-positive bacteria including MRSA was strongly inhibited by the compounds. Our computational modeling experiments indicated that spirohexaline A was inserted into the substrate pocket of UPP synthase and interacted with Glu (88) via a carbamoyl group of the compound, with Ala (76), Met (54) and Asn (35) via three hydroxyl groups and with certain hydrophobic amino acids via a spiro ring. Cyclabdan, produced by *Streptomyces species*. K04-0144, was isolated as a potentiator of  $\beta$ -lactam imipenem activity against MRSA. The compound consisted of a labdan skeleton and an N-acetyl cysteine. Cyclabdan potentiated imipenem activity by over 1000 fold, drastically reducing the MIC value of imipenem against MRSA from 16 to 0.03  $\mu\text{g}/\text{m}$ . The binding proteins of cyclabdan were investigated in the lysate of MRSA to identify FemA, which was involved in the formation of the pentaglycine interpeptide bridge in MRSA peptidoglycan (8).

**Medicinal plants Against MRSA (Methicillin Resistant *staphylococcus aureus*)**

Methanolic extract of leaves of *Hyssopus officinalis* (*H. officinalis*) at increasing concentrations have shown increased *S. aureus* inhibition. Phytochemicals like pinocamphone, iso-pinocamphone, linalol and 1, 8-cineole isolated from the oil of *H. officinalis* have reportedly shown notable antibacterial activities (9). Pinocamphone compound can abundantly be found in the oil of *H. officinalis* and hence needs to be tested for minimum inhibitory concentration. Ethanolic and methanolic extracts of *Rydingia limbata* also showed good inhibitory activities which may be because of phytochemicals like limbatolide B and limbatolide C, oleanolic acid and b-sitosteol. Ethanolic extracts of *Withania somnifera* has greater inhibitory effect as compared to its methanol extract and this inhibition might be due to the presence of active phytochemicals like withaferin, withanolides and Methanolic, ethanolic, n-hexane and acetone extracts of *Artemisia absinthium* and three widely used antibiotics were tested against *S. aureus*. Steroidal lactone from stem and leaves extracts of *Calotropis procera* were reportedly tested in-vitro against *S. aureus*. Chloroform extracts of



*Malva neglecta* (*M. neglecta*) have maximum inhibition against *S. aureus* which might be due to phytochemicals like flavonoids, hydroxyl cinnamic acids and phenol. Stem and leaves extracts of *Calotropis procera* were reportedly tested in-vitro against *S. aureus*.

Methanolic extract of root of *Asparagus racemosus* (*Asparagaceae*) has shown significant antibacterial effect with increasing concentration. Gobicusin A, asparacosin A and muzanzagenin are steroidal saponins compounds being isolated from *Asparagus racemosus* were tested for anti *S. aureus* effect. Ethanolic extract of leaves of *Cannabis sativa* (*Cannabaceae*) produced 20 mm zone of inhibition against *S. aureus* at 10 mg/mL Cannabinoid, a compound isolated from *Cannabis sativa* (10).

### Conclusion

*Staphylococcus aureus* is an important cause of disease and the antibiotic-resistant strains pose a threat to the community. Methicillin-resistance is of particular relevance because it imparts resistance to all  $\beta$ -lactams antimicrobial agents such as Penicillins, Cephalosporins and Carbapenems. Historically, MRSA (Methicillin-Resistant *Staphylococcus aureus*) was a human pathogen but in past decades MRSA has been reported as cause for mastitis and also been isolated from other food producing animals like pigs. If human and bovine strains, particularly the MRSA, develops the ability to spread in host population, zoonotic risks and implications of antimicrobial resistance could be severe.

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## Management of Dystocia due to Lateral Deviation of Neck in a HF Cow and its Correction with Fetotomy - A Case Report

Pramod Kumar, Anand Kumar, Shivendra Kumar Bhalothia and Tapendra Kumar

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### Abstract

*A successful delivery of fetus with lateral deviation of neck and its correction through fetotomy is reported. Post-operative care comprised of administration of antibiotics, anti-inflammatory drugs, ecobolics, along with supportive treatment.*

### Introduction

Dystocia is defined as delayed or difficult to calving, sometimes requiring significant human assistance (1, 2, 3). In cattle and buffalo, the incidence of dystocia is higher as compared to other farm animals (4). Dystocia due to lateral deviation of head and neck constitutes one of the most common types of postural abnormality in r presentation in all species and it may arise during late gestation rather than during birth (5, 6). Fetal causes of dystocia are more common in cows and account for 64.08%. The other causes -head deviation and limb flexion account for 20.4% and 19.4% respectively (7). Amongst all the reasons, the deviation of head and neck of fetus in anterior presentation is the most common (5) and may be in any direction (8).

The lateral deviation of head especially in a dead fetus becomes life threatening to the dam due to uterine contractions in inappropriately treated cases (9). The deviation can be corrected by using mutation and traction, cesarean section or fetotomy (6). This clinical case report describes successful management of dystocia due to lateral deviation of neck in a HF cow and its correction with fetotomy

### Case History and Observations

A pluriparous ten year old HF cow at full term was

presented to the TVCC, College of Veterinary and Animal Science, Bikaner, with a history of labor for more than 8 hours. Obstetrical maneuvers were performed by a local veterinary doctor to relieve dystocia but were not successful. The animal was dull, depressed, recumbent and straining. Per vaginal examination revealed a dead fetus in anterior presentation, dorso sacral position with left deviation of head and neck and extended forelimbs. The cervix was dilated and packed with both fore limbs, the birth canal was dry and vulva was swollen and edematous.

### Handling of Dystocia and Discussion

Based on the observations and per vaginal examination, the case was diagnosed as dystocia due to left lateral deviation of head and neck. Epidural anesthesia was performed at sacrococcygeal space with 2% lignocaine hydrochloride (7 ml) to avoid the straining and pain. Since the birth canal was dry, liquid paraffin was administered into birth canal and uterus as a lubricant. Subcutaneous fetotomy of both the extended fore limbs was done to create space in the birth canal. After proper lubrication, a knife was introduced in the birth canal and the skin was incised from the scapular point to metacarpal bone by the finger fetotome. The pectoral muscles and

the muscles around the scapula were broken. Traction was then applied on the limb under the skin using ropes. The limb was broken off from the scapular joint and taken out. The other limb was removed similarly.

Deviated head and neck was removed by percutaneous fetotomy with the help of Thygysons fetotome. By the help of sand rope carrier loop of wire was loaded around the neck in double barred Thygysons fetotome. The deviated head and neck was brought into birth canal by holding at muzzle area and traction was applied on lower jaw in dorsal and backward direction. For remaining portion of fetus, Cray's Scottler hook was applied and dead fetus taken out by traction which was applied on both fore legs skin part and rope attached to sand rope carrier (**Fig.1**). Cow was treated with fluid therapy once (5% DNS 2 Lt. I/V, Inj RL 1 Lt. I/V, Inj. Metrogyl 300 ml I/V and Inj Calcium boro gluconate 350 ml I/V slow), Inj. Mofoi 25ml I/M (Bovian), Inj Melonex 15ml I/M (Intas), Inj Avilin Vet (MSD), Inj Beeokm L 10ml I/M, Liq Utrasafe (Vetmankind) and Bol. Pesuria 4 Intrauterine (IIL) were administered for 3 days. The cow showed an uneventful recovery without any postpartum complications.



**Fig 1. Fetus showing separated appendages by subcutaneous and percutaneous fetotomy.**

Causes of dystocia can be either fetal or maternal in origin. Among all the dystocia conditions in cattle, fetopelvic disproportion constitute 45% and fetal malpresentation constitute 26% of the cases(10). In the present case the fetus was delivered by subcutaneous and percutaneous fetotomy which prevented the post-operative complications. In most cases these operations are performed within the uterus of the dam in order to remove the fetus per vaginum (11, 12). If the fetus is dead and accessible fetotomy should be the first choice of relieving dystocia when mutation fails to correct the presentation(13, 14, 15, 16).

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## Buffalo Calves Mortality - A Threat of Dairy Industry

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Buffalo calves are the dairy industry of the future. The success of the dairy industry depends on appropriate calf management. Buffalo calves management plays an important role in the development of the dairy sector in the country. Calf care is not only essential for sustenance of the dairy industry but also essential in the wake of preserving and maintaining our good quality germ plasm. Important aspects in the buffalo calves rearing are the health and proper nutrition management. Calf mortality is a common occurrence in buffalo rearing. The season has also been considered as an important risk factor (1). Poor and unscientific management in calf rearing practices such as delayed and under feeding of colostrum, prolonged suckling duration, not practicing weaning, deworming and dusting schedule regularly are some of the common issues.

### Factors Responsible for Calf Mortality

*Tiwari et. al.*, (2) said that calf health care practices in the commercial dairy farms are very poor. In fact these dairy owners find the calf rearing uneconomical. The poor care of calves in the commercial dairies is revealed by the fact that the

mortality rate in buffalo calves in these dairies was 81.09 percent. This is mainly due to the poor management of calves which are not even given the minimum care of naval cord disinfection, timely colostrum feeding, deworming, appropriate space, and proper milk feeding and timely treatment. In fact, it can be said that these dairy owners are really not interested in rearing the calves due to the notion of false economy prevailing among these owners that rearing of calf is not beneficial.

*Afzal et. al.*, (3) reported that the mortality in cattle and buffalo calves ranged from 29.1% to 39.8%. Martin and Wiggins estimated that 20% calf mortality resulted in reduction of 38% profit of a livestock farm. *Dechow et. al.*, (4) concluded that herd management system and selection of sire with high productive life evaluations was associated with lower mortality.

Buffalo calf mortality rate in less than one month of age averages about 10% and varies from 3-30% in individual herds. Losses up to 50% have occurred in large dairy herds. A calf mortality rate of 20% can reduce net profit by 38% (5).

- (1) Antibodies are not transferable from buffalo dam to her fetus through placental membranes and thus they are susceptible to various diseases. In buffalo calves in spite of feeding colostrum, a low antibody titter exists. Immunoglobulin levels have been reported to be 29.73 mg/ml in day olds and the quantity increases to 35.66 mg/ml on the second day of life(5).On the other hand in one estimate it has been noted that colostrum which is the only source of immunoglobulin for the buffalo calf, contains 68.75 mg of immunoglobulin per ml on the first day, 23.75 mg/ml on the second day and 1.01 mg/ ml on the fifth day of lactation.
- (2) Certain meteorological influences may have an effect on calf mortality rate. During the winter months, mortality may be associated with the effects of cold, wet and windy weather while during the summer it may be the hot, dry weather.
- (3) Most of the deaths occur during autumn and winter months before the age of 3 months.
- (4) Neglected tendency especially for male buffaloes calves.

**The causes of mortality in order of priority have been found**

- (a) Pneumonia
- (b) Enteritis
- © Toxaemia/Septicaemia
- (d) Worm infestation
- (e) Bloat

(5) The cause of high mortality in male calves could also be due to neglecting tendency by the management, particularly regarding feeding.

**Measures to Reduce Calf Mortality**

Some of the above prescribed management practices including feeding practices may be followed for minimizing the mortality rate.

**(a) Housing of Calves**

In commercial herds, calves after weaning may be kept in groups in large pens where individual feeding is advocated. The small herds' calves after weaning should be kept in separate pens (24 sq. ft) up to 3 months of age to avoid suckling instinct. Such methods will eliminate the possible calf scour and parasitic infestation

**(b) Feeding of Colostrum**

It is the milk secreted by the udder immediately after parturition and for the following 3-5 days. It contains 20% or more protein, a little more fat, 10 to 100 times more vitamin A, three times more of vitamin D

**Floor Space Requirement for Calves**

Age of calves (months)	Covered area (m <sup>2</sup> )	Open area (m <sup>2</sup> )	No. of calves/pen
0-3	1.0	2	24 / pen
3-6	1.5	3	16 / pen
6-12	2.0	4	12 / pen

and may be tinged pink due to blood corpuscles. It acts as a natural purgative for the calf, cleaning from its intestines the accumulated faecal matter.

Of much greater importance, it is through the medium of the colostrum, antibodies which protects against various bacteria, and viruses are supplied to the newborn calves.

**(C) Parasites of calves**

**1. Endoparasites**

- Deworming should be started from the first week of calf.
- A single oral dose of 10-15ml piperazineadepate is recommended for the calves preferably in the first week of life to control neonatal ascariasis especially in buffalo calves.
- Deworming should be done every month for first 6 months, thereafter once in three months.
- The deworming drugs and dose should be consulted with veterinary doctor.
- Over dose and under dose of deworming drugs should be prevented to check the side effects

**2. Ectoparasites**

- Apply Topical dewormers
- Pourn Method (Flumethrine)
- Water solution (Cypermethrine)
- Ivermectin, Doramectin, etc

**(d) Supplementation of Antibiotic**

1. Antibiotics: Tetracycline, Auriomycine

**(e) Feed Additives**

1. **Prebiotics:** Oligosaccharides
2. **Probiotics:** supplemented with bacteria *Lactobacillus acidophilus* 100 mg/calf/ day in milk for a period of two months. *Saccharomyces cerevisiae* yeast products have the ability to stimulate starter intake in calves.

**(f) Feeding**

1. Calf starter

- They are first day concentrate mixture fed to calves.
- Calves start eating small amount of dry starter from the 2nd week of life.
- A calf starter should be highly palatable.
- It should be high in energy (75% TDN) and contain 20-22% crude protein.
- Calf starter may be fed on free-choice basis until the calf starts consuming about 1-1.5 kg of the starter mix a day after which the amount may be restricted.
- Generally calves reach this stage by 2 ½ months to 3 months of age.
- Milk feeding can be discontinued earliest which the calf is consuming 0.4-0.5 kg of concentrate per day deepening upon the breed. A great variety of calf starters are available.

2. Individual feeding

3. Hay, Lucerne and Straw

**(g) Vaccination of calves as suggested by authors of this article.**

Age	Vaccination
8 weeks before weaning	Black quarter (1st vaccine)
2-4 months	FMD first vaccine
6-8 months	FMD booster vaccine
6 months	Anthrax Black quarter (2nd vaccine) Hemorrhagic septicemia vaccine
4-8 months	Brucella vaccine
Early once	FMD vaccine

**Conclusion**

The need to reduce buffalo calf mortality is important. In future, demand for milk and meat is expected to rise due to rise in human population especially in developing countries. In the commercial dairies, buffalo calves are highly neglected due to the notion of false economy prevailing among these owners that rearing of calf is not beneficial. But the neglect and poor care of calves in these dairies is creating a great damage to the nation in terms of loss of good quality germplasm. Therefore, there is an urgent need to educate the dairy owners about the importance of rearing calves in terms of economic profitability.



After Weaning Milk Feeding Practice



New Born Calf



Examination of calf



Calf pen

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## Microbial Biofilm Formation: A Threat to Treatment of Bovine Mastitis

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### Introduction

Microorganisms consisting of bacteria, virus, and fungi enclosed together with an extra polymeric matrix are called biofilms. Not all bacteria can produce biofilms. About 80% of the gram negative bacteria produce biofilms and these biofilms are resistant to the commonly used antimicrobial agents. Bovine mastitis, a serious problem caused by various microorganisms and the potential role of



Figure 1: Inflammation of Udder in Bovine Mastitis

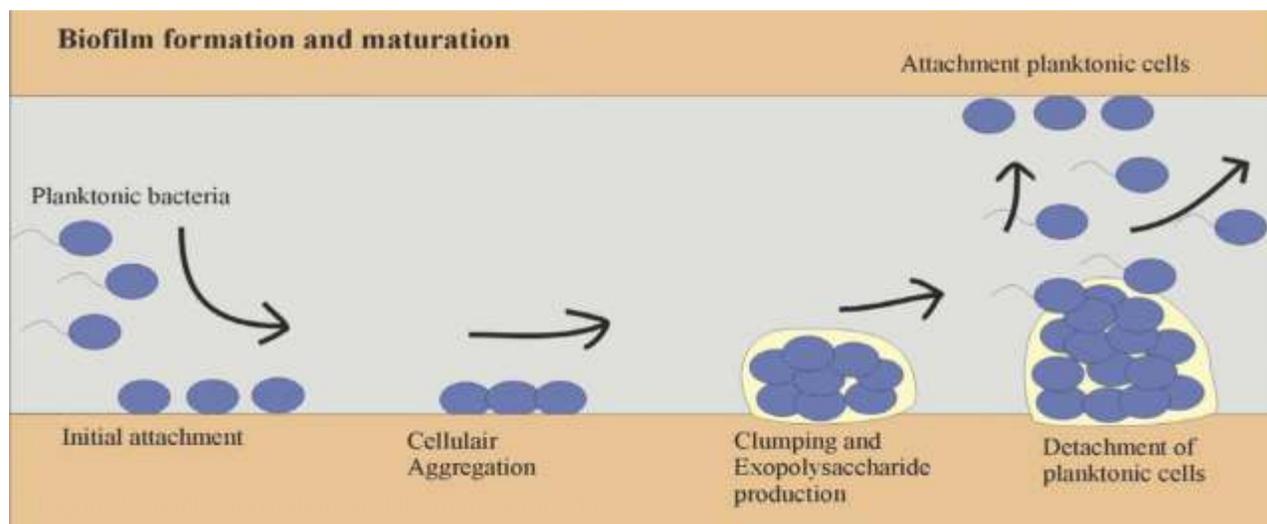
biofilm forming bacteria to establish the cause of chronicity of the infection is discussed in this paper. Due to biofilm formation there is change in genetic as well as subsequent physiological changes in the microorganisms resulting in loss of sensitivity to commonly treated antibiotics.

The main objective of the present paper is to review the general biofilm forming pathogens that cause bovine mastitis and various methods of biofilm formation

### Biofilm

Biofilm produces a secured environment which allows bacteria to thrive in a stressed environment(1). Biofilms are mostly composed of extra-cellular polymeric substance (EPS) like proteins (1-2%) including enzymes, DNA (1%), polysaccharides (1-2%) and RNA along with 97% water. Water forms the largest portion of biofilm which helps in the movement of nutrients inside biofilm (2).

Biofilm formation is a step-wise process in which the bacterial cell undergoes several changes after attachment to a solid surface (Figure 2). Biofilm formation has the following major steps-



- (a) attachment to a surface
- (b) formation of micro-colony of bacteria
- (c) formation of three-dimensional structure by bacterial colony
- (d) biofilm production by bacterial colony, their maturation and detachment or dispersal of bacterial cells from the biofilm (3).

The exo-polymers encasing the biofilm defend the bacteria from the host immune system and therefore, the bacteria are not susceptible to removal by host defence mechanisms and are resistant to antibiotics (4).

Few mechanisms which render the biofilm resistant to antibiotics includeing:

- (a) slow penetration of the antimicrobial agents in biofilm;
- (b) modify growth rate of the organisms within biofilm;
- (c) physiological changes in the bacteria due to the

biofilm, including "persister" cells(1).

**Biofilm formation by Mastitogenic Pathogens causing mastitis:**

Mastitis is caused by a number of aetiological agents, which total to nearly about 137 microorganisms (5)., those are associated with disease pathogenesis. The important aetiological agents of mastitis include are *Staphylococcus aureus*, *Streptococcus uberis*, *Streptococcus agalactiae*, *Enterobacter aerogenes*, *Enterobacter aerogenes*, *Escherichia coli*, *Actinomyces pyogenes*, and *Klebsiella spp.* apart from, some fungi and yeasts (6). Infectious pathogens such as *S. aureus* and *S. streptococcus agalactiae* are usually discussed as the most prevalent organisms responsible for causing bovine mastitis in the dairy herds (7). Biofilm production is considered a critical advantage for microorganisms causing mastitis which expedite chronic bacterial infection in the udder. Table 1 gives the overview of some important mastitis causing bacteria those having biofilm producing ability(8).

Pathogen	Mastitis type	Biofilm formation	sttitis type
<i>S. aureus</i>	Subclinical mastitis	+	
CNS	Subclinical mastitis	+	
<i>E. coli</i>	Clinical mastitis	+	
<i>E. faecalis</i>	Clinical mastitis	+	
<i>S. uberis</i>	Clinical mastitis	+	
<i>S. dysgalactiae</i>	Clinical mastitis	+	
<i>S. agalactiae</i>	Mastitis	+	

Table 1: Main mastitis causing pathogens and their biofilm formation ability; CNS: Coagulase Negative Staphylococci; +: biofilm-forming bacteria (8).

**Detection of Biofilm:**

The ability to produce biofilm by the isolated pathogens is usually determined by Congo red agar (CRA) method, tube method and tissue culture plate (TCP) method.

**Congo red agar (CRA) method**

Mostly Freeman et al. (9) method is followed. Briefly, the medium is composed of Tryptone Soya Agar (Hi-Media, Mumbai) 40 g/l, sucrose 50 g/l, and Congo red 0.8 g/l.. Congo red stain needs to be prepared as a concentrated aqueous solution and autoclaved (121°C for 15 minutes) separately from the other medium constituents, and then added when the agar cools to 55°C. Plates of the medium can be inoculated and incubated aerobically for 24 hours at 37°C. A positive result is indicated by black colonies with dry crystalline consistency. Non-slime producers usually remain pink, though occasional darkening at the centre of the colonies is observed, which gives a bulls eye appearance. An indeterminate result is indicated by the darkening of the colonies but with the absence of a dry crystalline colonial morphology. Isolates presenting two tones of black- bright black and dry opaque black, are classified as biofilm producers, whereas red, pink and Bordeaux colonies are classified as negative.

**Tube method**

The qualitative method of biofilm formation usually is performed according to the method described by (10). Glass tubes filled with 3ml of BHI broth (Hi Media, Mumbai) and 1% sucrose can be inoculated with a loop of pure culture. After 48hr of incubation at 37°C, the content of each tube is decanted. The tubes will then be stained with 1% safranin for 7mins. Then the tubes will be washed with distilled water for 5mins. A positive result is indicated by the

presence of an adherent film of stained material on the inner surface of the tube. Presence of stained material at the liquid air interface alone is not regarded as indicative of slime production.

**Tissue culture plate method**

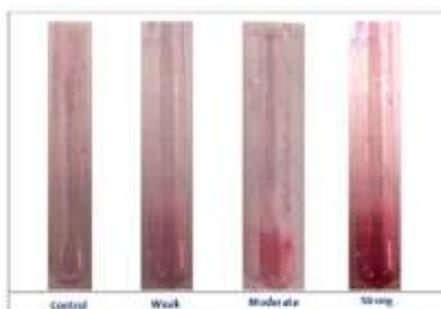
Modified method of *Christensen et al.* (11) usually is followed as the gold-standard method for biofilm detection. Organisms isolated from fresh agar plates are inoculated in 10 mL of trypticase soy broth with 1% glucose. Broths will be incubated at 37°C for 24 h. The cultures are then diluted 1:100 with fresh medium. Individual wells of sterile 96 well-flat bottom polystyrene tissue culture treated plates (\*Sigma-Aldrich, Costar, USA) are filled with 200 µL of the diluted cultures. The control organisms are also incubated, diluted and added to tissue culture plate. Negative control wells contain inoculated sterile broth. The plates are incubated at 37°C for 24 h. After incubation, contents of each well are removed by gentle tapping. The wells are washed with 0.2 mL of phosphate buffer saline (pH 7.2) four times. This removes free floating bacteria. Biofilm formed by bacteria adherent to the wells are fixed by 2% sodium acetate and stained by crystal violet (0.1%). Excess stain was removed by using deionized water and plates are kept for drying. Optical density (OD) of stained adherent biofilm is obtained by using micro ELISA autoreader (at wavelength 570 nm. The interpretation of biofilm production is done according to the criteria of *Stepanovic et al.* (12)

**Treatment of Biofilm forming Chronic Bovine Mastitis**

Treatment of bovine mastitis depends on the aetiological agents and clinical signs. Treatment is usually done using long-acting antibiotics which can be administered parenterally or by intra-mammary infusion. Despite of the extensive use of various



**Figure 3: Detection of biofilm formation by CRA method**



**Figure 4: Detection of biofilm formation by tube method**



**Figure 5: Detection of biofilm formation by tissue culture plate method.**

antimicrobial agents along with chemotherapeutic agents against mastitis, the achieved cure rate is generally less. The underachieved therapeutic results are due to various reasons including biofilm formation. The best solution to deal with biofilms in veterinary practice is through the development of coatings on udder surfaces that can eliminate microorganisms actively. Some promising methods are to incorporate novel approaches such as bacteriophages, enzymes and peptides that remove biofilms and increase antimicrobial activity which may be available in future for treatment (13).

### Conclusion

Mastitis has been a great concern among the dairy sector. It causes a huge economic loss in the milk production. Most of the mastitis bacteria are associated with the biofilm formation which makes them virulent and 1000 times more resistant to normal antibiotic therapy and also renders the host natural defence system inefficient which ultimately leads to chronic mastitis in the affected animals. The role of biofilm in such cases is crucial to formulate the control strategies to manage and prevent such chronic and recurrent mastitis to ensure milk safety and quality. Hence there is a need to develop multivalent biofilm forming mastitis vaccine to control the bovine mastitis.

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## Theileriosis: Impact on Livestock

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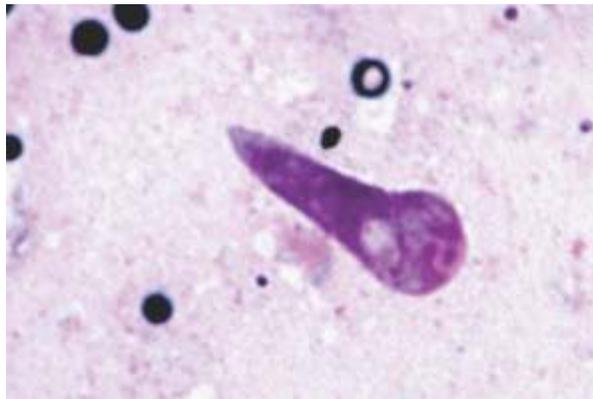
### Introduction

Haemoprotozoan infections are common in cattle and cause huge losses to the livestock industry and are major threat to the dairy industry throughout the world including India. Most of the haemoprotozoan parasites are transmitted by ticks. These tick transmitted diseases are of significant economic importance in Asia and has always been an awful barrier to the survival of both exotic and cross bred cattle in India. Hot and humid climate is very conducive for the development and survival of potential vectors such as ticks and flies and are frequent source of infection to susceptible animals. Theileriosis, babesiosis and anaplasmosis are the three major tick borne haemoprotozoan diseases of crossbred cattle in tropical and sub-tropical regions of the world. Due to the lack of adequate control measures, the annual loss estimated due to theileriosis alone in India is approximately Rs 80 crores. These haemoprotozoan diseases have significant economic impact on cattle production in terms of morbidity, mortality, decreased milk yield and lowered draft power.

Haemoprotozoan diseases have been reported from different geographical regions of India. The



incidence of theileriosis was found to be 27.2% in cross-bred cattle with highest prevalence of 45.4% observed during rainy season in Dehradun district, Uttarakhand, India. In Anand district of Gujarat, the overall incidence of haemoprotozoan diseases in cross bred cattle was theileriosis 37%, babesiosis 10.41% and anaplasmosis 2.82%. In Northern Kerala, theileriosis and babesiosis was reported as 16% and 0.6%, respectively in crossbred cattle in 2011. In 2013 an outbreak of theileriosis in cattle was reported from Punjab with a mortality rate of 4.86% (1).



**Etiology**

Theileriosis occurs from infection with protozoa of the genus *Theileria*, sub-order *Piroplasmorina*. *Theileria spp.* are obligate intracellular parasites. The two most common species of *Theileria* infecting cattle and water buffalo are *T. parva*, which causes East Coast fever and *T. annulata*, which causes Tropical theileriosis. Other *Theileria* species including *T. mutans*, *T. buffeli*, *T. velifera*, *T. taurotragi* and *T. sergenti* can infect domesticated and wild ruminants. *T. lestoquardi* is the most virulent species in sheep and goats. *T. separate* and the non-pathogenic species *T. ovis* also occur in small ruminants (2).

**Species Affected**

*T. parva* can infect cattle, African buffalo (*Syncerus caffer*), water buffalo (*Bubalus bubalis*) and waterbucks (*Kobus spp.*). Symptomatic infections are common only in cattle and water buffalo. *T. annulata* occurs in cattle, yaks, water buffalo and camels. *T. taurotragi* also have been recognized mildly pathogenic or non-pathogenic in cattle. *T. lestoquardi*, *T. separata*, *T. ovis* and other species occur in sheep and goats (3).

**Transmission**

Most of the *Theileria spp.* are transmitted by ticks and these ticks act as biological vectors. Saliva of feeding tick transmits theileria sporozoites to animals. *Rhipicephalus appendiculatus* is the most important vector for *T. parva*. *T. annulata* is transmitted by ticks of the genus *Hyalomma*. *Hyalomma spp.* also act as vectors for *T. lestoquardi*, *T. ovis* and *T. separate*. *T. buffeli* and *T. sergenti* are transmitted by *Haemaphysalis spp.* and *T. mutans* and *T. velifera* are transmitted by *Amblyomma spp.* Large number of ticks of the genus *Rhipicephalus* spread *T. taurotragi* (4).

**Incubation Period**

The incubation period for East Coast fever is 8 to 12 days in experimentally infected animals. It might be

as long as three weeks in naturally infected animals. The incubation period for tropical theileriosis is thought to be approximately 1 to 3 weeks.

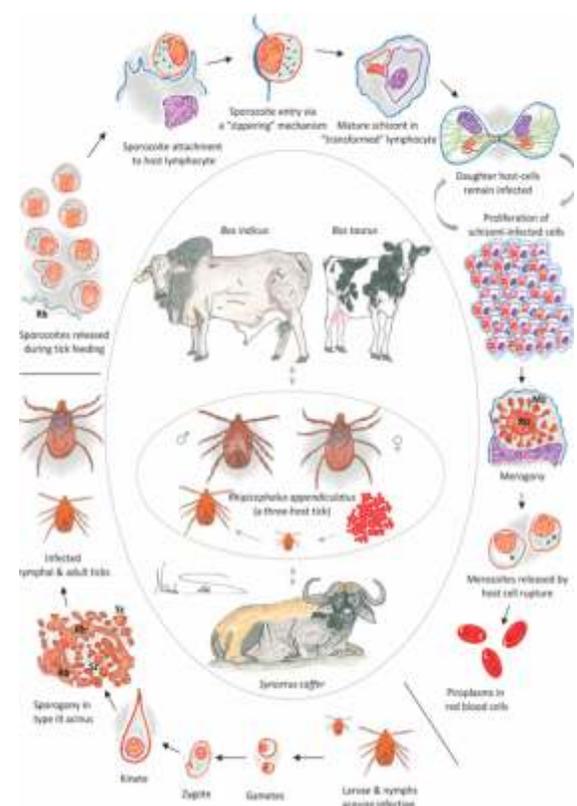
**Pathogenesis**

Usually, *T. parva* and *T. annulata* mature and enter the saliva after the tick attaches to a host; a tick must usually be attached for a few days before it becomes infective. However, if environmental temperatures are high, infective sporozoites of *T. parva* can develop in ticks on the ground and may enter the host within hours of attachment. Transovarial transmission does not occur with *Theileria spp.* In mammalian host, Theileria sporozoites undergo a complex life cycle involving the replication of schizonts in leukocytes and piroplasms in erythrocytes. Cattle that recover from Theileria infections usually remain carrier for months or years. Iatrogenic transmission can also occur via blood (e.g., on re-used needles).

**The Life Cycle of Theileria Annulata Includes Different Stages**

**1. Sporozoite stage**

When infected adult ticks attach to cattle, the sporozoites develop in the tick salivary gland and are injected along with tick saliva to the host. These sporozoites invade the lymphoid cells and schizonts are detected in 10–13 days- this is the pre-patent period of the disease.



## 2. Schizont stage

The schizonts parasitize lymphocytes; proliferate, invade and damage the lymphoid system and produce lesions in the skin, liver and spleen of host.

## 3. Piroplasm Stage

Theilerial piroplasm parasitizes the erythrocytes and causes destruction of these cells with decrease in erythrocyte count and haemoglobin level (5).

### Clinical Signs

Initial clinical signs include, swelling of the draining lymph node, usually the parotid, followed by a generalised lymphadenopathy in superficial lymph nodes such as the parotid, prescapular, and prefemoral lymph nodes which can easily be observed and palpated. Fever ensues and continues throughout the course of infection; this rise in temperature is rapid and may reach upto 106°F (42°C). Other clinical signs may include anorexia, lacrimation, corneal opacity, nasal discharge, terminal dyspnoea and hemorrhagic diarrhoea in later stage. Before death, the animal is usually recumbent and temperature falls.

### Life Cycle of *Theileria* spp.

There is loss of body condition with decreased milk yield. Terminally ill animals often develop pulmonary edema, severe dyspnoea and frothy nasal discharge. Pale mucous membranes (anaemia) or jaundice occurs, as piroplasms cause destruction of red blood cells. During the production of macroschizonts within macrophages, enlarged lymph nodes, generalised loss of condition and muscle wasting is seen due to massive release of cytokines from infected cells.

Some cattle reach a fatal condition called “turning sickness”, where, infected cells block capillaries in central nervous system and cause neurological signs. These neurological signs have been documented in some terminally ill water buffalo; but this “turning sickness” does not seem to be a feature of tropical theileriosis in cattle. Abortions can also be seen. In the acute cases of tropical theileriosis, death occurs in 15–25 days after infection.

*T. lestoquardi* is the most virulent species in small ruminants and often causes fatal disease. Clinical signs of this infection may include fever, anorexia, weight loss, listlessness, lymphadenopathy, edema of the throat, difficulty in breathing, anemia and icterus. Subacute, chronic or mild form of disease can also be seen (6).

### Post Mortem Lesions

A frothy exudate is frequently seen around the nostrils, in trachea and bronchi of infected animals. Lymph nodes are greatly enlarged and may be hyperplastic, haemorrhagic and oedematous. Generally muscles and fat appear normal, but depending on relative acuteness of infection, fat may become greatly depleted. Serosal surfaces have extensive petechial and ecchymotic haemorrhages, and serous fluids maybe present in body cavities. Haemorrhages and ulceration may be seen throughout the gastrointestinal tract – particularly in the pylorus part of the abomasum where necrosis of Peyer's patches can be observed (7).

Lymphoid cellular infiltrations (pseudoinfarcts) as white foci are found in the liver and kidney. Shortly after infection, the lymph node draining the site of tick bite will be enlarged. Anaemia, jaundice, enlarged lymph nodes, muscle wasting, pulmonary oedema and haemorrhagic enterocolitis may all be present at the time of severe clinical disease or death.

It is thought that extensive infection of macrophages stimulates huge outpouring of cytokines, predominantly TNF $\alpha$ , which accounts for many of the lesions observed. Macroschizonts may be seen in infected macrophage type cells within various organs.

### Diagnosis

Theileriosis should be suspected in tick-infested animals with a fever and enlarged lymph nodes. Terminal pulmonary edema and a high mortality rate in introduced breeds are also suggestive of theilerial infection. Indigenous animals with tropical theileriosis may be in poor condition with wasting and signs of anemia.

### Differential Diagnosis

- Heartwater disease (Ehrlichiosis)
- Trypanosomosis
- Babesiosis
- Anaplasmosis
- Malignant catarrhal fever
- Contagious bovine pleuropneumoniae
- The parasites must also be differentiated from other species of *Theileria*.

In sheep and goats, *T. lestoquardi* infections must be distinguished from babesiosis, Rift Valley Fever and malignant catarrhal fever (8).

### Laboratory Diagnosis

Theileriosis is diagnosed by the identification of schizonts in Giemsa-stained thin smear from blood and lymph node biopsy in the live animal. At the time of necropsy, schizonts may be found in impression smears from most of the internal organs. The diagnosis must be confirmed by detecting schizonts. Piroplasms are usually found in the blood of carrier animals.

*Theileria* spp. can be detected and identified via Polymerase chain reaction (PCR) tests and DNA probes. Antibodies against *T. parva* and *T. annulata* can be used for detection using with enzyme linked immune-sorbent assay (ELISA) or an indirect fluorescent antibody (IFA) test. Serological tests may not be sensitive enough to detect all the infected cattle and cross reactions can occur with other species of *Theileria*.

*Theileria* spp. resembles each other but can be differentiated with DNA assays or serological tests. Polymerase chain reaction (PCR) tests can detect and identify *Theileria* in carriers as well as acutely ill animals. Some PCR assays can identify *Theileria* upto the species level.

### Serological Tests

- Indirect Fluorescent Antibody (IFA) test is most widely used diagnostic test for *Theileria* species in which both schizont and piroplasm antigens may be used. The IFA test for *T. parva*, does not distinguish among the different immunogenic stocks.
- Serological tests based on the ELISAs are being used increasingly for the detection of parasite specific antibodies. The new indirect ELISAs for *T. parva* and *T. mutans* based on recombinant parasite-specific antigens have demonstrated higher sensitivity and specificity.

### Samples for Diagnosis

The schizont is the pathogenic stage of *T. parva* and *T. annulata*. It initially causes lymphoid proliferation and later lymphoid destruction.

- Blood or Buffy coat smears air-dried and fixed in methanol and lymph node for demonstration of schizonts.
- Impression smears from lung, spleen, kidney and lymph node, air-dried and fixed in methanol, for demonstration of schizonts
- Lung, kidney, brain, liver, spleen, and lymph nodes for histopathology: demonstration of schizonts and infiltrations of immature lymphocytes

- A nervous syndrome called 'turning sickness' is sometimes observed and intravascular and extravascular aggregations of schizont-infected lymphocytes are observed, causing thrombosis and ischaemic necrosis throughout the brain (9).

### Treatment

There are three effective drugs available for the treatment of theileriosis namely; parvaquone, buparvaquone, and halofuginone lactate, which are used worldwide. Research work regarding the efficacy of these drugs showed that buparvaquone, a second-generation hydroxynaphthoquinone is the most effective treatment available so far. Early treatment with buparvaquone is 100% effective in eliminating the protozoan parasites from the blood and lymph nodes. Tetracycline @ 5-10 mg/kg body wt., Oxytetracycline @ 10 mg/kg body wt. and Buparvaquone @ 2.5 mg/kg body wt. (I/M) twice in 48 hours are also found to be effective for treatment.

### Prevention and Control

Theileriosis transmission usually does not occur by casual contact. The newly introduced infection to an area may be eradicated with movement controls, by culling infected animals and by preventing ticks from becoming infected. In endemic areas, acaricides can be used to decrease the tick burden. Other methods of tick control such as rotational grazing and use of nets over windows are also useful to control and reduce the chance of disease occurrence. The transfer of blood between animals must also be avoided. Antiparasitic drugs are effective in animals with clinical signs but animals may remain carriers. Treatment is most effective if the disease is diagnosed in early stages of infection (10).

### Sanitary Prophylaxis

Bovine theileriosis is generally controlled by the use of acaricides to kill ticks, but this method is not sustainable because acaricides are too expensive and cause environmental damage and over time ticks develop resistance to them requiring newer acaricides. More sustainable and reliable methods for the control of theileriosis include combination of strategic tick control and vaccination of animals. However, these are yet to be successfully applied on a large scale in endemic areas. Sanitation and disinfection measures are not generally effective in preventing transmission of theileriosis (11).

### Live Attenuated Vaccines

Reliable vaccines of known efficacy have been developed for *T. parva* and *T. annulata*. For *T. annulata*, the vaccine is prepared from schizont-infected cell lines that have been isolated from

cattle and attenuated during in-vitro culture. The vaccine must remain frozen until shortly before administration. Vaccination against *T. parva* is done via a subcutaneous dose of tick derived sporozoites vaccine and a simultaneous treatment with a long acting tetracycline formulation.

A robust immunity develops in recovered animals to homologous challenge, which usually lasts for the lifetime of an animal. Immunisation of animals with a broad-spectrum immunity is desirable to cover a range of *T. parva* strains that exist in the field. Immunised animals usually become carriers.

### Recombinant Vaccines

Experimental sub-unit vaccines are being developed for tropical theileriosis and ideally contain antigens from both sporozoite (as the p67 protein) and schizont stages. An improved p67 vaccine has been tested in the field and might be available soon (12).

### Conclusion

Haemoprotozoan disease like theileriosis is very common in cattle and causes devastating losses to the livestock and dairy industry throughout the world including India. It causes great economic losses and is a barrier to the survival of exotic and cross bred cattle in India. It affects the cross breeding programme and hence a major barrier for improving the genetic make-up of livestock. There is a need to educate people about the theileriosis infection. In addition, effective control strategies like mass vaccination using the available vaccines in India should be put in place to reduce the prevalence of theileriosis infection in the animals.

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## An Overview of Brucella Vaccines

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### Brucella Vaccine

The importance of vaccination in the control of brucellosis is well recognized. Since long, works have been carried out to find a vaccine which confers good resistance without disadvantages till date. An ideal vaccine has not been developed which has the qualities attributed to an ideal biologic.

These include

- Immunogenesis of long duration.
- Minimum interference with diagnostic tests.
- Easy production with long stability and easy storage and
- A minimum of adverse effects in vaccinated animals and harmlessness to humans.

There are usually three criteria to evaluate the effectiveness of vaccines: i) use in laboratory animals and date extrapolation to natural host, ii) use in natural hosts in controlled conditions and results compared with unvaccinated controls, and iii) use in field conditions where pre – vaccination and post – vaccination prevalence are compared and/or those of subpopulation of vaccinates and

controls.

Vaccines are often classified as either living or dead. In general with Brucella antigen, the living vaccine gives a more complete and lasting immunity. The bacteria multiply in the host usually for limited periods, and the resistance conferred is largely of the cell mediated type. Antibodies are considered to play a minor role in protection and numerous studies in cattle have shown a poor relationship of post – vaccinal circulating antibody to subsequent immunity (1).

### A. Live Vaccines

#### I. *Brucella Abortus Strain 19*

The isolate was recovered from the milk of a jersey cow in June 1923 and was virulent. The culture was maintained at room temperature for 1 year or more and when tested in guinea pigs, had lost its virulence. It was the 19th of the stock culture series by Buck.

The outstanding characteristics of *B. abortus* strain 19 are (i) low stable pathogenicity, (ii) relatively high immunogenicity, and (iii) antigenicity. The attenuation, cultural and other biological characteristics of Strain 19 are stable and where not

altered by several passages through guinea pigs or by intravenous passages through pregnant cattle.

Strain 19 was first used to produce a standardized liquid vaccine in the U.S. in 1939 and introduced for field usage in 1941. Later on, it was produced in lyophilized form. The occurrence and persistence of serum antibodies following Strain 19 vaccination interfere with detection of infected cattle, which is the major disadvantage.

The magnitude and duration of serum antibodies following vaccination are directly related to many factors viz. age at vaccination, dosage method of administration and status of pregnancy. Vaccination in early calf hood has usually been recommended so that serologic tests will be negative by breeding age. Over 90% of calves vaccinated at 3 – 8 months of age can be expected to be negative within 9 months. There is a greater tendency to retain titres if cattle are pregnant when vaccinated, regardless of vaccination method. One dose of vaccine will confer adequate immunity for at least five pregnancies. The revaccination gave better immunity but that the disadvantages of serology, outweighed the benefits.

Strain 19 multiplies only short time after inoculation and rarely persists for an extended period of time and this may be decreased by vaccinating cattle at an early age. Strain 19 is markedly less violent than other biovars of *B. abortus*. In spite of considerable research efforts to find a superior alternative, strain 19 remains the most acceptable vaccine against brucellosis in cattle because of its safety, immunogenicity, practicality of production, and convenience of use in cattle.

### II. *B. abortus* Strain 45/20

*B. abortus* Strain 45 was first isolated in 1922 in Great Britain from a cow. It is a rough strain and was developed by 20 passages in guinea pigs. It was used for some years in cattle as a live strain, but studies showed it could revert to virulent smooth mutants. Then, killed cells in saline or with many types of adjuvant are tried.

### III. *B. melitensis* Rev-1

It is an attenuated strain that has been widely used in the control of brucellosis in sheep and goats. The evaluation of Rev-1 for possible use in cattle has been for two major reasons. Firstly, in countries where *B. melitensis* infection in sheep and goats is wide spread, cattle also become infected with this species. Therefore, vaccination of cattle with a homologous species of *Brucella* might be advantageous. Secondly, since Rev.1 is a potent immunogen, therefore, a lower dose of than Strain 19 could provide equal or superior immunity and post – vaccinal antibody could be reduced.

The Rev.1 vaccinated heifer showed no vaccine organisms in milk or placenta following calving and blood titer in all vaccinates disappeared within six months. Calfhood vaccination with Rev.1 in dose of one tenth and on hundredth of that Rev.1 is superior immunogen than strain 19, its use in cattle has been very limited.

### III. *M. Vaccine*

A mucoid growth phase of *B. suis* was used in guinea pigs in 1940 and later in cattle under experimental and field conditions. It has found to have inferior immunogenicity than Strain 19 although post – vaccinal agglutinins were less.

### IV. 104 – *M. Vaccine*

The strain was isolated from a cow which aborted in 1929 and developed a vaccine in Russia in 1950. It is reported to be stable in antigenic structure, degree of virulence and immunogenicity. The post – vaccinal antibodies are less than STRain 19.

### V. *B. suis* Strain 2

The attenuated strain of *biovar 1* was developed in china. Its virulence is approximately the same as Strain 19 and is stable. It is given orally via drinking water. A dose of 25 to 50x10<sup>6</sup>(count is not clear) live cells in cattle gave a protection rate of 71.4%. Excellent results were reported when used on dairy farm and the vaccine did not induce persisting antibodies.

### VI. *B. abortus* Strain RB 51

It is a genetically stable, rough mutant that lacks the polysaccharide O-side chains on the surface, which are responsible for the development of the diagnostic antibody responses of an animal to brucellosis. Therefore, strain RB51 vaccine does not stimulate the production of antibodies on standard diagnostic tests. The vaccine dose produces other types of antibodies that can be detected with a special assay to detect if an animal has been vaccinated. It produces a cell-mediated response that is primarily responsible for its protection against brucellosis.

Strain RB51 is as efficacious as *B. abortus* Strain 19 but is much less abortigenic in cattle and was in use in USA from 1996. It does not produce any clinical signs of disease after vaccination, nor does it produce a local vaccination reaction at the injection site. The organism is cleared from the blood stream within 3 days and is not present in nasal secretions, saliva or urine. Immunosuppression does not cause recrudescence, and the organism is not spread from vaccinated to non – vaccinated cattle. The vaccine is safe in all cattle over 3 months of age. Calves must be vaccinated with the calf does (10 to 34 billion organisms) between 4 and 12 months of

age. Only animals in high risk areas should be vaccinated.

In case of human exposure, strain RB51 is sensitive to range of antibiotics used in the treatment of human brucellosis, but is resistant to rifampicin and penicillin.

## B. Killed Vaccines

### I. Whole cells

#### a. *B. melitensis* A38

The killed 38 adjuvant vaccine induced high and persistent titres, caused persistent and unacceptable local reaction at the site of infection.

#### b. *B. abortus* Strain 45/20

The killed cells in saline or in many types of adjuvant are tried. The nature of the adjuvant plays a vital role in effectiveness and cause marked local reactions at site injection therefore, it is not preferred over strain 19.

#### c. *B. abortus* Strain 19 Head Killed

A head killed and treated with formal vaccine using *B. abortus* Strain 19 was prepared. The antigenic sites were saturated with hyper immune bovine serum to deprive them of the ability to produce agglutinins. The studies in controlled and field herds found immunogenic weak, thus provide poor protection.

### II. Extracts

Several extracts were used, like (i) sodium dodecyl sulfate extract prepared by treatment of living cells of Strain 45/20 by ethanol precipitation (ii) salt extractable protein preparation (CSP) or chemically modified CSP (DCSP) from Strain 19 in Freud's complete adjuvant. But none of the preparation was an effective immunogen and preparation of a

nonliving vaccine which has the desirable property of immunogenesis without adverse effects will be difficult.

### C. Sub – unit Vaccine:

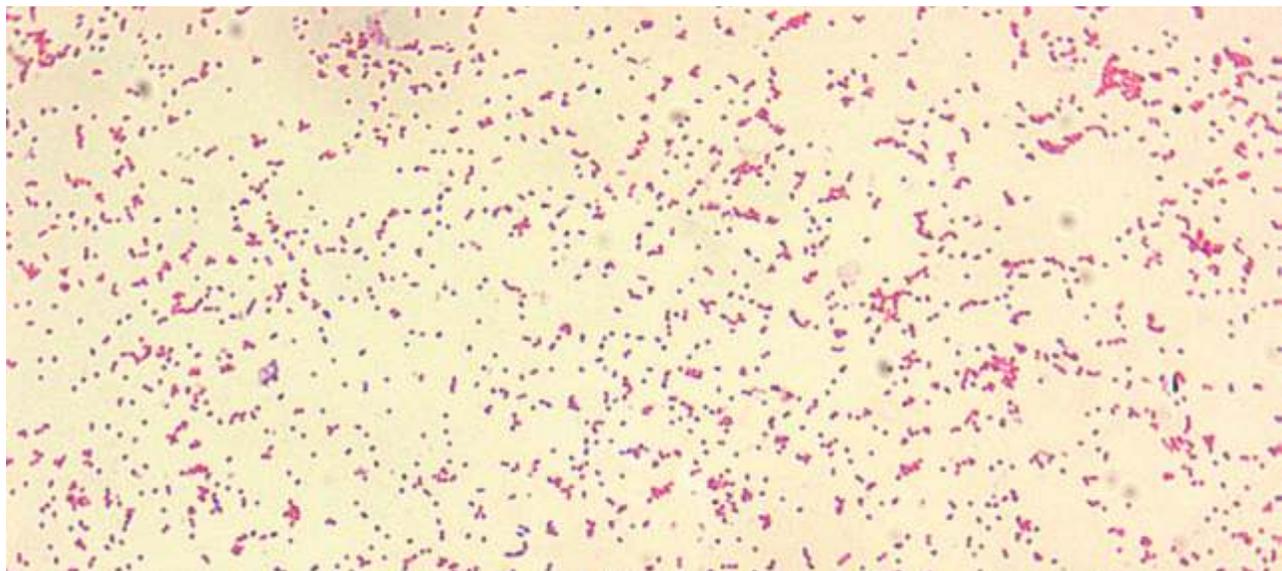
Research is going on produce sub-unit vaccine but the results have not be encouraging.

### D. Phage Lysate Vaccine

A phage lysate bacterin is defined as a composition comprising bacteria and/or fragments of bacteria killed by lytic bacteriophage which will induce an immune response, either cellular or humoral or both. The bacterial components in such a composition are produced by infection of bacteria by lytic bacteriophage followed by production of new bacteriophage particles released in a subsequent lysis of the bacteria in what is termed a lytic burst. Substantially all of the bacteria in the suspension are killed by the phage infection, meaning that preferably 99% of the bacteria in the suspension are killed by the bacteriophage (2). If any residual live bacteria are present are removed by means such as centrifugation or filtration so as to render the bacterin bacteriologically sterile, particularly for the bacterial host organism used to make the bacterin.

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## Swine Dysentery: A Re-Emergent Challenge for Profitable Pig Production

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### Abstract

Swine dysentery (SD or bloody scours) is a “gut” disease which is very expensive to treat medically and difficult to effectively remove once pigs and facilities are contaminated. It is a severe mucohaemorrhagic enteric disease of pigs caused by *Brachyspirahydysenteriae*; it causes a large impact on pig production and leads to severe losses due to mortality and sub-optimal performance (1). In the late 1990's veterinarians and farmers started considering bloody scours in pigs to be a problem of the past. However, it began re-emerging in many parts of the world since 2005 (2).

*Brachyspirahydysenteriae* is a Gram negative, motile, helically coiled (spiral-shaped), anaerobic bacterium which is genetically distinct from other spirochetes, and have adapted to occupy specialized niches in the large intestines of various birds and animals, including swine. Transmission mainly occurs by ingestion of infected faeces of affected animal. *Brachyspirahydysenteriae* may also spread in faeces by animal caretakers who do not change their clothing or footwear. Factors which promote the transmission, establishment and persistence of the disease are the presence of vectors, diets with high energy and protein and poor husbandry practices (3).

*B. hydysenteriae* can colonize the large intestine without the support of other microorganisms. However, several reports suggest that other colonic anaerobes act as supporting organisms for establishing infection. The animal affected by swine dysentery exhibits pathological lesions like muco haemorrhagic colitis coupled with muco fibrinous exudates and epithelial layer desquamation. Microscopically the colon of affected animals shows presence of organism in intercellular gap of epithelium, goblet cells of colonic crypts and disrupted epithelial cells of lamina propria of intestine. Inflammatory cell infiltration and fibrin deposition along with epithelial necrosis are also noticed microscopically (4).

The re-emergence of *Brachyspira* species including antimicrobial resistant strains of *B. hydysenteriae* and novel species like *B. hampsonii* as pathogens has re-ignited significant concerns for pork-producers worldwide.

Swine dysentery (SD or bloody scours) is a severe mucohaemorrhagic enteric disease of pigs caused by *Brachyspira hydysenteriae*; it causes a large impact on pig production and leads to severe losses due to mortality and sub-optimal performance (1). The disease virtually disappeared from many regions during the 1980's and 1990's because of a better understanding of cause and transmission, availability of swine dysentery free breeding stock, better bio security/sanitation, and availability of cost-effective treatment/elimination drugs for use in a herd eradication program. However, it began re-emerging in many parts of the world since 2005 (2). Accordingly, there has been a renewed interest in swine dysentery and *Brachyspira* spp. infections in pigs, particularly in areas where the disease was previously eliminated.

The present article discusses the salient features of Swine dysentery emphasizing the etio- pathogenesis and pathology of the disease.

### Etiology

*B. hyodysenteriae* is a Gram negative, motile, helically coiled (spiral-shaped), anaerobic bacterium. It is 6–8.5 µm long, 0.32–0.38 µm wide and has 7–14 periplasmic flagella inserted at each cell end. The cell is covered by a loose outer membrane. *B. hyodysenteriae* outer envelope contains Lipooligosaccharides (LOS), a semirough form of lipopolysaccharide (3). Several CDS (protein coding sequences) predicted as putative virulence factors have been identified and proposed as virulence factors in the bacterial genome. *B. hyodysenteriae* was shown to differ from all the other spirochetes, including *Leptospira*, *Borrelia* and *Treponema*, in signal transduction and in amino acid transport and metabolism systems (4).

### Epidemiology

Swine Dysentery has a worldwide distribution. The incidence varies in different countries and regions, and changes with time. SD remains a relatively common and important endemic problem in many countries in the European Union, South America and Southeast Asia (2).

### Host Range

*Brachyspira hyodysenteriae* naturally infects pigs (including feral pigs) and occasionally some species of birds (rheas, chickens, ducks, and geese). On infected farms it has been isolated from mice, rats, dogs, and feral birds, including seagulls (5).

### Transmission

*Brachyspira hyodysenteriae* is shed in faeces for variable periods. The incubation period of the disease is from 2 day to 3 months, but usually disease occurs 10–14 days after exposure. Transmission mainly occurs by ingestion of infected faeces of affected animal(6). Wild rodents are potential vectors of *Brachyspira spp.* (7). Wild boars may also act as a potential source of infection (8). Apart from feral animals, domestic animals present in the farms, principally dogs, can acts as a reservoir of *Brachyspira spp.* Wild-living water-birds and laying hens transmit or disperse the pathogens in their migration by excretion of organisms in faeces (9). Insect vectors like cockroaches and flies harbour *Brachyspira spp.* and constitute a reservoir and source of infection for pigs (10).

### Pathogenesis

*Brachyspira hyodysenteriae* following ingestion from faeces survives the acidic environment of the stomach due to the covering of organism with mucus from dysentery of shredded animal. They

eventually reach the large intestine, where it invades the mucus and crypts of the mucosa in the large intestine and penetrates into colonic enterocytes and goblet cells(11). Organism at epithelial cells of lumen and crypts of caecum and colon stimulates the outpouring of mucus. Then they produce tissue destruction by Hemolysins and Lipooligosaccharides (LOS) which plays main role in damaging epithelial barrier in colon(11). Epithelial necrosis and vascular leakage may lead to conditions favouring overgrowth of opportunistic bacteria. Subsequent sub mucosal invasion by secondary bacteria and the protozoan *Balantidium coli* may contribute to lesion formation. No production of septicaemia has been noticed (12). Diarrhoea appears as a result from colonic malabsorption due to a failure of epithelial transport mechanisms to actively transport sodium and chloride ions from lumen to blood, and not from the activity of enterotoxins and / or prostaglandins released from the inflamed tissues, because there is no evidence of increase in cAMP and cGMP in colonic mucosa of dysenteric pigs (13).

### Clinical Signs

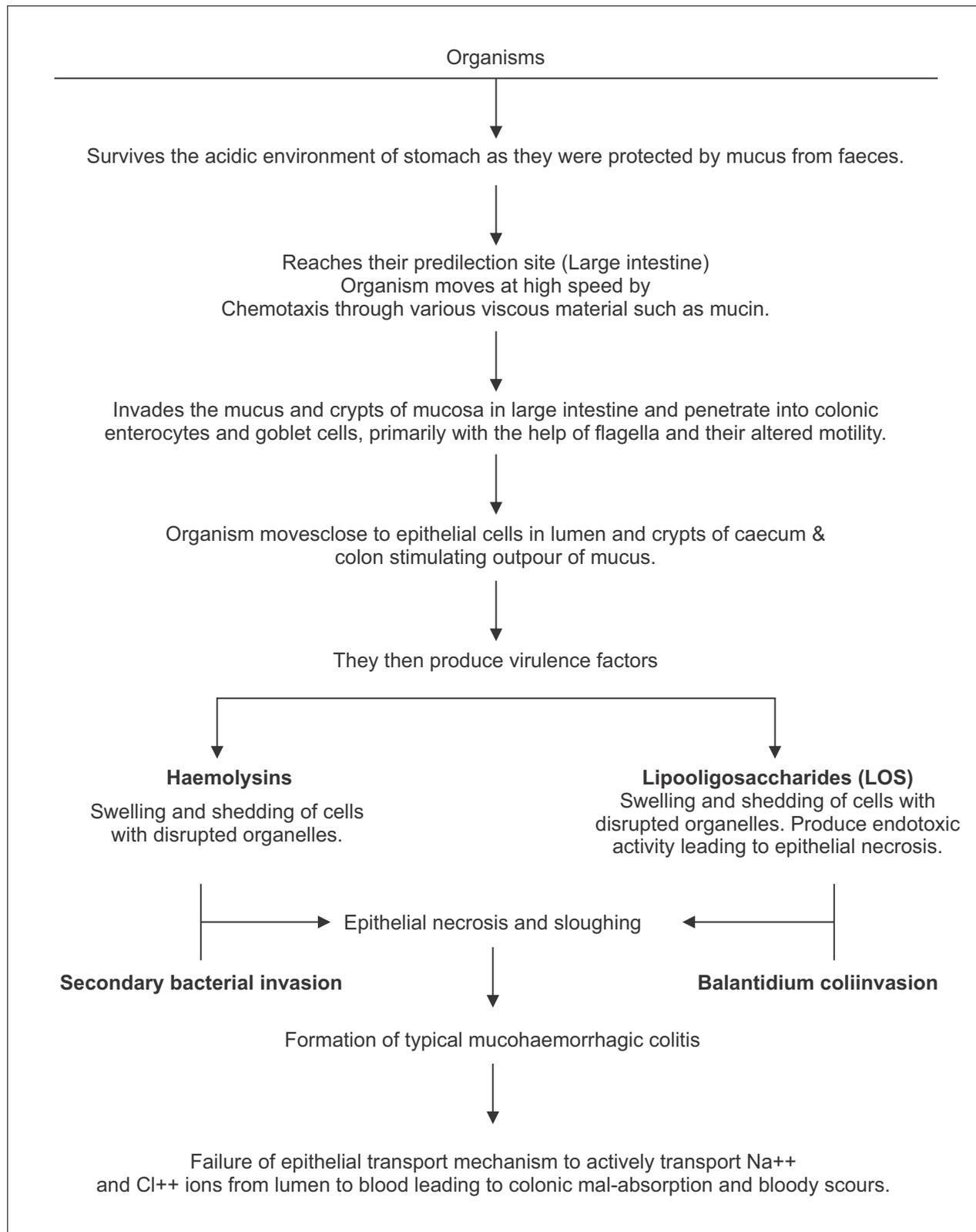
The first evidence of swine dysentery is usually soft, yellow to grey faeces. Partial anorexia, increased rectal temperature of 104–105°F (40–40.5°C) and arched back due to abdominal pain can be seen (14). A few hours to days after infection, large amounts of mucus and often flecks of blood are found in the faeces. This progresses to watery stools containing blood, mucus, and shreds of white mucofibrinous exudate, with concurrent staining of the perineum (3). Appearance of white mucofibrinous grains in the stools is pathognomonic as the disease progresses.

Occasionally, pigs are per acutely affected and die within a few hours. Most pigs recover over several weeks, but their growth rate remain depressed. On endemically infected swine farms, clinical signs often recur cyclically at 3- to 4-week intervals in affected animals. Reappearance may occur after removal of antimicrobials from the water or feed(2).

### Gross Lesions

Typical changes in acute swine dysentery include hyperaemia and oedema of the large intestinal walls and mesentery. Mesenteric lymph nodes may be swollen. Small amount of clear ascitic fluid can be seen. There may be white, slightly raised foci on the serosa caused by submucosal aggregates of mononuclear cells. The mucosa is usually swollen, with loss of the typical rugose appearance, and is covered by mucus and fibrin, with flecks of blood. The colonic contents are soft to watery and contain

Flowchart depicting the stepwise pathogenesis of Swine dysentery



exudates (3).

As the condition progresses the oedema in the colon wall may decrease. Mucosal lesions become more severe, with increased fibrin exudation and formation of thick, mucofibrinous pseudo membranes containing blood. As lesions become chronic the mucosal surface are usually covered by a thin, dense, fibrinous exudate, resembling superficial necrosis (15).

Lesions start in the centrifugal and centripetal coils near the apex of the colon and may extend to the whole colon through the caecum, and in some instances the whole large intestine may become involved (16). The distribution of lesions within the large intestine varies. Sometimes the entire organ may be involved, while at other times only certain segments may be affected. Lesions tend to become more diffuse in the later stages of the disease. Hepatic congestion, hyperaemia or congestion of the gastric fundus may occur; however, such lesions are not specific for SD (17).

### Microscopic Lesions

Significant microscopic lesions are found only in the caecum, colon, and rectum. Typical acute lesion includes a thickened mucosa and submucosa, due to the vascular congestion and extravasation of fluids and leukocytes in the affected portions of intestine. Goblet cell hyperplasia may be present and the epithelial cells at the base of the crypts may be elongated and hyperchromic (18). There may be spirochetes seen in goblet cells of the colonic crypts and the intercellular gaps in the epithelium. Spirochetes may also be found attached to the luminal surface and inside of the disrupted epithelial cells (19).

There may be an increase in number of leukocytes in the lamina propria, with accumulation of neutrophils in and around capillaries near the lumen. Some spirochetes may be seen in the lamina propria, particularly around blood vessels. Clumps of epithelial cells may detach from the lamina propria, resulting in exposure of capillaries followed by focal areas of haemorrhages. Bleeding may occur from small vessels under the areas of eroded epithelium, and this may be invaded by the colonic microbiota (20).

Later changes include accumulation of fibrin, mucus and other cellular debris in mucosal crypts and on the luminal surface of the large intestine. Superficial necrosis of the mucosa may be extensive, but deep ulceration is not typical. Increased numbers of neutrophils may be seen throughout the lamina propria. Chronic changes are not very specific, with less hyperaemia and oedema being present. There is often more advanced superficial necrosis of the mucosa, which usually

has a thick, fibrinous pseudo membrane (21).

### Laboratory Diagnosis

Spirochetes can be seen in smears from the colonic mucosa or faeces, but this does not distinguish between the different *Brachyspira species* (3). A definitive diagnosis of SD requires the demonstration of *B. hyodysenteriae* which can be done by selective anaerobic culture and analysis of phenotypic properties of the isolated organisms from the culture. Trypticase soy agar, C.V.S. media and Blood agar are commonly used for the cultivation of the organisms (4).

Antigen based methods, including fluorescent antibody test, growth-inhibition test, and rapid slide agglutination test have been described for identification of *B. hyodysenteriae*, but these have largely been superseded by polymerase chain reaction (PCR) testing (22). PCR amplification of specific sequences are widely used for detection and identification of *B. hyodysenteriae*. The most usual targets for amplification are portions of the 23S rRNA gene, the nox gene and the tlyA gene (23).

### Conclusion

There-emergence of *Brachyspira species* including antimicrobial resistant strains of *B. hyodysenteriae* and novel species like *B. hamptonii* as pathogens has re-ignited significant concerns for pork-producers worldwide. Limitation in the success for vaccine development and efficacy has marked the disease as one of the potential re-emergent pathogen of swine population. Routine surveillance at local, national and international level is required not only to monitor *Brachyspira species* infections in pigs, but also carriage in other species which may act as reservoirs of infection (particularly migratory water birds). Purchased/ imported animals should be quarantined for at least 4 weeks and treated to eliminate *B. hyodysenteriae*. Infectious materials (fomites such as workers boots, farm implements, feed or animal trucks) must be properly sanitized for prevention of the disease. Apart from the therapeutic interventions, proper managemental measures are necessary to control the disease from its spread. The re-emerging status of the disease calls for further innovation in various aspects of pig rearing for a healthy and profitable herd free from swine dysentery.

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## Traumatic Hyphema in Kathiawari Foal: A Case Report

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### Abstract

The present case study reports traumatic Hyphema - the ocular emergency in foal. The diagnosis was made through history and direct ophthalmoscope. The successful therapeutic management was made by recommended scheduled of treatment which includes mydriatics, topical and systemic corticosteroids.

### Introduction

Hyphema is blood in the anterior chamber of the eye. It may appear as a reddish tinge, or it may appear as a small pool of blood at the bottom of the iris or in the cornea. Hyphema in horses is common in association with trauma. It may resolve spontaneously once the underlying pathology has been removed (1), and prognosis is reportedly good if blood fills less than half the anterior chamber. However, in the absence of perforation of the globe, hyphema will usually result in glaucoma and has a poor prognosis (2). The present communication reports a rare case of Traumatic Hyphema and its clinical management in Kathiawari Foal.

### Case History and Clinical Examination

The two years old Kathiawari foal was presented to TVCC, Junagadh with history of partial blindness, swelling and reddening of both eyes since last 7 days due to blunt object trauma. The animal was otherwise healthy with normal clinical parameters. The direct ophthalmoscopic examination revealed partial filling of anterior chamber with settled blood layer (Fig.1). Based on history and clinical examination, the case was diagnosed as partial Traumatic Hyphema.

### Treatment and Discussion

Hyphema may be idiopathic or may result from many other causes viz., trauma, clotting disorder, highly vascularized tumor, severe uveitis, retinal dysplasia etc. Erythrocyte release in anterior chamber undergoes phagocytosis by the cells lining the trabecular meshwork. The surface of the eyeris provides fibrinolys in which aids in resolving clots in the anterior chamber (3). The recommended treatment of hyphema includes surgical innervations, corticosteroids, intracameral tissue

plasminogen activators etc. The treatment was started under complete restraining of animal with subconjunctival administration of 0.25 ml of Dexamethasone at upper and lower lining of conjunctiva along with and Inj. Tribivet @ 10 ml I/V on the day of presentation. The animal showing improvement on second day after treatment so treatment is further repeated with CIPLOX-Dc eye drops (containing ciprofloxacin 0.3 % w/v, dexamethasone 0.1 % w/v) @ 2-3 drops t.i.d for 20 days where as Inj. Tribivet @ 10 ml I/V, Inj. Flumxine meglumine @ 1.1 mg/kg I/V and Inj. Prednisolone @ 10 ml I/M given for 5 days (Fig.2). The animal was showing complete recovery after 20 days of treatment. The treatment of hyphema is controversial because of conflicting experimental results with different drug regimens in different species. In the vast majority of patients surgical drainage of the hyphema is not useful because rebleeding is frequent.

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**Fig.1. Showing presence of hemorrhage in anterior chamber of eye**

**Fig.2. Showing improvement in condition after 10 days post treatment**



## SIROHI

**a) Distribution:** Sirohi district of Rajasthan. The breed also extends to Palanpur in Gujarat.

**b) Numbers:** The total goat population in the Sirohi distribution area, according to the 1972 census, was 0.295 m, of which 0.007 m adult males and 0.204 m adult females

**c) Climate:**

	Average	Range	
<b>Average monthly temperature (°C)</b>			
Minimum	16.5 - 24.9	9.3-22.3	19.3-31.5
Maximum			
<b>Average monthly relative humidity (%)</b>			
Morning	55 - 48	29-95	24-91
Evening			
<b>Annual rainfall (cm)</b>	169		

**d) Breed characteristics:**

**i) Size:**

	Adult male	Adult female
<b>Body weight (kg)</b>	50.37 ± 2.52 (16)	22.54 ± 0.17 (343)
<b>Body length (cm)</b>	80.0 ± 1.02 (16)	61.3 ± 0.2 (343)
<b>Height at withers (cm)</b>	85.6 ± 1.4 (16)	68.4 ± 0.2 (343)
<b>Chest girth (cm)</b>	80.3 ± 1.0 (16)	62.4 ± 0.2 (343)

**ii) Conformation:** Compact, medium-sized animals. Coat colour predominantly brown, with light or dark brown patches; a very few individuals are completely white. Most animals are wattled. Ears are flat and leaf-like, medium-sized and drooping; ear length: 18.8 + 0.6 cm (15). Both sexes have small horns, curved upward and backward; horn length: 7.7 ± 0.15 cm (144). Tail is medium in length and curved upward; tail length: 16.7 ± 0.14 cm (153). Udder is small and round, with small teats placed laterally.

**e) Flock structure:** Average flock size is 60 (range: 10 to 200), containing 1 adult male, 42 adult females and 17 young.

**f) Reproduction:** Under farm conditions, kidding percentage: (5): 89.3% (328). Litter size: singles: 91.5%; twins: 8.5%.

**g) Mortality (5):** 0 to 3 months: 1.9% (219); 3 to 12 months: 4% (179); adults: 2.5% (451).

**h) Breeding:** Generally pure breeding. Males are selected on size from within flocks. There is some introduction of Marwari for increasing hair production.

**i) Performance**

• **Milk:** Average lactation yield (5, 7): 71.18 ± 1.55 kg (219); length: 174.8 ± 2.75 days (219).

• **Meat:** body weight (kg) (source: 5)

<b>At birth</b>	2.82 ± 0.02 (309)
<b>At weaning</b>	9.92 ± 0.12 (288)
<b>6 months</b>	13.48 ± 0.19 (144)
<b>9 months</b>	16.95 ± 0.21 (118)
<b>12 months</b>	21.27 ± 0.23 (117)



In individual feed-lots, from 3 to 6 months of age (4, 5): average daily gain: 61.4 ± 5.36 g (23); efficiency of feed conversion (%) (4, 5): 10.1 ± 0.93 (23). Age at slaughter: 6 months. Dressing percentage on pre-slaughter live-weight basis (4, 5): 47.3 ± 0.9 (13). Bone/meat ratio (5): 1:4.497 ± 0.357 (9).



## Dystocia Due to Breech Presented Foetal Anasarca in a Marwari Doe: A Case Report

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### Introduction

The major contributory factor in the economic losses in goat farming is the dystocia as a result of which death of the either foetus or dam occurs. Reports of the foetal anasarca are very less reported in small ruminants. Foetal anasarca is characterized by wide-spread swelling of the skin due to subcutaneous and inter-muscular accumulation of fluid in muscles, umbilicus and legs resulted into the formation of the generalized oedema (1). The affected foetus is usually carried to term, and concern is caused by the lack of progress in second-stage labour. This is due to the great increase in foetal volume caused by the excess of

fluid in the subcutaneous tissues, particularly of the head and hind limbs (2). In the case of the head, there is so much swelling that the normal features are masked and the resultant appearance is quite grotesque. It is an interesting point that an undue proportion of these anasarcaous foetuses are presented posteriorly, in which case the enormous swelling of the presenting limbs is very conspicuous (3). Generally, in such cases, surgical manoeuvre is required however present communication reports a successful per vaginal delivery of the kid.

### Case History and Clinical Observation

A 3-year old pluriparous Marwari doe with full term gestation was presented at the clinic with the history

of discomfort, constant abdominal straining since four hours, and ruptured water bag. Doe was earlier attended by local quack but was unable to deliver foetus. The external examination revealed swollen vulva, continuous abdominal straining, and ruptured water bag. Physiological parameters recorded were in the normal range except, rectal temperature (103.8 °F) and slight tachycardia. Per vaginal examination revealed dilated cervix, posterior (breech) presentation and dorso-sacral position of the kid, having oedematous mild distended abdomen with absence of the vital reflex in the kid.

### Treatment and Discussion

After proper lubrication of the birth canal using liquid paraffin and rinsing the perianal region with 1 % potassium permanganate lotion, attempts were made to deliver the kid by the forced traction, but it was failed due to large sized kid. Hence, it was decided to drain out the anasarca fluid with the help of the obstetrical knife. Per vaginally, knife was inserted and anasarca fluid was evacuated by puncturing abdomen. Kid size got reduced and with further lubricating the birth canal, kid was delivered. Per vaginal re-examination did not reveal another foetus in the womb. Dead kid showed enormous fluctuating swelling of all four legs. Further, head has a disproportionate conformation just like a bulldog appearance with short stumpy neck (**Fig.1**). The dam was administered with injections Meloxicam (Melonex, Intas Pharma, Ahmedabad) IM @ 0.25 mg/b.wt., Ceftriaxone and Tazobactam 2250 mg IM, 2 ml Vitade IM, 5 ml Vit. B complex IV (Tribivet, Zydus AH), and 4 boluses of Furea were placed in uterus. Therapy continued for three more days resulted into uneventful recovery of doe with good milk production.



(Fig.1). Anasarcaus Bulldog Kid

Foetal anasarca has been observed mainly in calf, but occasionally in kids and foals (4). A fetus with anasarca may be prone to dystocia because the generalized edema will cause the fetus not to pass

through the pelvic canal (5). Therefore, surgical intervention is usually required for the delivery of oversized anasarca fetus (6); however, in the present case the successful vaginal delivery of an anasarca kid is depicted. Roberts (2004) (2) stated that foetal anasarca may develop in a single foetus or one of the twins and was due to simple autosomal recessive gene. Rarely mild hydrops of the amnion or allantois and oedema of the placenta may accompany foetal anasarca (7). Most anasarca fetus is expelled dead. The fluid effusion accumulation in subcutaneous space might be due to lack of lymph nodes and existence of autosomal recessive allele which affect the embryological development of normal lymph nodes (8, 9).

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## Surgical Management of Traumatic Proptosis in an Assam Hill Goat

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### Abstract

*This paper presents the successful surgical management of traumatic proptosis in an Assam Hill goat. A three-year-old goat was presented with a history of proptosis of an eyeball after an automobile accident. The proptosis was surgically managed by lateral canthotomy followed by temporary tarsorrhaphy. Post operatively, the goat was treated with parental antibiotics, NSAIDs and Vitamin A injections along with intra ocular antibiotic drops. The goat recovered uneventfully.*

### Introduction

Proptosis or prolapse of eyeball is an abnormal forward displacement of globe from its orbit which can occur secondarily to any blunt trauma to the head (1). The main etiology of proptosis is due to trauma and rarely due to other causes (2). The traumatic protrusion of eyeball in animals is usually due to blunt trauma either by automobile accidents or infighting (3). The swelling, eyelid spasms and orbital haemorrhage displace the globe further from orbit and prevent its retraction process (3). In animals, prognosis of proptosis varies greatly based on the degree of damage, period of external exposure and type of infection. As per the author's knowledge, very limited literature is available on

proptosis of eyeball in goats. In this context, this paper presents the successful surgical management of traumatic proptosis in an Assam Hill goat.

### History and Clinical findings

A three year old Assam Hill goat was presented with a history of automobile accident and prolapse of the right eyeball since two hours. On general inspection, the goat was found dull, depressed and in pain. Detailed clinical examination revealed protrusion of right eyeball associated with severe periorbital swelling without fracture of skull (Fig. 1). Other physiological parameters like body temperature, respiratory rate and heart rate were

found within the normal ranges. Based on the clinical findings, the case was diagnosed as "Traumatic proptosis". Hence, it was decided to replace the protruded eyeball by surgical management with temporary tarsorrhaphy to



**Fig. 1: Traumatic proptosis of eyeball in goat**



**Fig. 2: Corrected eyeball with temporary tarsorrhaphy**

reduce further complications.

**Treatment and Discussions**

The goat was restrained in left lateral recumbency and the swollen eyeball was cleaned with normal saline solution to remove debris and dirt. The affected eyeball was then lubricated with Neosporin ointment. Ice cubes were applied over the affected eye to decrease swelling and tried to insert inside the eye socket but failed to do so. Hence, surgical replacement was the only remedy to insert the prolapsed eyeball. The goat was then sedated with anaesthetic mixture of xylazine and ketamine hydrochloride@ 0.02 and 10 mg/kg b. wt. i.v. respectively. The affected right eye was then desensitized with 2% Lignocaine hydrochloride.

Standard lateral canthotomy was performed to replace the protruded eyeball which was followed by temporary tarsorrhaphy (Fig. 2) to retain the globe in its normal position. Post-operatively the goat was given antibiotics (Ceftriaxone @ 20 mg/kg b.wt. i.m.), non steroidal anti-inflammatory drug (Meloxicam @0.3 mg/kg b. wt. i.m.) and Vitamin A injections for consecutive seven days. The goat was also instilled topical antibiotic eye drops (Gentamicin) at every eight hours interval for 10 consecutive days. Regular dressing of the surgical wound was carried out on every alternate day and the sutures were removed after 12 days of post operation. Owner reported that the animal had an uneventful recovery without any loss.

Proptosis of eyeball is an acute ophthalmic emergency which requires immediate management. Depending on viability of extra ocular tissue and eye, the management options for proptosis of eyeball are evisceration, enucleation or replacement with tarsorrhaphy depending on viability of ocular tissues and eye (4). Common complications of traumatic proptosis encountered were corneal ulceration, hyphema, strabismus, chemosis, hyperemia, third eyelid prolapse and facial bone fractures (5).In the present study, goat was presented within two hours of accidental trauma with severe swelling of eyeball which was objecting the replacement of globe hence lateral canthotomy was performed to replace the prolapsed eyeball into its normal position followed by temporary tarsorrhaphy.

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Binjharpuri Cow

General Information			
<b>Species</b>	Cattle		
<b>Synonym</b>	Deshi		
<b>Breeding Tract</b>	<b>State</b> Odisha Odisha Odisha	<b>District</b> Jajpur Bhadrak Kendrapara	
<b>Location</b>	<b>Minimum</b>	<b>Maximum</b>	
	Deg Min	Deg Min	
<b>Longitude</b>	85 40	86 44	
<b>Latitude</b>	20 43	21	
<b>Comment on Breeding Tract</b>	Whole Jajpur district and adjoining areas of Kendrapara and Bhadrak. Heavy concentration is in Bari, Binjharpur and Dasrathapur area of Jajpur district		
<b>Main Use</b>	Food-Milk; Work- Draught; Manure		
<b>Comment on main use</b>	Dual purpose breed		
<b>Origin</b>	Indigenous breed. Named after the local area "Binjharpur" of Jajpur district in Orissa		
<b>Herd Book or Register Established</b>	No		
<b>Breed Societies(if Any)</b>	No		
<b>Adaptability to Environment</b>			
<b>Morphology</b>			
<b>Colour</b>	White. Some animals are Grey, Black or Brown in colour		
<b>Number of Horns</b>	2		
<b>Horns Shape and Size</b>	Curved upward and inward.Male: 21.17±2.86, Female: 12.70±1.31cm		
<b>Visible Characteristics</b>	Medium sized, strong dual type animal. Hump, neck, and some region of face and back are black in colour irrespective of coat colour in males		

	MALE	FEMALE	
Height(Avg cm)	124.4	107.3	
Body Length(Avg cm)	126.32	115.1	
Heart Girth(Avg cm)	144.2	136.2	
Weight(Avg Kg)	254.71	207.05	
Birth Weight(Avg Kg)	19.42	17.83	Overall 0
Management			
Management System	Extensive		
Mobility	Stationary		
Feeding of Adults	Grazing		
Comments on Management Conditions	Rice bran, water and paddy straw is provided to the animals in the morning before the animals are let loose for grazing. Working bullocks are fed with Kurchi- a mixture of rice bran, wheat bran, kitchen waste and some quantity of rice warmed with water		
<b>Performance</b>			
	<b>Average</b>	<b>Minimum</b>	<b>Maximum</b>
Age at first			
Parturition (Avg.Months)	40.48	0	0
Parturition Interval(Months)	13.47	0	0
Milk yield per lactation(kg)	0	915	1350
Milk Fat(%)	0	4.3	4.4
Peculiarity of the breed			
Population			
Year	<b>Population</b>	<b>Other Information</b>	
2007	67000	Project on survey, evaluation and characterization of Binjharpuri cattle, implemented by OLRDS, and OUAT, Orissa	
2013	79428	Source: Estimated Livestock Population Breed Wise Based on Breed Survey 2013. Department of Animal Husbandry, Dairying & Fisheries, Government of India, New Delhi	





## Rampur Greyhound

### Origin

The Rampur Greyhound is a breed of dog native to the Rampur region of Northern India, which lies between Delhi and Bareilly. The Rampur hound is a large member of the sighthound family. In North West of India it is often described as a smooth-haired sighthound that is substantially built. It was the favored hound of the Maharajahs for jackal control, but was also used to hunt lions, tigers, leopards, and panthers. It was considered a test of courage for a single hound to take down a golden jackal. The Rampur Greyhound is built to cover great distances at high speed but is also capable of great endurance.

### Appearance

The length from the withers to the base of the tail is about 36 inches, with a chest which is deep but not very wide across the shoulders, and with well-sprung ribs. The tail is long and tapering slightly curving upwards and carried low; it is about 24"-27" in length. The neck, about 12 inches in circumference, is long, arched, and muscular, and rather broad where it joins the body. The roughly 9-inch-long jaws are expected to exhibit a powerful scissor bite. The males measure 60–75 cm (24–30 in) in height. The females measure 55–60 cm (22–24 in) in height. They weigh about 27–30 kg (60–65 lb).

### Temperament

The breed loves human companionship, and like most sighthounds tends to keep itself clean and well-groomed. They may appear lazy but will charge if needed. They are affectionate to their owners. Generally gentle and sensitive around its own family's children. Before obtaining a Rampur hound, size and exercise requirements should be taken into consideration. Relatively robust, it needs plenty of space to stretch its legs and probably would be not be happy to be confined to a small apartment.

Rampur hounds are typically a healthy and long-lived breed, living up to fifteen years, and hereditary illness is rare. Greyhounds demonstrate unusual blood chemistry, which can be misread by veterinarians not familiar with

the breed; this can result in an incorrect diagnosis.

### History

His Royal Highness Ahmad Ali Khan of Rampur, Nawab of Rampur State, bred these dogs by combining the blood lines of very powerful but ferocious Tāzī, brought in by the Afghans, and the English Greyhound that was more obedient but less resistant to the varying climatic conditions. He gave the name 'Rampur Hound' to the dogs he bred. The Rampur Hound far exceeded his expectations. While it got its looks and stalwart character from its Tāzī Afghan ancestors, from the English Greyhound it got its speed. Here was a dog that would seldom back down in confrontations, and could more or less keep up with the fastest prey.

With the fall from power of the Maharajahs in 1947, so too fell the popularity of the Rampur Hound. The effect of the arrival of the English was evident to the Rampur, as well as the native Indian people. Additional English greyhound was bred into some of the lines, making it very difficult to find a purebred Rampur Greyhound today.

With the decline in hunting in India, the dog's popularity plummeted. It was no longer fashionable or practical for the rich to keep them, while the poorer population simply could not afford to keep one. In recent years, however, its popularity has begun to rise once more, and the breed's numbers along with this.

**Rampur Greyhound- Circa 1915. An old example of a Rampur Greyhound.**



Other names: North-Indian Greyhound / Rampur Hound | Origin: India | Domestic dog (*Canis lupus familiaris*)



## Diagnosis and Treatment of Cushing's syndrome in the Companion Dog: An Update

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### Introduction

Representing the clinical consequence of continued circulatory cortisol excess, Cushing's syndrome, described in the dog for the first time in 1939, is characterized by abnormally increased physical activity level (1). The typical clinical signs on presentation are alopecia, pendulous abdomen, polyuria, polydipsia, polyphagia, and muscular dystrophy in the hind limbs and/or the cranium (2). The classic appearance of the dog patient is truncal, symmetric, non-pruritic alopecia. Thinning of the skin may result in loss of tone. Diagnosis of Cushing's disease is seriously hampered by the marked case-to-case variation in the clinical signs. For example, the surface skin aberrations remain undetected by the owners in nearly 35% of the cases (3). On presentation of a dog patient to the clinic with the symptoms of Cushing's syndrome, it is necessary to establish the precise cause of the endocrine imbalance, hyperadrecorticism (HAC). Differentiating between the specific pituitary and adrenal teratogenic origin of the hormonal disorder is crucial for planning the effective rationalized treatment regimen. Nearly 80%

pituitary-dependent hypercorticism (PDH) cases are the outcome of adenoma of the pars distalis, and the remaining 20% are adrenal-dependent, the result of cortisol hypersecretion by gonadal adenoma/ carcinoma. Occasionally, Cushing's syndrome may also arise from continuous exposure to high levels of exogenous cortisol in long-term corticosteroids drug therapy (4).

Cushing's syndrome is a challenging, multi-faceted disease that requires an individualized, multi-disciplinary treatment approach. Nearly 80% of patients harbour a cortical pituitary adenoma, the actual source of the excessive secretion of Adreno corticotropin hormone (ACTH) → cortisol → hypercorticism (HAC). The treatment of choice is neurosurgical removal of the root cause, pituitary adenoma. Second-line treatments include medical therapy, bilateral adrenalectomy, and radiotherapy. In the dog patients, where surgery is not possible, drug treatment schedules target the malfunctioning hypothalamic/pituitary/ gonadal (HPG) axis. Bilateral adrenalectomy is the treatment of choice that ensures immediate control of the persistent endocrine disorder. However, this surgical option

requires a careful evaluation of the individual case in view of the apparent disadvantage of inducing permanent hormonal deficiency, which mandates life-long glucocorticoid and mineralocorticoid combination oral replacement therapy. Pre-surgery medical therapy in severe hypercorticism (HCA) cases is aimed to control the negative metabolic side effects, and minimize the time lag of radiotherapy bio-efficacy.

### Signalment

Dogs with HAC, related to primary adrenocortical tumors (PDH) are generally older (over 9 years of age) than those with pituitary related disease(4). Poodle, Dachshund, different Terrier breeds and Boxers appear to be genetically at increased risk. Nearly 60-65% of the dogs with adrenocortical tumors are female.

### Pathophysiology

In the adrenal cortical parenchyma, zona glomerulosa cells synthesize the mineralocorticoids. Of these, aldosterone, a steroid hormone promotes increased sodium retention and water reabsorption in the distal renal tubules concurrent with increased intestinal sodium absorption. Markedly reduced mineralo-corticoids biosynthesis / release following any structural or functional defect may lead to excessive loss of cell water with life-threatening tissue dehydration and electrolyte imbalance. Hyperkalemia: high circulatory levels of potassium (K+) concomitant with hyponatremia: low sodium (Na+) and hypochloremia: low chloride (Cl-) level, arising from accelerated urinary loss of salt (Na Cl) represent the pathobiochemical profile. *Zona fasciculata*, the middle and thickest zone of the adrenal cortex comprises columns of prominent secretory cells, arranged in 1-2 layers. The zona reticularis is comprised of polyhedral compact cells, arranged in freely anastomosing cords with relatively less lipid content and densely granular cytoplasm. The vital functions of glucocorticoids (mainly cortisol in dogs) include promotion of protein catabolism and stimulation of hepatic gluconeogenesis from the circulatory pool of L-amino acids.

The adrenal cortex is stimulated by the adrenocorticotrophic hormone (ACTH), synthesized in the adenohypophysis in the brain parenchyma. In turn, the activated cortical cells produce glucocorticoids, mineralocorticoids and small amounts of sex steroids, androgens and estrogens. The medulla comprises randomly distributed columnar or polyhedral secretory cells with a rich blood supply. These cells have large, vesicular nucleus, basophilic cytoplasm with fine chromaffin granules, representing the catecholamines: epinephrine and norepinephrine (5).

### Clinical profile

Dogs with Cushing's syndrome (HAC) exhibit clinical signs varying in magnitude from mild to severe. The classical symptoms of polydipsia and polyuria (PU/PD) are consistently observed in up to 90% cases. Polyphagia, attributed to glucocorticoid excess, causes abnormal weight gain and muscle weakness. Panting and alopecia may also be observed. Many of the affected dogs are prone to develop UTI from immune-suppressive effects of cortisol excess.

Polydipsia (daily water intake exceeding 100 ml/kg) and polyuria (urine production exceeding 50 ml/kg) represent the consistent pathoclinical feature in nearly all HAC dogs. Polyuria is apparently the sequel to the increased GFR, suppressed release of antidiuretic hormone (ADH), or impaired efficacy of ADH on the renal tubules axis. Dogs with macroadenomas (5%) may exhibit signs of diabetes insipidus following compression of the posterior lobe of pituitary and hypothalamus. Increased appetite (polyphagia) is a common observation, and many owners erroneously believe this as a sign of good health. Voracious appetite, scavenging or stealing food, however, is a matter of much concern to the pet physician, especially if the dog exhibited anorexia, previously. Client's improved pet care awareness is highly desirable.

The affected dogs occasionally develop 'Cushing's pseudomyotonia', characterized by persistent appendicular muscle contractions, usually more severe in the hind limbs, forcing the animal to walk with a stiff stilted gait. Ambulation may be difficult. Spinal reflexes are impaired because of the rigidity, but notably pain perception is not enhanced. High frequency bizarre discharges are recorded on electromyography (6).

The skin, especially over the ventral abdomen, becomes thin and atonic. Elasticity may be assessed clinically with the simple, yet highly informative 'skin tent' test. In healthy dogs, the skin fold reverts back to the smooth contour, but in the HAC dogs it remains tented with a striated appearance.

Anoestrus/ testicular atrophy represent the negative feedback effect of high cortisol titres on the HPG axis. A few HAC cases may subsequently develop neurological signs: dullness, depression, disorientation, loss of learned behaviour, anorexia, aimless wandering or pacing around the premises, head pressing, circling, ataxia, stumbling against objects, and occasionally seizures.

Systemic hypertension in more than 50% of dogs with untreated Cushing's syndrome is stated to be the outcome of excessive secretion of rennin/

activation of the rennin-angiotensin system/increased sensitivity of the blood vessels to the catecholamines with decreased vasodilator prostaglandins titre (7). However, the exact patho-clinical mechanism remains to be delineated. Associated impaired vision from intraocular hemorrhage/retinal detachment is on record (8).

### Pathobiochemical Profile

In dogs with Cushing's syndrome high blood cortisol titre generally induces the characteristic stress leukocyte profile: lymphopenia and eosinopenia with concurrent neutrophilia, and monocytosis. Mild erythrocytosis and thrombocytosis are also commonly observed. Markedly increased serum alkaline phosphatase (ALP) activity- a dependable indicator of the severity of disease, response to treatment and the prognosis- is observed in nearly 90% dogs. Significant proteinuria occurs in the majority of cases. Urinary tract infections (UTI) occur in about 50% of the cases. As such, urine culture should be part of the routine diagnostic panel (9).

### The Diagnostic Protocol

#### Screening tests

(i) Urinary cortisol-creatinine ratio (UCCR): The dilution-corrected values of urinary corticosteroids are recorded. Concurrent creatinine measurement is designed to adjust for the urinary dilution factor. Since excess circulating corticosteroids spill over into the urine before excretion, urinary cortisol concentration reflects the status of biosynthesis and release of the hormone from the adrenal cortex, and is used to rule out hyperadrenocorticism.

(ii) Low dose dexamethasone suppression test (LDDST): This test estimates and compares the titres of cortisol in the bloodstream of the patient before and after injection of the dexamethasone challenge dose that would inhibit cortisol production by the adrenal cortical cells in the normal dogs. Failure of cortisol suppression would signify the endocrine disorder, hyperadrenocorticism (HAC).

(iii) ACTH stimulation test: This test is used to differentiate between primary and secondary adrenal insufficiency by measuring the circulatory titres of cortisol before and after injection of the standard ACTH dose. Post-injection plasma cortisol concentrations below the normal range represent reduced bio-response to the tropic hormone challenge, indicating adrenocortical atrophy, associated with subnormal adrenal functional status (Addison's disease). Concentrations above the normal range indicate hyperadrenocorticism. The ACTH stimulation test has the highest specificity combined with sensitivity. It is the only test that can be performed on the companion dogs

with clinical signs of HAC on recent or on-going corticosteroid therapy. It is also used to monitor the patient's clinical response to treatment.

(iv) Dexamethasone suppression cum corticotrophin stimulation test: The combined protocol is aimed to screen the pathoclinical status of HAC with the ACTH stimulation test and demarcate the pituitary and adrenal gland origin with the suppression test, HDDST by recording the comparative cortisol titres, before and after the test dose of injected dexamethasone, followed by the subsequent ACTH challenge (after a gap of 4-6 hrs). In the patient that responds normally to ACTH stimulation but resists dexamethasone suppression, hyperadrenocorticism stands corroborated.

#### Differentiating tests

Further to corroborate the pituitary or adrenal origin of the endocrine disorder, differentiating tests are employed.

(i) High dose dexamethasone suppression test (HDDST): This highly dependable differentiating test in tandem with LDDST aims to determine the comparative circulatory cortisol titres before and after the injection of the standard test dose of dexamethasone. However, the high dose dexamethasone challenge will inhibit the production of cortisol only in the pituitary-dependent (not in adrenal-dependent) disease.

(ii) Corticotrophin (ACTH) assay: This procedure determines with precision the endogenous circulatory ACTH concentration, and is considered the most accurate biomedical tool for differentiating between pituitary-dependent and adrenal-dependent hyperadrenocorticism (HAC). It has the added advantage of a single blood sample requirement. In dogs with an adrenal tumor, ACTH titres remain undetectable or low-normal. In dogs with pituitary-dependent disease, however, these are high-normal to high (10).

#### Imaging Techniques

High definition imaging of the pituitary and adrenals is very useful in determining objectively the best treatment option, and evaluation of the prognosis. The pituitary can be visualized by computed tomography (CT), or nuclear magnetic resonance imaging (MRI). In healthy dogs, the pituitary gland measures 6 to 10 mm in length and 5 to 9 mm in height. The size of the pituitary can be evaluated on the CT image from the ratio of height and the area of the pituitary, calculated from the centre. A value exceeding 0.31 would indicate enlargement. Macro adenomas are detected on contrast-enhanced computed tomography (CECT) images. Enhanced image of the neurohypophysis is named 'pituitary

flush', and distortion or disappearance in the early phase of imaging, helps to pinpoint micro- and macro-adenomas.

The adrenal glands are examined for symmetry, size, geometry and echogenicity with CT. However, ultrasonography is often preferred being less expensive, faster, and not involving anesthesia. The structure of the adrenal gland is of more relevance than the size. In contrast-enhanced ultrasonography, following i.v. administration of the contrast agent, time-intensity curves are generated for the adrenal cortex, adrenal medulla and the ipsilateral renal artery of the adrenal glands. Dogs with the PDH disorder exhibit rapid and chaotic contrast enhancement in the adrenal: cortex and medulla. The occasional bilateral adrenal tumors reveal a heterogeneous echo profile, indicating necrosis and hemorrhages suggestive of neoplasia (3). Ultrasound-guided fine needle aspiration (FNA) biopsy is done. Thoracic radiography/ CT scan is used to identify pulmonary metastases.

### Therapy

The remedial strategy aims to eliminate the cause of hyperadrenocorticism (HAC) in the dog patient. New therapeutic options have emerged. Depending on the pathogenesis, medical treatment with the proven agent Trilostane® or Mitotane®, or surgical intervention (trans-sphenoidal hypophysectomy, adrenalectomy), or radiotherapy is selected.

### (I) Medical

A potent adrenocorticolytic agent, Mitotane® (Lysodren, o,p-DDD; Bristol-Myers Squibb) is the preferred remedial therapy in dog patients with HAC. The drug molecule is activated within the cortical tissue cells to a reactive acyl chloride intermediate, and covalently bound to the neoplastic cell protein, promoting progressive necrobiosis. For the PDH patients, the proven bi-phasic induction-cum-maintenance therapeutic strategy is aimed to balance the animal's minimum physiological needs, but block the high-risk persistent ACTH over-stimulation from the malfunctioning pituitary. The induction phase begins with daily administration of Mitotane® 50 mg/kg with the meals for 5-14 days leading to markedly reduced post-stimulation serum concentrations of cortisol (reference range 1 to 5 mg/dL). The dog patient is examined on day 8 or 9, and the ACTH stimulation test is conducted. The induction phase may be extended up to 7 days, depending on the clinical judgment. If post-ACTH stimulation cortisol levels still remain elevated, Mitotane is administered at the increased daily dose @ 50-75 mg/kg for up to 25 days for complete chemical ablation of the diseased adrenal cortex. Starting from day 3, glucocorticoids-

mineralocorticoids combination oral replacement therapy needs to be continued life-long.

A competitive inhibitor of 3 $\beta$ -hydroxyl steroid dehydrogenase enzyme, Trilostane® blocks the biosynthesis of both aldosterone and cortisol. Trilostane competes with pregnenolone as the substrate for the enzyme and results in decreased production of progesterone, the precursor all steroid hormones in vivo. Excessive ACTH production, however, continues in the PDH cases. Trilostane dose recommendations in the dog patient depend on the body weight: up to 5 kg @ 30 mg PO q24h or q48h, 5 to 20 kg @ 60 mg PO q24h, over 20 kg @ 120 mg PO q24h.

### (ii) Surgical intervention

**Hypophysectomy:** Located within the bony sella turcica, the pituitary is not easily accessible. With the use of contemporary imaging techniques: computed tomography (CT), or magnetic resonance imaging (MRI), the sphenoid bone is incised ventrally through the trans-oral or ventral cervical approach and complete hypophysectomy may be performed manually, or with ultrasonic aspirator. Life-long thyroid and glucocorticoid hormone supplementation is required. Desmopressin, a synthetic antidiuretic hormone is administered for two weeks, post-surgery.

**Adrenalectomy:** This is the preferred surgical treatment of adrenal tumors with excision through the ventral midline, or the pericostal approach. The latter has the advantage of minimal incision size and accidental traumatic injury to the adjoining abdominal organs. The ventral midline approach, however, permits more complete evaluation. Immediately before and after surgery, oral glucocorticoids-mineralocorticoids package must be administered. Removal of the adrenal tumor results in a transient compromised adrenal functional status, till the atrophied contra-lateral endocrine gland regains the ability to respond to ACTH. From the experience in human patients, supra-physiologic supplementation doses may be required to pre-empt the effects of acute steroid withdrawal, gradually tapered off in 2 to 3 weeks, post-surgery.

### (iii) Radiotherapy

Documented evidences support the use of irradiation technology, depending on the clinical judgment of the individual case. Radiotherapy reduced the tumor size and improved the neurological profile in the dogs with PDH with the median survival rate of 1 to 2 years (11, 12). In a study on 6 dogs with prominent (height 8 mm or more on MRI) pituitary tumors without neurological issues, fractionated cobalt 60 irradiation therapy

(total 11 fractions, each of 4 Gy over a period of 3.5 weeks, total 44 Gy) with pathoclinical monitoring before, immediately after, and at quarterly intervals up to 12 months produced dramatic reductions in the tumor size, though the clinical signs remained unabated in 50% cases (13).

**Conclusion**

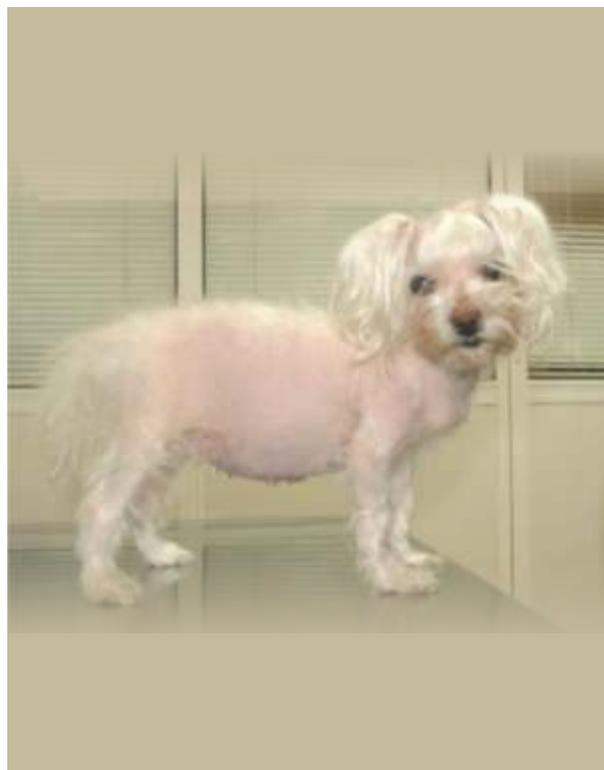
In the companion dogs, Cushing's syndrome: hyperadrenocorticism, HAC is pituitary-dependent or adrenal-dependent endocrinopathy, arising from persistent abnormally high circulatory cortisol titres. The clinical consequences of pituitary-dependent, hypercortisol-induced microadenoma can be managed with the adrenocorticostatic drug, Trilostane®, but the drug will not abolish the tumor. Hypophysectomy is, therefore, preferred in dogs with an enlarged pituitary but in a good condition with long life expectancy. Inoperable pituitary tumors can be treated by radiotherapy. In dogs with cortisol-secreting adrenocortical tumors, the best treatment option is adrenalectomy. If surgery is not possible Mitotane® is recommended.

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## Periodontal Disease in a Female Shih Tzu Dog: Diagnosis and Treatment

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### Abstract

*Princess Becah, female Shih Tzu dog was presented to the Milford Veterinary Clinic with a cyst under the left eye. Anamnesis revealed on-going periodontal disease process over the last six months. A tentative diagnosis of tooth root abscess was made, corroborated with the in-house diagnostic inputs: blood work and full mouth radiographs. The dental cleaning performed under closely monitored general anesthesia. The lower left 3rd incisor (303) was mobile, and the upper left carnassial (208) clearly revealed tooth root exposure with abscess formation and fistulation. The pet was suitably prepared with antibiotic pre-medication, and the two damaged teeth were carefully extracted with post-operative pain management and fluid therapy. The recovery was uneventful, and the patient was released to the client's custody the same evening.*

### Introduction

Periodontal disease is a compendium of plaque-associated inflammatory conditions affecting the tooth periodontium. Inflammation, originating as gingivitis, subsequently spreads to the entire attachment edifice: periodontal ligament, root cementing matrix, and the alveolar bony tissue culminating in loss of the tooth/ teeth. Possibly the most common in small animal practice, periodontal disease causes much discomfort to the affected

companion animal. The initial oral cavity pathogenic bacterial infection may spread and induce infection of distant internal organs (1).

In perspective, preventive dental hygiene is of paramount importance for the pet's general health and well-being. The perception that the prevalence is increasing may actually represent the increasing awareness of the preventable malady. The only way to prevent periodontal disease early is through home dental care gold standard: Systematicdaily

brushing, and the annual veterinary dental cleaning protocol, always under anesthesia. The

teeth are examined thoroughly, cleaned well, and the entire denture radiographed to identify any bone loss, periodontal pockets in the tooth root/surrounding structures, and nutritional support advised to the client (2).

**Case history**

Princess Becah, 8-year-old female Shih Tzu, was presented to the Milford Veterinary Clinic on April

16, 2019 with an open cyst under the left eye. Anamnesis revealed that the lesion started about six months earlier, slowly enlarged, and burst open recently with pus draining out. The pet on raw organic diet is now eating more, occasionally vomiting out bile-stained food. The patient's vitals were all within the respective normal limits. The upper left carnassial tooth 208 (3) exhibited root exposure with marked accumulation of tartar, gingivitis and clear signs of infection. The tentative diagnosis: tooth root abscess.

**In-house diagnostics**

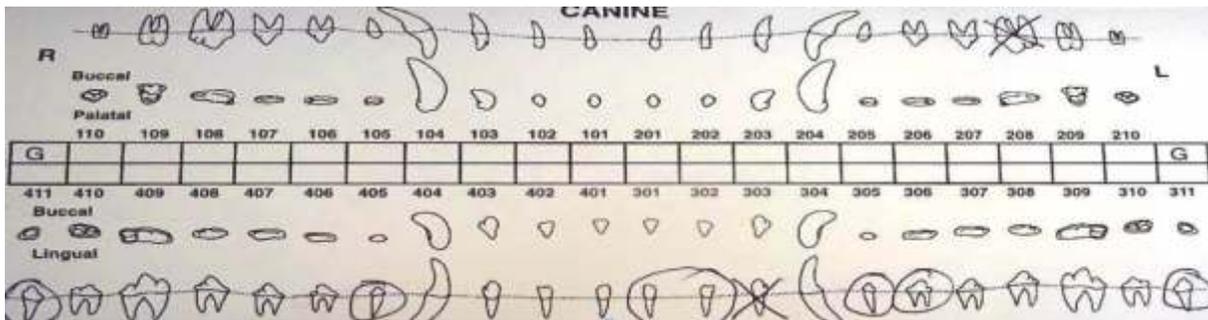


Fig. 1. Dog's denture: Standardized identification numbers.

**Triadan Tooth Numbers  
Canine and Feline Dentition**

	Permanent		Deciduous	
	RIGHT	LEFT	RIGHT	LEFT
Maxillary	100+	200+	500+	600+
Mandibular	400+	300+	800+	700+

In the tables below, for permanent teeth, I = incisor, C = canine, P = Premolar, and M = molar. For deciduous teeth, i = incisor, c = canine and p = premolar.  
\* = tooth not normally present.

RIGHT Permanent Teeth - Dog - Maxillary Teeth											LEFT Permanent Teeth - Dog - Maxillary Teeth										
M2	M1	P4	P3	P2	P1	C	I3	I2	I1		I1	I2	I3	C	P1	P2	P3	P4	M1	M2	
110	109	108	107	106	105	104	103	102	101		201	202	203	204	205	206	207	208	209	210	
411	410	409	408	407	406	405	404	403	402	401	301	302	303	304	305	306	307	308	309	310	311
RIGHT Permanent Teeth - Dog - Mandibular Teeth											LEFT Permanent Teeth - Dog - Mandibular Teeth										
M3	M2	M1	P4	P3	P2	P1	C	I3	I2	I1	I1	I2	I3	C	P1	P2	P3	P4	M1	M2	M3

RIGHT Deciduous Teeth - Dog - Maxillary Teeth					LEFT Deciduous Teeth - Dog - Maxillary Teeth											
p4	p3	p2	*	c	i3	i2	i1		i1	i2	i3	c	*	p2	p3	p4
508	507	506	*	504	503	502	501		601	602	603	604	*	606	607	608
RIGHT Deciduous Teeth - Dog - Mandibular Teeth					LEFT Deciduous Teeth - Dog - Mandibular Teeth											
p4	p3	p2	*	c	i3	i2	i1		i1	i2	i3	c	*	p2	p3	p4
808	807	806	*	804	803	802	801		701	702	703	704	*	706	707	708

RIGHT Permanent Teeth - Cat - Maxillary Teeth					LEFT Permanent Teeth - Cat - Maxillary Teeth													
M1	P4	P3	P2	*	C	I3	I2	I1		I1	I2	I3	C	*	P2	P3	P4	M1
109	108	107	106	*	104	103	102	101		201	202	203	204	*	206	207	208	209
RIGHT Permanent Teeth - Cat - Mandibular Teeth					LEFT Permanent Teeth - Cat - Mandibular Teeth													
M1	P4	P3	*	*	C	I3	I2	I1		I1	I2	I3	C	*	*	P3	P4	M1
409	408	407	*	*	404	403	402	401		301	302	303	304	*	*	307	308	309

RIGHT Deciduous Teeth - Cat - Maxillary Teeth					LEFT Deciduous Teeth - Cat - Maxillary Teeth											
p4	p3	p2	*	c	i3	i2	i1		i1	i2	i3	c	*	p2	p3	p4
508	507	506	*	504	503	502	501		601	602	603	604	*	606	607	608
RIGHT Deciduous Teeth - Cat - Mandibular Teeth					LEFT Deciduous Teeth - Cat - Mandibular Teeth											
p4	p3	p2	*	c	i3	i2	i1		i1	i2	i3	c	*	p2	p3	p4
808	807	*	*	804	803	802	801		701	702	703	704	*	*	707	708

Tables provided by AVDC based on Floyd, M: The Modified Triadan System in *J Vet Dent*, 8, 4, 19-20, 1991

**Treatment**

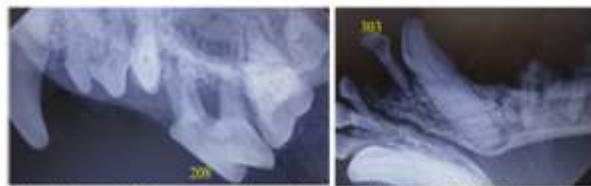
Princess Becah, initiated to broad-spectrum oral antibiotic regimen (Clavamox® 62.5 mg one tablet BID, continuously for 14 days) by the well-briefed owner starting the day before the scheduled dental

care oral surgery, was presented to the clinic on 17th April, 2019. The patient was pre-medicated with Torbugesic® 0.05 ml and Acepromazine® 0.02 ml, subcutaneously. IV catheter was placed and infusion of lactated Ringer's solution was started @ 20ml/hr. Anesthesia, induced with Ketamine 0.25 ml



**Fig. 2. Post-operative view of the patient's oral cavity.**

and Midazolam 0.25 ml IV, was maintained with isoflurane gas. Accumulation of tartar of moderate density mainly over the molar arcades peaking to virtually 4/4 tartar scale in the left upper carnassial, 208 was clinically significant. The tartar mass was gently chipped off and then scaled with ultrasonic probe. After dental cleaning the teeth were polished. Full mouth radiographs (Fig. 3) revealed signs of early osteolysis, especially in the 300 series lower left incisors.



**Fig. 3. Dog's dental radiographs of the affected teeth**

The infected partially detached mobile 3rd incisor 303, and carnassial 208 (Fig. 3) clearly revealed tooth root exposure with the abscess and fistula opening below the left eye. As per the standard protocol (4), after carefully lifting the gingiva and

dental ligaments upwards, the totally damaged carnassial 208 was sectioned with a high-speed drill. The dental forceps, rongeurs was used to extract the tooth, split in two halves in quick succession. The gingival ulcer was closed with 4-0 absorbable sutures. Then the lower left incisor, 303 was elevated and extracted. The surgical wound in the gum was closed. The abscess area was flushed with sterile saline solution. The opening near the left eye was clipped and cleaned with chlorhexidine solution. Topical antibiotic ointment was gently applied.

The patient was initiated into oral Clavamox® antibiotic regimen. Pre-surgery pain medication, Carprofen® was given @ 4.4 mg/kg, subcutaneously. Oral Carprofen @ 2.2mg/kg BID was scheduled for 4 days post-surgery. Fluids infusion was continued for a few hours. The recovery was uneventful, and the patient was released to the owner's care the same evening.

**Discussion**

Periodontal disease in the dog patient is caused by pyogenic bacterial infection that spreads underneath the gum line, undetected. Veterinary dental care is recommended if the dog exhibits any of the cardinal signs: swollen, bleeding or painful gums, episodes of acute depression, pawing at the facial or mouth area frequently, bad breath, change (howsoever small) in the chewing and/ or eating habits, missing or misaligned teeth, discolored broken or crooked teeth, excessive drool, growths or bumps in the mouth cavity or face, and brownish yellow tartar crust deposited within or along the gum line. An anesthesia-free dental cleaning provides no tangible relief to the pet's oral health. Scaling with a dental scalpel only makes the teeth appear deceptively white. However, actually scaling without proper polishing promotes firm adhesion of the surface-primed bacterial plaque. Thus, anesthesia-free dental cleanings are most damaging to the pet's oral/ general health: imparting the false sense of security, leaving the periodontal disease undetected and untreated. The primary cause of both gingivitis and periodontitis is the rapid accumulation of the highly deleterious dental plaque on the tooth surface, both supra-gingival

(i.e. on the crown) and sub-gingival (i.e. below the gingival margin). Dental plaque is composed of aggregates of bacteria and their degradation products, salivary components, oral cell debris, and occasional exfoliated epithelial cells and inflammatory cells (5).

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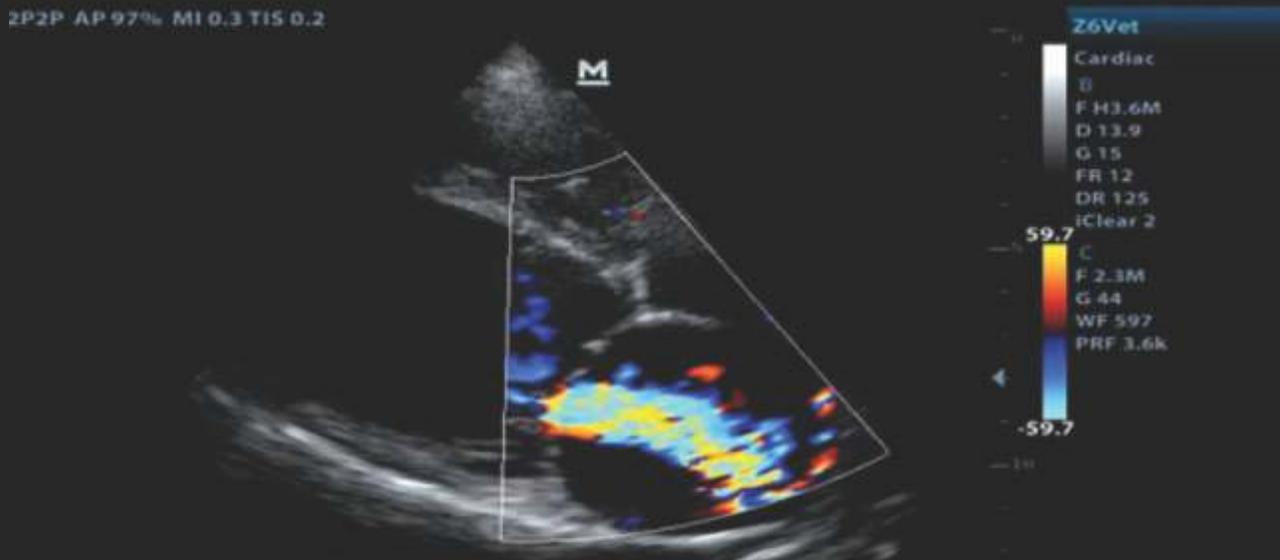


Fig. 1.

## Dilated Cardiomyopathy in Dogs

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Dilated cardiomyopathy (DCM) is the most common cause for primary myocardial failure in dogs. The condition is characterized by myocardial dysfunction - predominantly systolic -with progressive dilation of heart chambers (1). This is characterized by weakness of heart muscle leading to poor myocardial contractility causing ineffective pumping of blood out of heart. There can be dilation of all four chambers or mostly left cardiac chambers. Large and giant breeds are most commonly affected including Doberman pinscher, St Bernard, Boxers and Dalmatians with highest prevalence among Doberman pinschers (2). Dilated cardiomyopathy is an idiopathic syndrome with genetic predisposition. Middle aged to older aged dogs are more at risk.

### Etiology

Etiology can be multifactorial but it mainly occurs as an idiopathic form.

Most common causes include (1);

- 1) Nutritional(L-carnitine and Taurine deficiency)
- 2) Familial
- 3) Infectious-Parvovirus in pups, Trypanosoma cruzi
- 4) Immunological-autoantibodies to components

of myocardium

- 5) Toxins-Doxorubicin
- 6) Biochemical anomalies-anomalies of mitochondrial activity and calcium influx (1,3)
- 7) Genetic- Autosomal dominant trait(Doberman (4),Great Dane, Boxer(mutation in strain gene), Golden Retriever(mutation in dystrophin gene),Portuguese water dogs-autosomal recessive trait (2)

### Pathophysiology

The heart muscle becomes flabby and weak leading to loss of ability to contract causing reduction in cardiac output. It can be systolic or diastolic dysfunction, primarily systolic leading to dilation of cardiac chambers. The clinical signs occur when pressure in dilated chambers increases causing extravasation of fluid causing pulmonary edema or ascites. Secondary valvular insufficiency occurs due to chronic stretching of valves.

### Clinical Manifestations

There are two phases of DCM, preclinical or occult phase and overt or clinical phase (5). In occult phase, animal is asymptomatic and will be of one to four year duration. Clinical signs of DCM are due to impairment in delivery of oxygenated blood to

Fig. 1. Enlarged left atrium and left ventricle associated with dilated cardiomyopathy. The Doppler mosaic jet is suggestive of regurgitation associated with inability of valve leaflets to meet.

tissues leading to lethargy, weakness, weight loss and collapse or due to congestion of lungs causing respiratory distress, coughing, exercise intolerance and abdominal distension (6). Upon clinical examination, there will be pulmonary crackles, jugular venous distension or pulsation, hepatosplenomegaly, cardiac murmur in valvular insufficiency, muffled heart sounds in pericardial effusion, S3 or gallop sound, weak femoral or arterial pulse, pale mucus membrane, increase in capillary refill time and arrhythmia.

### Diagnosis

Dilated cardiomyopathy is best diagnosed by echocardiography. Findings are chamber dilation mostly left atrium and ventricular enlargement, myocardial hypokinesia and mitral regurgitation (**Doppler-fig1**). Electrocardiography is used to diagnose arrhythmias like atrial fibrillation, ventricular arrhythmia, ventricular premature complexes, and tachycardia (5). Holter monitoring is useful for assessment of cardiac arrhythmias accurately (1, 5). Thoracic radiography facilitates assessment for cardiomegaly, pulmonary edema and pleural effusion (6). In certain cases prerenal azotemia due to poor renal perfusion or low cardiac output leads to high Creatinine and BUN value. Liver enzyme levels are elevated due to prehepatic congestion. Severe congestive heart failure may be associated with hypoproteinemia, hyponatremia and hyperkalemia.

### Treatment

Treatment of DCM aims at improving cardiac contractility and dilating peripheral blood vessels.

Dobutamine is a direct acting catecholamine with beta-1-adrenergic agonistic activity which improves cardiac output with minimal effects on heart rate. Dobutamine at a dose rate of 2.5-10 micrograms/kg/min diluted to 25 micrograms/ml in 5% dextrose or normal saline is given as constant rate infusion. Continuous electrocardiogram monitoring throughout drug administration is required since sinus tachycardia or ventricular dysarrhythmia may develop during drug infusion.

Digoxin is a positive inotropic agent with negative chronotropic properties used for management of DCM and supraventricular tachyarrhythmias. Treatment is also directed to reduce fluid accumulation by furosemide administration at a dose rate of 4-8 mg/Kgiv every 30 minutes until patient urinates (7). Enalapril, an ACE inhibitor at dose rate of 0.5 mg/kg to reduce sodium and fluid retention and reduce after load. Supplemental oxygen therapy at 50-100 ml/kg/min to supply 40% to 50% oxygen is indicated in severe acute DCM cases. Other vasodilators like nitroprusside, topical application of nitroglycerine paste at an 8 hour

interval is also indicated.

Pimobendan an inodilator, is being used with encouraging results in the management of congestive failure along with other drugs. Use of furosemide or torsemide is also suggested. Specific arrhythmias, as the case may be, will have to be treated (1).

### Prognosis

The prognosis of the disease is usually poor to be guarded. Animals in occult phase may survive for a period ranging from six months to two years. Some may respond to treatment and survive for months or years. There is no reliable way to predict the progression and response of animals to treatment.

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## Surgical Management of Cutaneous Squamous Cell Carcinoma in Scrotum of a Dog

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### Abstract

*An eight year old non-descript male dog was brought with the history of having a mass in the left scrotum for the past two months. Clinically a firm, ulcerated spherical tumour was seen in the left scrotum. Under general anaesthesia, the tumor was surgically removed and histopathologically the tumour was confirmed as squamous cell carcinoma.*

*The skin of the scrotum is thinner than other skin, typically pigmented, and may contain fine hairs on the surface (1). Mast cell tumors, melanocytoma, malignant melanoma, vascular hamartoma, hemangiosarcoma, hemangioma, and cutaneous histiocytoma were the most common tumor types identified on the canine scrotum. Only 4.2% of scrotal neoplasms were of epithelial origin that was squamous cell carcinoma. Low incidence of this neoplasm on skin that is exposed to ultraviolet light radiation may be due to the amount of melanin pigment that is present within the epidermis of the scrotum (2). The present report records a case of cutaneous squamous cell carcinoma in scrotum of a dog and its successful surgical management.*

### Case History and Observations

An eight year old, intact male non-descript dog was presented with the history of having a mass in the scrotum for the past two months which gradually increased in size. Clinical examination revealed a spherical, firm, ulcerated tumour in the left scrotum without testicular involvement (Fig.1). There was no palpable peripheral lymph node enlargement. Thoracic radiographs showed no evidence of pulmonary metastasis. All the biochemical and haematological parameters were within the normal range. Based on the history, clinical examination and radiographic observations, the case was suspected for scrotal tumor and orchiectomy with scrotal ablation was decided upon and the animal was prepared for aseptic surgery.

### Treatment and discussion

The dog was premedicated with atropine sulphate @ 0.04 mg/kg body weight s/c and xylazine hydrochloride @ 1 mg/kg body weight i/m. General anaesthesia was induced with Ketamine Hcl and Diazepam @ 5mg and 0.5 mg/kg body weight i/v. An elliptical incision was made at the base of the scrotum and subcutaneous haemorrhages were controlled by

ligating the blood vessels. Open orchiectomy with scrotal ablation was performed by following standard operating procedure. The subcutaneous tissue was opposed with simple continuous pattern using PGA 2-0 and the skin was sutured with simple interrupted suture pattern using silk 2-0. Post operatively the animal was given Inj. Intacef @ 25 mg/kg b.wt for 5 days and Inj. Melonex @ 0.2 mg/kg b.wt. for 2 days and the wound was dressed with povidone iodine solution daily. The skin sutures were removed on eighth post operative day and the animal recovered uneventfully. There was no recurrence of tumor till one year of post surgery.

On gross examination, the scrotal tumor was round, firm, red and had the dimensions of 2.5 x 2.0 x 2.5 cm without testicular involvement (Fig.2). The tissue sample was preserved in 10 per cent formalin for histopathological examination. Paraffin embedded tissue sections were cut into 5 $\mu$  thickness and staining with Haematoxylin and Eosin (H&E). Histologically, variable sized irregular masses of concentrically arranged proliferating squamous cells invaded into the dermis with keratin pearls formation. Mitotic figures were also seen. Intracellular bridges were also observed. Based on the microscopical features, the



Fig.1: Dog-Scrotal skin - Cutaneous squamous cell carcinoma squamous cell carcinoma of a scrotum in a dog.

mass was suggestive of well differentiated squamous cell carcinoma.

Squamous cell carcinomas are malignant tumors arising from the squamous epithelium of skin and mucous membranes. The incidence of the tumors arising from the skin is higher than tumors from any



Fig.2: Gross appearance of cutaneous squamous cell carcinoma.

other location in canines which represent 5% of skin tumors in dogs (3, 4). They are usually found in those areas within the epidermis where there is a dearth of pigmentation, hair or a very sparse hair coat. The incidence is low in the scrotum due to the amount of melanin pigment that is present within the epidermis of the scrotum (2). The peak incidence of squamous cell carcinoma in the dog is between 6 and 10 years of age. In the present case the age of the dog was 8 years and the tumour was seen in the scrotal skin invaded into the dermis.

The etiology of canine squamous cell carcinoma is poorly understood, although environmental factors, including prolonged exposure of lightly pigmented skin to ultraviolet radiation have been associated with the cutaneous form (5). It is believed that there may be some association with papilloma virus.

Squamous cell carcinomas generally grow slowly, but are aggressive in nature. They, however, do not metastasize to the regional lymph nodes in the early stage. Regardless of tumor location, squamous cell carcinomas can be classified into different grades of differentiation: well differentiated, moderately differentiated and poorly differentiated. Differences in clinical and histopathological features may be observed among them. The well differentiated subtype is the most commonly observed in canine species, which is characterized by masses or cords of neoplastic epithelial cells that proliferate and invade

the dermis and subcutis. The hallmark of this subtype is the presence of keratin pearls in varying numbers and size, which are composed by concentric layers of squamous cells with gradual increase of keratinization toward the center (3). In the present case, well differentiated squamous cell carcinoma was observed and revealed the presence of multiple keratin pearls formation with variable sized cords of neoplastic squamous cells.

Most cutaneous squamous cell carcinomas appear as firm, raised, frequently ulcerated plaques and nodules; sometimes they can be extremely exophytic and have a surface that resembles a wart. Prognosis of squamous cell carcinoma is dependent on the location of the tumor (6). In late stages, the prognosis is always grave as squamous cell carcinoma is known to metastasize to different vital organs mainly lymph nodes, lung, liver etc (7). Thus early diagnosis is very important in cases of malignant tumors to prevent further complications generated by metastasis. In the present case early diagnosis and timely orchietomy with total scrotal ablation could save the animal.

### Summary

A case of cutaneous squamous cell carcinoma in a scrotum of 8 year old dog and its surgical treatment is reported.

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## Surgical Management of Inguinal Nongravid Hysterocoele in a Bitch

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### Abstract

*Inguinal hernias occur less frequently than umbilical hernias and result from a defect in the inguinal ring through which abdominal contents protrude and mostly occur in middle aged intact bitches. A 2 ½ year old non-descript bitch was brought with a history of having a mass in the left ventral abdomen since three months. Based on clinical and radiographic examination the case was tentatively diagnosed as left inguinal nongravid hysterocoele. Surgically the contents were reduced and herniorrhaphy was performed and the animal made an uneventful recovery.*

### Introduction

*Inguinal hernia* are frequently documented in female dogs and are most often diagnosed in intact, middle aged bitches (1, 2) and are mostly due to trauma that weakens the abdominal musculature resulting in abnormality of the inguinal ring. The common contents in the hernial sac include fat, uterus, omentum, bladder and ovary (3, 4). *Inguinal hysterocoele*, where there is herniation of uterus through the inguinal canal comes under the category of caudal ventral abdominal hernias (4). Early presentation of the case, proper and timely diagnosis and treatment are necessary for a favourable outcome in the surgical management of *inguinal hysterocoele*. A case of an inguinal nongravid hysterocoele in a 2 ½ year old nondescript bitch and its successful surgical management is placed on record.

### Case History and Observations

A 2 ½ year old nondescript bitch was presented with the complaint of a progressively growing mass in the left ventral abdomen since three months. No information about her cyclic phase was available and it had whelped only once a year back. On clinical examination, lameness related to inguinal swelling was noted. There was a swelling in the left

caudal ventral abdomen at the inguinal region and there was a hernial ring with reducible hernial contents. The swelling was painless with a soft, doughy consistency and measured 10 cm by 6 cm in length and width respectively (**Fig.1**). All the physiological parameters appeared to be normal. Lateral radiograph of the mass revealed presence of soft structures and the animal was not pregnant. Based on the history, clinical and radiographic examinations, the case was diagnosed as inguinal nongravid hysterocoele and surgical correction was decided upon and the animal was prepared for aseptic surgery.

### Treatment and Discussion

The bitch was premedicated with atropine sulphate @ 0.04 mg/kg body weight s/c and xylazine hydrochloride @ 1 mg/kg body weight i/m. General anaesthesia was induced with Ketamine Hcl and Diazepam@5mg and 0.5mg/kg body weight i/v respectively. The animal was positioned on dorsal recumbency and an incision of about 4 cm long was made on the swelling to expose the hernial contents. Inguinal sac was exposed through blunt dissection and the content was reduced. The sac was opened and the canal was enlarged by incising through the inguinal ring in a craniomedial direction.

It was observed that the omentum along with the nonpregnant uterus were the hernial contents (**Fig. 2**). The hernial contents were gently reduced back to the abdominal cavity and the hernial ring was closed with Polyglactin 910 in simple continuous suture pattern. Excessive hernial sac was excised and margins apposed using number 2/0 chromic catgut in a simple interrupted pattern. Subcutaneous tissue was apposed with 2-0 Polyglactin 910 in subcuticular suture pattern. Herniorrhaphy was performed by overlapping suture pattern with non absorbable no.1 polyamide. The skin was apposed in simple interrupted sutures using silk (**Fig.3**). Post operatively the animal was given Inj. Intacef @ 25 mg/kg b.wt for 5 days and Inj. Melonex @0.2 mg/kg b.wt. for 2 days and the wound was dressed with povidone iodine solution on alternate days. The skin sutures were removed on eighth post operative day and the animal recovered uneventfully. There was no recurrence over the next six months post surgery.

*Inguinal hysterocele* can be acquired due to traumatic or non-traumatic causes, and toy breeds of dogs and Dachshund are predisposed to this condition. Anatomic causes like shorter and large diameter of the *canalis inguinalis*, nutritional causes, high oestrogen levels (oestrus and/or pregnancy), increased abdominal pressure due to obesity or pregnancy predisposes this condition (5). In the present case, the patient was an intact bitch and it could have been possible that one of the predisposing factors of development of the hernia might be estrogen. This reasoning is further supported by the fact that the patient was not obese, and there was no history of trauma that weakens the abdominal wall therefore eliminating the possibilities of trauma, nutritional or metabolic factors playing a role in development of the hernia. Contents of inguinal hernias include omentum, fat, ovary, uterus, small intestine, colon, bladder and spleen. In the present case the hernial contents were omentum and nonpregnant uterus.

Conventional hernial repair through the inguinal ring (4) or a ventral midline incision parallel to the flank folds lateral to the hernial ring are feasible (6).

Spaying can prevent the occurrence of *inguinal hysterocele* in bitches (7). If further breeding is not intended, an ovariohysterectomy may be performed but in the present case the owner did not agree for panhysterectomy. Incisional dehiscence, recurrence of hernia and incarceration of the uterus were reported as complications in the surgical correction of inguinal hernias (1, 2). Even though the occurrence of *inguinal hysterocele* is rare, timely diagnosis and treatment could prevent untoward complications as also observed in the present case.

### Summary

Inguinal nonpregnant hysterocele in a 2 ½ year old nondescript bitch and its successful surgical management is reported.

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**Fig.1: Clinical appearance of inguinal hernia in a bitch**



**Fig.2: Hernial contents: Nongravid uterus and omentum**



**Fig. 3: Post surgical correction of inguinal nongravid hysterocele**



## Health Care and Training for a New Puppy Dog Owner

Indian Immunologicals Limited

### Introduction

Pets especially dogs make the best of companion animals. It is advisable to acquire a pup and train it to your surroundings and family members, than taking on adult which might have adjusted to some other surroundings. When you acquire a pup and bring it first time to your house, please do not rush to it and show your eagerness and appreciation of how cute it is or grab it by the forelegs, etc. These actions may scare the pup. The best way to handle a pup is to cradle it by supporting its chest and hind legs. Allow the pup to settle down quietly and let it snoop around to acquaint itself to the new surroundings.

### Bedding

A box with soft clean bedding can make a good bed for a pup. Prepare the bed before you get the pup home. If the pup you acquired has a light coat then it is suitable for indoors. The bedding should be in a clean, warm, dry place. Don't keep the bedding near windows, air conditioners etc., where it is exposed to extremes of temperature. Artificial heat also harms its skin. Hence, bedding should be away from stoves, heat etc. The bedding should be cleaned at regular intervals. If the pup has got

heavy coat, it is suitable for outdoors and would like to sleep outdoors. It is advisable to build a small dog house to protect the pups from sun, wind and fog.

### Bathing

It is a myth that dogs need bath everyday / week. There is no need for giving a bath to the pup every week. In fact the first bath should be given when the pup is several months old. If you give a bath at three to four weeks interval, it is sufficient to keep the pup clean. During winter, do not allow the pup outdoors for at least 6 hours after the bath.

### Grooming

Brushing and combing stimulates circulation to the skin. It tones up the skin and gives a healthy look for the coat. The pup should be frequently groomed to develop a shiny and glossy coat. Regular grooming keeps the dirt and loose hair away. The nails should be trimmed periodically.

### Toys

Artificial rubber balls etc., will keep the pups busy and playful. They will also provide exercise for teeth and gums. The toys will also divert the attention from the domestic / household products and prevent destruction of them. Make sure that the toys

are large enough so that they will not be swallowed by the pup.

**Deworming**

The pups are more prone to infestation with intestinal parasites. If there are only a few worms, we may not feel their presence. However, if the

infestation is too heavy, you can observe the pup losing weight and all the visible mucous membranes become pale. It is advisable to diagnose the parasitic infestation before deworming is attempted. Regular deworming and health checkup at least three to four times in a year will keep the pup healthy.

For any other health problem, please consult your friendly neighborhood veterinarian.

<b>A few things to remember</b>
A light tap on the nose with a finger and a firm voice is enough punishment for a pup. Do not strike it with a hand or newspaper or stick.
Be patient with your pet
Do give plenty of exercise
Once you observe the pet dull or ill, take it to a veterinarian immediately
Do not feed bones which split up easily
Don't pick the pup by its fore limbs
Avoid feeding chocolates and other sweets unless they are specifically manufactured for dog use
Do not allow the pup to play in dirt and streets
Discourage the pup from sleeping on the furniture
Too frequent bathing is not required
Do not allow it to trouble your guests
Keep the water dish always filled with fresh water
Complete all the formalities of licensing if applicable from the local authorities
Vaccinate the pets using tried and tested vaccines like Raksharab and Megavac.

**Nutrition**

One of the most important consideration with a pup is to give the correct amount of the right food at set time. A week or so before taking your pup home have a word with the breeder from whom you are buying the pet. Know about its correct diet like how many meals a day it takes and so on. It is probable that the pup will be somewhere around ten to twelve weeks old, and already weaned from its mother milk to solid / semi solid foods. Do not overload its small stomach with food. What a puppy needs for bone and flesh development is a diet rich in protein, such as fish, eggs, milk and meat. Your pup needs regular meals. The amount at each meal should be no more than what the pup will hungrily eat up. Spread the meals over 15 to 16 hours at about four hours interval.

**Breakfast**

A cereal and fine biscuits mixed in warm milk with beaten eggs. Also mix a dessert spoon of rusks

containing vitamins & essential elements.

**Lunch**

Lightly cooked minced meat / liver, fish moistened with gravy. Avoid using too much spices in the gravy.

**Afternoon:** A diet of warm milk.

**Evening:** Repeat the afternoon meal

**Night:** Repeat the breakfast meal.

If the meat is from good source, it can be fed raw. If not, cook the meat before feeding your pup. During this time, it is essential that the pup should have a bowl of fresh drinking water. Indeed this must continue throughout its life. By the time it reaches fifteen weeks it will probably start to refuse milk especially at bed time. But do ensure it continues with its bed time biscuits. At four months the number of feedings can be reduced to four and at six months to two feeds a day. It is at this stage that it can go on to its adult diet.

**Daily Food Requirements**

Body Weight	Calories/Day	Canned	Semi-Moist	Dry
5 lb (2.3 kg)	250	1/3 - 1/2 can	3/4 - 1 cup	1/4 - 3/4 cup
10 lb(4.5 kg)	420	2/3 - 1 can	1 1/2 cups	3/4- 1 1/4 cups
20 lb (9.1 kg)	700	1 1/2 - 3/4 cans	1 1/5 cups	1 1/4 - 2 1/4 cups
40 lb (18.2 kg)	1200	2 - 3 cans	4 1/4 cups	2 1/2 - 3 1/2 cups
80 lb (36.4 kg)	2000	3-5 cans	7 1/2 cups	4 - 6 cups
100 lb(45.5 kg)	2400	4 - 6 cans	8 - 9 cups	5 - 8 cups
180 lb(81.8 kg)	3500	6 - 9 cans	12 - 13 cups	7 - 11 cups

**Training**

When you train the pup, the first thing to remember is to have a consistent temperament. The animals do not like unpredictable and inconsistent temperaments. The training should be gradual and gentle. Avoid yelling, grabbing, kicking during training. Do not start the training immediately after a full meal. A pup should be well nourished and healthy to learn new tricks. Too ambitious training programme will not be successful. The dogs can be trained to conduct, to herd sheep and cattle, to lead the blind, etc. They can be trained to become watch dogs, hunting partners and sniffer dogs to solve the crimes or to detect explosives, drugs, etc. Always remember that the dogs habits, good or bad, are acquired through training from the master. If you earn the friendship and trust of a pup in its early life, you can train it whatever way you want without any problem. But it is very important to earn the trust of your pet. The training can start with a few simple commands like to walk on the leash without pulling, to sit or to come to you when called.

Training a pup can be a good experience for the owner as well as the pup. A proper time preferably before feeding is ideal for training. Open air or indoors are equally ideal for training depending on the weather. Walk a few paces from the pup and call it by name. The hearing of a pup is very sharp, so make the tone appealing. Once the pup comes to you, do it again by moving away from it. When the pup comes to you promptly, a pat or a hug is a reward enough for it to learn faster. It can also be treated with a small snack as a reward for obeying the command.

Making the pet to walk on leash may be slightly difficult job as the pup would like to run and pull you along when it comes outdoors. Always keep the pup on your left side. Be firm when it is trying to pull you. Make it understand that a steady pace is what is

expected of it. Teaching simple obedience lessons

**Housebreaking your dog**

This is the first basic obedience lesson for every dog. The dogs can learn it easily just when they are old enough to walk. The dogs will take it easily as they are naturally fastidious animals. Your dog is as interested as you are regarding cleanliness. Even the puppy will not mess up its bedding. The first rule in housebreak-ing your dog is to familiarize it with the house and the boundaries. Keep it on a regular feeding and exercise schedule. Follow the three steps:

- a) Tie the puppy near its bed for the first few days. Dogs don't like wet bedding, so look at it as often as possible.
- b) When the puppy goes out and relieves itself, appreciate it and pat it. It will make it understand that it did a right thing.
- c) Gradually reduce the number of outside trips. Now the puppy can be kept free without tied up. By this time, it will mingle with the family and still not wet the carpets.

**Training the dog to "heel"**

Heel is a basic lesson in dog's training. It determines its adaptability to other lessons. It is essential that the pup learns to heel very well. This lesson should wait till the pup overcomes some of its curiosity and playfulness. It will learn more quickly and it will be less frustrating at that time.

- a) Introduce your pup to collar and leash as soon as it overcomes its inquisitiveness.
- b) Start with the pup on your left side and hold the end of the leash in your right hand. The left arm should hold the middle of the leash of your side. Start walking at a comfortable pace giving the command to heel. The pup may wander from side to

side in the beginning, but keep on training it and repeat the command as you progress.

c) During the next few days keep the pup closer to your side. Don't pull it if it lags behind. Give short, quick jerks repeating the command. Each walking can last for about 15 minutes. At the end of the walking you can reward the pup with a small treat to encourage it.

d) When the pup learns to heel consistently, it is time for the next lesson. It should adjust its pace to yours. Teach it to take the turns to left or to right, always keeping the correct position. Be patient. In case it fails, train it repeatedly and appreciate when it does things the right way.

**Teaching the dog to "sit" and "lie down"**

These are the most important obedience lessons that will make the dog's life easy to dwell with the family. When well trained, it will respond to the commands "sit" or "lie down" even when it is running at full speed. You will find it very easy, to successfully train your dog even without previous experience by just remembering the 3 R's of dog training: Reason, Repetition and Reward.

a) Teaching the dog to sit is an easy job. To train the dog to sit, hold the leash close to the collar with your right hand and push the rear of the dog with the left hand saying "sit". It will understand the command and this can be repeated a few times to make it understand the command. Remember, the pets like a soft voice.

b) While walking at heel, give the command "sit", pull the leash upwards with the right hand and push downwards on the dog's hind quarters with your left.

c) When the dog learns the lesson on tight leash, gradually make the dog do it on loose leash and

then without leash. Appreciate it and give it a small treat at the end of every successful lesson.

d) After the "sit" command, it is time for it to learn "lie down". Start with the sitting command and then command it to lie down. Kneel beside the dog and pull down the leash forcing it down

e) Don't pat your dog when it responds to lie down. (this makes it want to get up again). The appreciation can be showed by way of a small treat put on the ground so that it need not move to get it.

**Health Care**

Make it a point to visit your nearest friendly veterinarian once in a month or so. This is just to get your pet accustomed to its handling and to have a regular checkup. A good veterinary care, proper feeding and management would reduce most of the pups ailments. In case your pup comes in contact with ectoparasites, do not panic. Immediately take it to a veterinarian for prompt attention.

**A Healthy Pup**

When choosing a puppy, examine carefully from head to toe, to make sure it is bright and healthy.

**Using a Choke Chain**

When using a choke chain, make sure you put it on correctly. When applied the right way, the chain will automatically loosen when you stop pulling. When put on backward, it will not loosen when you stop pulling.

**Nail Clipping**

A dog's nails need to be trimmed about every two weeks. Dogs that regularly walk on concrete wear down their nails and need less frequent trimming. Use a sharp trimmer. A dog's nail is shaped like a crescent.

<b>Always consult your veterinarian if something, in your home - health exam doesn't seem right or if you notice any of the signs below</b>
Loss of appetite for more than a day.
Trouble in eating or mouth pain.
Sudden weight loss or weight gain noticed by weighing or by a rib check.
Prolonged gradual weight loss.
Fever.
Pain.
Vomiting more than three times. Call Vet immediately if vomitus is bloody or dark.
Diarrhoea for more than a day. Call Vet immediately if bloody.

Change in bowel habits for more than a day.
Excessive drinking for more than a day.
Increased urination, sudden accidents in the house, difficult urination, straining, bloody urine or decreased urination.
Excessive salivation.
Sluggishness, unwillingness to exercise or behaviour changes for more than a day.
Excessive itching or scratching, including ear rubbing or head shaking.
Lameness that does not improve within a day
Seizures or convulsions.
Eye discharge for more than a day. Squinting or discomfort

**Vaccination Schedule**

Make it a point to visit your nearest friendly veterinarian once in a month or so. This is just to get your pet accustomed to its handling and to have a regular checkup. A good veterinary care, proper

feeding and management would reduce most of the pups ailments. The following schedule may help your pup and it is not necessary to follow as it is and the pet owner must consult the neighborhood Veterinarian for the vaccinations.

Weeks	What to do
3 weeks	Deworming the pup for round worms and hook worms
4 weeks	Vaccination against Parvovirus and Distemper if bitch is not vaccinated.
5 weeks	Booster Vaccination against Parvovirus and Distemper.
6 weeks	Vaccination against Parvovirus and Distemper if bitch is vaccinated.
7 to 8 weeks	Booster Vaccination against Parvovirus and Distemper
8 to 9 weeks	Parvo, Adeno, Leptospirosis, Coronavirus, Parainfluenza, Bordetella
8 to 12 weeks	Deworming and Antirabies Vaccination
15 to 16 weeks	Booster vaccination for Rabies, followed by yearly boosters.Regular deworming every 3 to 4 months, thereafter.\

Disease	Description	Vaccination
<b>Canine Distemper</b>	A usually fatal viral disease that causes respiratory, gastrointestinal and nervous system problems.	Every 3 weeks until 12-14 weeks (6,9,12 weeks or 8, 11, 14 weeks), followed by an annual booster
<b>Canine Hepatitis</b>	A viral disease of the liver.	Included with distemper vaccine
<b>Parainfluenza</b>	A virus that is part of the group of viruses and bacteria known to cause kennel cough, the canine equivalent of our cold.	As above
<b>Viral Diarrhoea</b> Canine Parvo virus Canine Corona virus	A dangerous and sometimes deadly (especially in puppies) intestinal disease	As above, but some vets vaccinate for Parvo until 5 months of age against corona at 8-9 weeks of age.

Disease	Description		Vaccination
<b>Leptospirosis</b>	A bacterial disease that affects the liver and kidneys.		As above, but many veterinarians do not include until 9 weeks of age
<b>Rabies</b>	A deadly viral disease of the nervous system that affects all mammals, including humans. May be difficult to detect.		Included with distemper vaccine
<b>Kennel cough</b>	A group of viruses such as CAV2, CAV 1, Parainfluenza and bacteria such as Bordetella bronchiseptica are known to cause kennel cough		First at 8-9 weeks and booster after 4 weeks and annual vaccination
Disease	Primary vaccination	First Booster	Further Booster
DISTEMPER	7 to 9 Weeks	12 to 14 weeks age	Every Year
HEPATITIS	-Do-	-Do-	-Do-
LEPTOSPIROSIS	-Do-	-Do-	-Do-
ADENOVIRUS	4 th Week	8 - 12 Weeks	-Do-
PARVOVIRUS	4 th Week	8 - 12 Weeks	-Do-
PARAINFLUENZA	4 th Week	8 - 12 Weeks	-Do-
RABIES	12 th Week	6 Months age	-Do-
<b>▶ GUIDELINES BEFORE VACCINATION</b>			
▶ Vaccinate only Healthy dogs.			
▶ Deworm and detick before vaccination.			
▶ Vaccinate in early morning hours or on evening hours.			
▶ Advise the owner not to put the dog under stress (i.e. Vigorous exercise) for 3 days after vaccination.			
▶ Advise the owner not to give a bath to the dog a day before and 3 days after vaccination.			
▶ Advise the owner not to feed the dog with heavy diet such as eggs, meat, beef etc; and give only light feeds like bread, biscuits and rice for 3 days after vaccination.			
<p><b>Information and advice contained is for your consideration only. Please consult your veterinarian for specific advice concerning the care and treatment of your pet. Additional boosters are required as suggested by your Vet. The above is a suggested schedule but may vary from place to place and by practice.</b></p>			

**First Aid**

After doing the necessary first aid please consult your friendly veterinarian.

**Cardiac Massage**

This is required if your dog's heart fails,

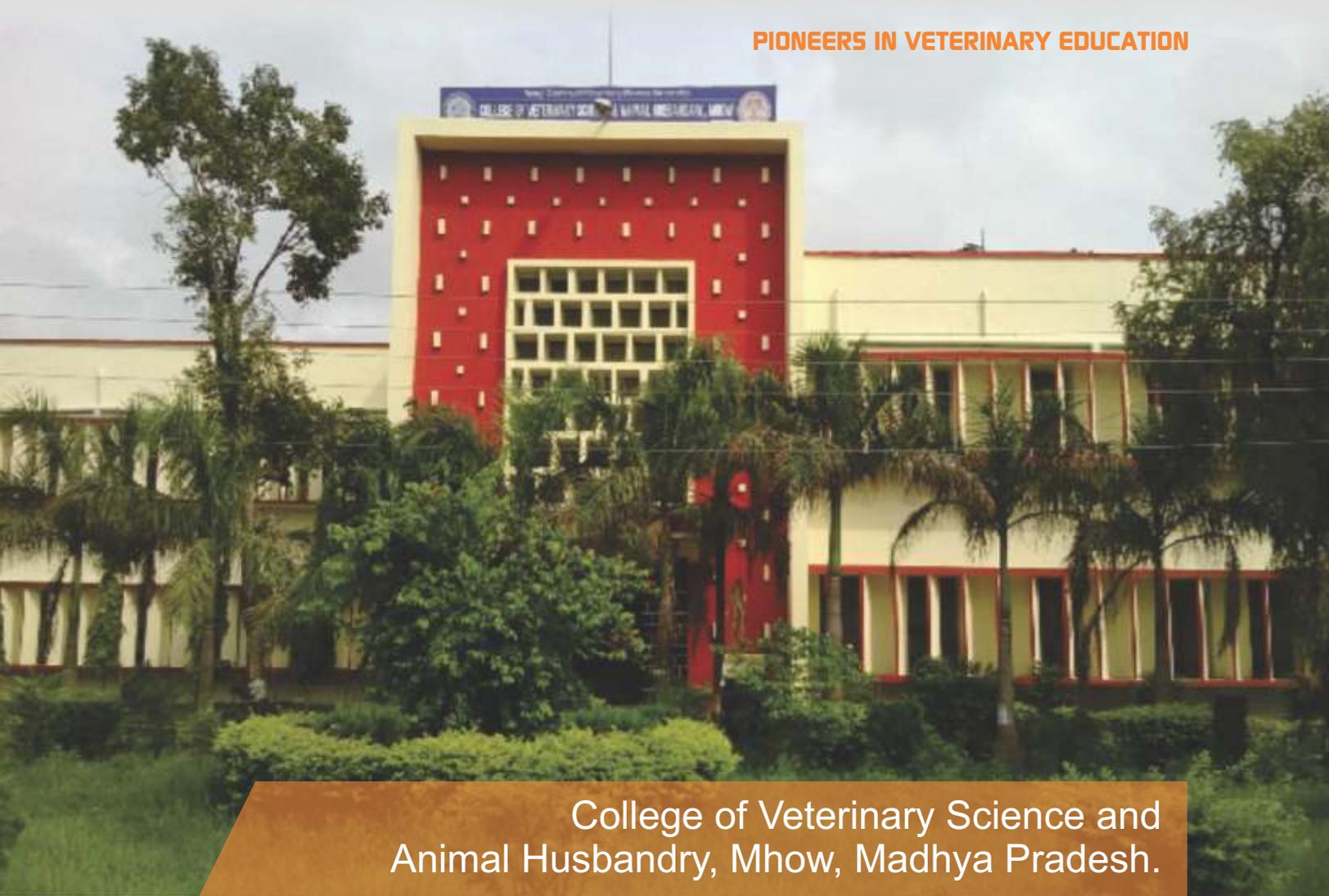
1. With the dog lying on its right side, first listen for a heartbeat. If not feel for its heart beat with your fingers on the chest wall behind the dog's elbows on its left side.
2. If you feel nothing, squeeze rhythmically with your palms, placing one hand on top of the other, as shown, at two second intervals.

**Fractures**

Broken lower leg bones can sometimes be straightened gently bandaged and then taped or tied with string to make shift splint, e.g. a piece of wood or rolled up newspaper or cardboard. Otherwise support the leg to prevent any movement. Take the dog to the Vet immediately.

**Artificial Respiration**

Use mouth to mouth resuscitation by cupping your hands over its nose and mouth and blowing into its nostrils every 5 seconds. This is for helping a dog which has a clear airway but cannot breath. It is usually resorted following a road accident, shock, drowning, etc. if unsuccessful, take the dog to the Vet immediately.



## College of Veterinary Science and Animal Husbandry, Mhow, Madhya Pradesh.

### Historical Glimpses

From its origin almost sixty years ago, College of Veterinary Science and Animal Husbandry, Mhow (M.P.) has offered a distinctive education in a unique setting. The Institute takes a legitimate pride in its contributions and distinguished services pro rata in

achieving the goals. The Institute has its long heritage and glorious history. The foundation stone of the college was laid on 27th December 1954 by Shri V.V. David, the Minister of Labor and Dr. R.L. Kaushal the founder Principal.

The College was established as "College of Veterinary



Late Dr. R. L. Kaushal  
Founder Principal  
(1955-65)



Science and Animal Husbandry cum Livestock Research Institute” on 12th July 1955 in the lap of Malwa region.

The present premises of the college were inaugurated by the first Prime Minister of India Pt. Jawaharlal Nehru.



College became the constituent unit of the Jawaharlal Nehru Krishi Vishwa Vidyalaya, Jabalpur on 2nd October 1964. In its more than 60 years of eventful journey the institute has been the torch bearer for the scientific development in the area of Veterinary Science and Animal Husbandry. After the establishment of Rajmata Vijayaraje Scindia Krishi Vishwa Vidyalaya, Gwalior on 19th August 2008, this college became the constituent unit of this Vishwa Vidyalaya. Later the college became the constituent unit of newly established Madhya Pradesh Pashu Chikitsa Vigyan Vishwa Vidyalaya, Jabalpur on 3rd Nov. 2009. Now the new name of University is Nanaji Deshmukh Veterinary Science University (NDVSU) w.e.f. 3rd Nov. 2012.

Total area of institute is about 263 acres. The existing facilities are as per norms. The Institute at its headquarters functions through 17 departments and other units like TVCC, ILFC, Ambulatory clinic, Production farms (Dairy, Poultry, Piggery, Goatry), ARIS cell, a modern computerized library, medical dispensary, NSS, NCC, etc. Big boy's hostels and girl's hostels facilities to accommodate approx. 240 boys and 65 girls, respectively, and one family hostel are there in campus. Diploma college building is also the part of campus.

Apart from providing useful information's through regular publications, newsletters, handouts, etc., the institute is involved in various development activities /programmes by various scientific, professional and social organizations. The strong foundation of this Institute is its infrastructure, faculty competence, student's capabilities and academic achievements, patience, persistence and perseverance.

**Strength of College**

1. **Strength:** Late Dr. Radhakrishnan former president of India an eminent educationist had

said “A nation cannot rise above the level of the teachers” this institute has devotated, highly qualified teachers of high academic standard. Trained from prescribed, appropriate professional and academic bodies and engaged in effective quality control and management of teaching & research and extension load.

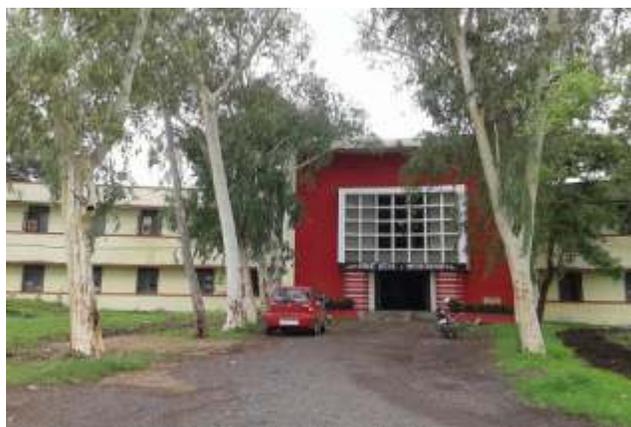
2. Sustainable and substantial growth of the profession have attracted more admissions in the veterinary education.
3. The institute has well established infrastructure to maintain standard of veterinary education as recommended by VCI and sufficient space for further expansion as per the need in future.
4. Adequately equipped laboratories are available for bio technological intervention and molecular examination of material for diseases diagnosis and conformation and training to student during UG, PG and Ph.D.
5. Livestock farm instructional livestock farm is available for dairy animals and one of the best poultry farms of indigenous poultry 'kadaknath' a rare bird in country.
6. Sufficient number of spacious classrooms equipped with LCD, laptop and screen are available as smart classrooms.
7. TVCC, of the college is equipped with modern gazets for diagnosis of diseases, C- Arm X-Ray, lapro-endoscopy unit, ultra-sonography; clinic pathological diagnosis lab is also available in TVCC. The diagnosis is possible by PCR, ELISA, biochemistry parameters and enzymatic studies.
8. A well-equipped examination hall is available with CCTV and Jammer to reduce the possibilities of manace of copying.

9. For extracurricular activities an auditorium is available.
10. To keep students fit and exercise regularly, a well-equipped Gym is available.
11. Adequate no. of boy's hostels is available to accomodate more than 200 boys.
12. Placement cell is working efficiently and number of students get job in respectable organizations.
13. College is having its own rain water storage reservoir to start fish production.
14. In the vicinity of college four national level institute like IIT, IIM, Centre for advance technology (CAT); A department of atomic energy of India institute and BRUSS (Baba Saheb Bheem Rao Ambedkar University for Social Science) exists and scientific innovation and informations are shared.
15. College is also having adjoining institute of

- biological products and vaccinology of M.P. Govt.
16. College is having one of the good sports ground to facilitate sport activity among students.
17. College is having NCC Unit setup by the Remount Veterinary Corps of Indian Army with horse line and riding facilities.

**Organizational Set- up and Infrastructure**

The organizational set up of college is in conformity as per VCI. Various committees and sections of college exercise their power at various levels to coordinate and regulate administration, education, research and extension. The Dean is the main authority empowered to monitor, supervise and control various activities of the college. Academic section is the principal body which frames and is responsible for maintenance of standard of education and examination of the college. Account section works on matters relating to purchase and finance of the college. The faculty members of the college are highly qualified and efficient.



Library of the college has many sections like stock section, reference section, and study section for undergraduates, post graduates and faculties, journal section, newspaper section, etc. Hindi and English paper is daily delivered in library to update the day to day information to student, faculty and staff. In addition, employment newspaper is available. All the livestock farms of college are utilized for production, teaching, research and extension purposes. To disperse the knowledge from laboratories to the farmers and public, extension department is responsible which organizes camps, give demonstration to the farmers on various aspects of animal health and production. Clinical camps are organized by medicine department and TVCC.

**Introduction**

S. No.	Departments of college	Laboratories
1.	Microbiology	Bacteriology and Mycology lab, Virology and Molecularbiology lab, Immunology lab, washing and sterilization lab
2.	Pathology	UG lab, PG lab
3.	Anatomy	UG lab, PG lab , Histology lab, Osteology lab cum museum, Dissection lab
4.	Physiology	UG lab, PG lab
5.	Parasitology	UG lab, PG lab
6.	Pharmacology	UG lab, PG lab
7.	Genetics	UG lab, PG lab, computer lab
8.	Nutrition	UG lab, PG lab, one analytical room
9.	VPH	UG lab, PG lab
10.	Biochemistry	UG lab, Central lab
11.	Poultry Science	UG lab
12.	Gynaecology	2 UG lab, PG lab, AI lab
13.	Surgery	UG lab, PG lab
14.	Medicine	UG lab, PG lab
15.	LPT	Analytical lab, milk technology lab, meat technology lab
16.	LPM	UG lab, PG lab, ILFC lab (dairy farm)
17.	Extension	Audio visual lab, Training hall

extension education to livestock owners of nearby areas and entire M.P.

**Vision**

1. Produce competent and skilled professionals in the field of animal health and production and allied sectors.
2. To empower the graduates with latest knowledge and technologies to achieve heights in profession.
3. To undertake region-wise, population –wise, environment-wise, season-wise, required field

**Mandate**

1. Strengthening hand on training to students with special emphasis on capacity building.
2. To provide quality education to students.
3. Providing opportunity to faculty to improve their scientific and working capacity and capability to make the institute a vibrant organization.
4. Undertaking need based applied and basic research and dissemination of outcome through publications.
5. Educating livestock owners, farmers and to catalyze them for improvement in productivity and reproductivity of their livestock and economy.
6. To impart health services to livestock and

based basic research adopting modern technology.

4. To validate indigenous traditional knowledge on scientific basis.
5. To provide efficient extension services at the doorstep of farmers and livestock owners and motivate them to adopt animal husbandry based vocations as a support for their economic growth and social empowerment.
6. To ensure enhanced production and reproduction through effective disease surveillance and

diagnosis, healthcare and vaccination programme.

**Challenges**

1. To substantially improve the faculty strength.
2. To achieve at par research excellence through external research fund support from the state and central government agencies.
3. To produce veterinarians and other technocrats related to animal health and allied sectors who become job providers not the job seekers.
4. To refurbish and maintain the infrastructure.

**Teaching & Curricular Attainments**

The college offers undergraduate and postgraduate teaching programmes. College has highly qualified staff and infrastructure to maintain a high standard of teaching. There are 17 departments as per VCI norms with well equipped laboratories. Modern teaching aids like computer, LCD projectors, transparencies and Internet facilities are available. An independent Agriculture Research Information System (ARIS) cell functions in the college for computer teaching and information technology related to advancements in Veterinary Science. The departments have excellent laboratories and museums with huge collection of rare specimens and slides. A well equipped Teaching Veterinary clinic service complex has been established in the college for teaching, clinics and diagnostic. The disease investigation laboratory in the complex provides facility of clinical haematology and microbiology, biochemistry, histopathology and examination of biopsy materials. The important diagnostic tools, viz electrocardiography, radiography and sonography for the diagnosis of animal diseases and teaching to students are available in the complex.

**The college of Veterinary Science and A.H. is running three academic programmes:**

1. Bachelor of Veterinary Science and A.H. (B.V.Sc. and A.H.) (As per Veterinary Council of India regulation).
2. Master of Veterinary Science and A.H. (M.V.Sc. and A.H.).
3. Doctor of Philosophy (Ph.D.).

**Classrooms and Laboratories**

The classrooms and laboratories are spacious and are well equipped with modern equipments and instruments. Classrooms are in theater pattern and are provided with modern audio-visuals like LCD projectors, computers with accessories. For surveillance CCTV cameras have also been installed in classrooms. There is a big museum in the Department of Anatomy & Histology where there is huge collection of skeletons of various animal species.



**Research**

Research has been an integral part to keep pace with the advancements in the field of Veterinary and Animal Science. The scientists have been engaged in research work since the inception of the college. The scientists and faculty members have published more than 2000 research papers in various national and international journals of repute. A new species of filarid worm *Gallifilaria mhowensis* was recorded for the first time in the world from this college. One of the pioneer research works on Ranikhet vaccine in poultry was carried out in the institute and various prestigious awards and honours were received by the faculties of the institute. Hari Om Ashram Trust Award in 1975 was given to Dr. B.S. Malik, Dr. S.K. Tanwani and Bhatnagar and Nair memorial award in 1978 to Dr. B.S. Malik and Dr. S.K. Tanwani for the research work on CDF66 strain.

Systematic and coordinated approaches to research in the different disciplines have been adopted. Research work on public health diseases of zoonotic importance, food borne and other microorganisms, environmental pollutants, toxicities, herbal formulations, feed analyses and formulation related to animal nutrition and studies on economic traits of livestock and poultry are being carried out as a part of curricular research programme.

A NATP project on weather based forecast of animal diseases and a ICAR ad-hoc research project on "Survey, Evaluation and Characterization of Kenkatha breed of cattle" is completed recently.

**Ongoing Externally Funded Projects**

Sr. No.	Title of Project	Department	Funding Agency
1.	Special Programme for Dairy Development - Protein Supplementation	LPM	RKVY
2.	Strengthening facilities for conservation and scientific evaluation of Kadaknath breed of fowl.	AGB+Poultry	RKVY
3.	Strengthening of facilities for Development of improvised vaccines against bacterial diseases of dairy animals in M.P.	Microbiology (Finished)	RKVY

**Extension**

Consultancy and advisory services are provided in the area of livestock health, production, feeding and management to the State Veterinary Department, Livestock owners, NGO's and people belonging to tribal areas. To extend the benefits of research findings and to make the utility of the college extension activities in rural areas training programmes for the farmers and field veterinarians, animal health camps, livestock awareness camps, kisan goshti, film shows, exhibitions, cattle fairs, Kisan Mela, farmers meet, vaccination and treatment camps and exhibitions demonstrations

and lectures are being organized by the institute. A short period training program on dairying and poultry keeping for the rural masses were also organized. A well-equipped Ambulatory Clinic of the college visits to several rural areas for timely treatment of diseased animals and pregnancy diagnosis, artificial insemination and prevention of infectious diseases. Professional services have also been rendered through the well-equipped Veterinary hospital with X-Ray and indoor facilities. The aligned animals from adjacent villages as well as the patients coming from far-flung areas of the state take the advantage of this hospital. The technical staff of this college arrange lectures and demonstrations for farmers as well as for field veterinarians.

**Hospital**



**Aris cell**



**Auditorium**



There are 09 computers in the ARIS Cell. Internet facilities are provided through ERNET V Sat. The internet facilities are also extended to other department and sections of the college. Data base in the form of VET CD and BEAST CDs (1973 to 1995) are available for PG students.

**Library**

1. Book Bank Facility, SC-ST Book Bank Facility and Reference Books facility are available.
2. Books, Journals, Thesis, Newsletters, News papers- Danikbhaskar, Patrika, Times of India, Rojgar Nirman, Employment News etc. are available.



3. Establishment work of “e-Library” facility is in progress, which will be helpful to access the e-Journals and other e- resources.
4. No. of books (1.4.2014 to 31.3.2015) – 1330 Books (purchase is in progress) of Foreign category, Indian reprints category and Indian Books Paperback / Hard bound category has been added in the college library.
5. No. of e- resources (1.4.2013 to 31.3.2014) - 35 Educational CDs are also added in the college library and 32 Video CDs on ICAR technologies are already present in library. Which are highly informative and helping to upgrade the knowledge of the students, teachers. It is also helpful in the extension work of the college.
6. Wi-Fi facility- The work for this facility is in progress, which will be beneficial for the students, research scholars and faculty persons in the library premises.
7. Journal and thesis section- it has been created in the library.
8. Library Digitalization- The software for library automation has been procured in this year which would helpful in efficient library management and also to provide Good Library Services.

**College Farms**

**Agriculture / Pasture Farm**

About 38.55 Hectares of land is under cultivation, 2.55-hectare land is irrigated and 18 hectares of land is partially irrigated. This farm is producing soybean and wheat, apart from these barseem fodder production is done to fulfill the nutritional requirements of the dairy animals.

In dairy farm of the college, cattle (Gir, HF) and Murrah Buffaloes are being reared. Milk produced at dairy farm is distributed to students, faculty, staff members and other nearby people. Dairy farm animals are also used for teaching, extension and research purposes on different aspects of animal health, production and reproduction. To maintain good health regular vaccination of dairy animals and testing during illness are done.

**Dairy farm**



**Piggery and poultry farm**



**Poultry**

Kadaknath birds are reared in poultry farm, under project. These birds are also used for teaching, extension and research purposes.

ASCAD trainings held in college in Depts. of Microbiology, VPH, Medicine, Surgery, Nutrition

# Guidelines to Authors

Raksha Technical Review recommends the following Guide lines to authors while submitting their manuscripts. These Guide Lines are essentially adopted from standard international journals to keep the quality of the material published.

## Introduction

Raksha Technical Review is a comprehensive and pre-eminent journal for the Veterinary profession which would like to act as an interface between academics, those in Research and Development and Veterinary profession.

**Types of paper:** Raksha Technical Review publishes primary research papers, review articles, short communications, letters and commentary. Contact details for submission: Papers should be submitted to the Editor or Publisher using the following email addresses:

**rtr@indimmune.com**

**Page charges:** We do not levy any page charges for publishing the articles

## PLEASE NOTE

**Ethics in Publishing:** Send only original articles. The work described in your article must have been carried out by you in accordance with the guidelines in vogue in your institute and by following all statutory requirements. Manuscripts submitted as review or commentary must be your own informed opinion on the subject.

**Conflict of interest:** All authors must disclose any financial and personal relationships with other people or organizations that could inappropriately influence (bias) their work. Examples of potential conflicts of interest include employment, consultancies, stock ownership, honoraria, paid expert testimony, patent applications / registrations, and grants or other funding.

**Submission declaration and verification:** Submission of an article implies that the work described has not been published previously (except in the form of an abstract or as part of a published lecture or academic thesis) , that it is not under consideration for publication elsewhere, that its publication is approved by all authors and tacitly or explicitly by the responsible authorities where the work was carried out, and that, if accepted, it will not be published else where in the same form, in English or in any other language.

**Contributors:** Each author is required to declare his or her individual contribution to the article: all authors must have materially participated in the research and /or article preparation, so roles for all authors should be described. The statement that all authors have approved the final article should be true and included in the disclosure.

**Authorship:** All authors should have made substantial contribution to all of the following: (1) the conception and design of the study, or acquisition of data, or analysis and interpretation of data, (2) drafting the article or revising it critically for important intellectual content, (3) final approval of the version to be submitted.

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**Language and language services :** Please write your text in good English (American or British usage is accepted, but not a mixture of both)

**Use of word-processing software:** It is important that the file be saved in the native format of the word processor used. The text should be in single-column format. Keep the layout of the text as simple as possible. Most formatting codes will be removed and replaced on processing the article. In particular, do not use the word processor's options to justify text or to hyphenate words. However, do use bold face, italics, subscripts, superscripts etc. When preparing tables, if you are using a table grid, use only one grid for each

Individual table and not a grid for each row. If no grid is used, use tabs, not spaces, to align columns. The electronic text should be prepared

in a way very similar to that of conventional manuscripts.

## Essential title page information

- Title concise and informative. Titles are often used in information-retrieval systems. Avoid abbreviations and formulae if possible
- Author names and affiliations. Where the family name may be ambiguous(e.g., a double name), please indicate this clearly. Present the authors' affiliation addresses (where the actual work was done) below the names. Indicate all affiliations with a lower-case superscript letter immediately after the author's name and in front of the appropriate address. Provide the full postal address of each affiliation.
- Corresponding author. Clearly indicate who will handle correspondence at all stages of refereeing e-mail address and the complete postal address. Contact details must be kept up to date by the corresponding author.
- Present/permanent address. If an author has moved since the work described in the article was done, or was visiting at the time, a "Present address" ( or 'Permanent address") may be indicted as a footnote to that author's name. The address at which the author actually did the work must e retained as the main, affiliation address. Superscript Arabic numerals are used for such footnotes.

**Abbreviations:** Define abbreviations that are not standard in this field in a footnote to be placed on the first page of the article. Such abbreviations that are unavoidable in the abstract must be defined at their first mention there , as well as in the footnote. Ensure consistency of abbreviations throughout the article.

**Footnotes:** Footnotes should be used sparingly. Number them consecutively throughout the article, using superscript Arabic numbers.

**Tables:** Number tables consecutively in accordance with their appearance in the text. Place footnotes to tables below the table body.

**References:** Citation in text

Please ensure that every reference cited in the text is also present in the reference list ( and vice versa).  
Reference style

**Text:** Indicate references by number(s) in brackets in line with the text. The actual authors can be referred to, but the reference number(s) must always be given.

**List:** Number the references (numbers in brackets) in the list in the order in which they appear in the text.

**Example:** 1) Crowther, J.R. and Abu Elzein, E.M.E. (1979). Detection and quantification of Foot and Mouth Disease virus by enzyme labeled immunosorbent assay . J.Gen.Virol.42:747

**Submission checklist:** The following list will be useful during the final checking of an article prior to sending it to the journal for review. Please consult this Guide for Authors for further details of any item.

Ensure that the following items are present: One Author designated as **corresponding Author:**

- Email address
- Full postal address
- Telephone and fax numbers
- All necessary files are uploaded
- All figures and photographs with captions
- All tables (including title, description, footnotes)

## Further considerations

- Manuscript has been "spell checked" and "grammar-checked"
- References in the correct format for this journal
- All references mentioned in the Reference list are cited in the text, and vice versa
- Permission has been obtained for use of copyrighted material from other sources ( including the Web)

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Looking forward to your early response.

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**Toll Free Number: 1800 425 5363**

The rating scale is 1 to 5 (1 is poor and 5 is excellent)

1. Appearance

1	2	3	4	5
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2. Article

1	2	3	4	5
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3. Information and content

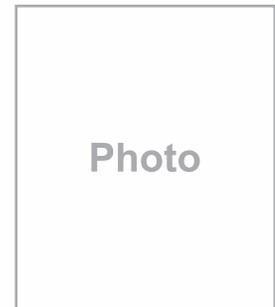
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4. Language and vocabulary

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5. Quality of photographs

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- **Appearance:** Color combination, design and layout
- **Article:** Reputation of the author, relevance of the topic of the column and value addition to you.
- **Information and content:** Order of articles, quality of articles and relevance of the information
- **Language and vocabulary:** Spelling, grammatical correctness and appropriateness of vocabulary
- **Quality of the photographs:** Colour, contrast, placement and relevance

Suggestions, if any: .....

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## Human Health

### • Biologicals

- ABHAYRAB  
(Vero Cell Antirabies Vaccine)
- ABHAY RIG  
(Rabies Immunoglobulin)
- ABHAY-TOX  
(Tetanus Toxoid Vaccine)
- ABHAY-TAG  
(DPT Vaccine)
- ELOVAC-B  
(Hepatitis B Vaccine)
- VAXTAR-5  
(Pentavalent Vaccine)

### • Nutraceuticals

- VIVAGUT  
(Advanced Probiotic)
- VIVAGUT KID  
(Advanced Probiotic for Children)
- VIVAFIT  
(Mineral & Vitamin Supplement)
- VIVACAD  
(For Osteoporosis & Calcium Supplementation)
- VIVAFLORA  
(Pre & Probiotics for Bacterial Vaginosis)

### • Hormones

- INCEPTOVA HCG  
(Chronic Gonadotrophin Inj)
- INCEPTOVA HMG  
(Menotrophin for Inj)
- INCEPTOVA FSH  
(Urofollitropin for Inj)

## Animal Health

### • Biologicals

- RAKSHA OVAC  
(Foot and Mouth Disease Vaccine)
- RAKSHA OVAC ULTRA  
(NSP Free Foot and Mouth Disease Vaccine)
- RAKSHA TRIOVAC  
(FMD, HS & BQ Combined Vaccine)
- RAKSHA BIOVAC  
(FMD & HS Combined Vaccine)
- RAKSHA HS  
(Adjuvanted Vaccine of Pasteurella Multocida)
- RAKSHA HS BQ  
(Combined Vaccine for HS & BQ)
- RAKSHAVAC T  
(Theileriosis Vaccine)
- BRUVAX PLUS  
(Brucellosis Vaccine S19)
- RAKSHA ET  
(Clostridium Perfringens Type D Vaccine)
- Raksha PPR  
(Peste Des Petitis Ruminants Vaccine)
- RAKSHA SP  
(Sheep Pox Vaccine)
- RAKSHA BLU  
(Bluetongue Vaccine)
- RAKSHARAB  
(Cell Culture Antirabies Vaccine)
- STARVAC R  
(Cell Culture Antirabies Vaccine)
- STARVAC 7  
(Multicomponent Vaccine)
- MEGAVAC 6  
(Multicomponent Vaccine)
- MEGAVAC P  
(Canine Parvovirus Vaccine)
- MEGAVAC CC  
(Canine Coronavirus Vaccine)
- MEGAVAC P NASAL  
(Vaccine for Parvo Protection)
- BRUVAX RB 51  
(Canine Coronavirus Vaccine)
- MEGAVAC 7  
(Multicomponent Vaccine)
- CYSVAX  
(Porcine Cysticercosis Vaccine)

### • Formulations

- NIMOVET  
(Nimesulide Inj., Bolus)
- OXFENVET  
(Oxfendazole - Broad Spectrum Dewormer)
- CLOSITEL  
(Closantel - Proven Flukicide)
- IVECTIN  
(Ivermectin - Endo Ecto Parasiticide)
- XYLAXIN  
(Xylazine HCl - Pre Anesthetic)
- TIKKIL  
(Cypermethrin - the Acaricide in Powder Form)
- BOVOPLEX ORAL  
(The Vital Health Tonic)
- INIMOX FORTE  
(First Line Antibiotic against Wounds & Pyrexia)
- BOVICEF  
(Cefiofur Inj., Antibiotic against Metritis)
- SEFTRIVET  
(Ceftriaxone Inj., The Ideal Choice against Mastitis)
- GARBHAMIN  
(Chelated Minerals & Coated Vitamins)
- ACTIPET  
(Multivitamins & Aminoacids Supplement)
- PET FORTE  
(Oral Calcium Suspension)
- Ca<sup>++</sup>9  
(Calcium Chewable Tablets)
- HEX-D  
(Herbal Digestive Powder)
- PROWOMB  
(Herbal Uterine Syrup)
- TIKKIL RAZ  
(Broad Spectrum Ectoparasiticide)
- TIKKIL-H  
(Ecto-parasiticial Herbal shampoo)
- ZUSPRAY  
(A Herbal Spray for Open wounds)
- ELP  
(Toldimfos sodium)
- GLUFLU  
(Flunixin Meglumine Inj)
- MORBAXIN  
(Marbofloxacin 10%)

### • Nutraceuticals

- VETFEN 600  
(Animal Feed Pellets with Fenbendazole)
- CALSAGAR PLUS  
(Calcium Feed Supplement)
- GOUDHARA SHAKTI  
(Bypass Fat for Overcoming Energy Deficiency in Early Lactation)
- GOUMIX  
(Area Specific Mineral Mixture)
- GOUSAC  
(Rumen Specific Live Yeast Culture)
- GOUVIT  
(High Quality Vitamin Premix)
- KSHIRSAGAR  
(For Improved Milk Production)
- KSHIRSAGAR CHELATED  
(For Stress free Milk Production)
- 4P  
(Feed Supplement for Pre & Post Partum)
- MILKFUL  
(Feed Supplement for Subclinical Mastitis)
- R-VITA  
(For Raksha Vitamins and Minerals)

## **Vision**

“Shaping Global Healthcare by Spearheading the One Health Initiative”.

## **Mission**

“To Innovate, Produce and Market Quality Healthcare Products and Services to Improve and Extend lives”.



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