

RAKSHA TECHNICAL REVIEW

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**Lumpy Skin Disease
(LSD) in Cattle**

**Pericardial Lipoma in
A Senescent Maltese Dog
A Case Report**

**Management of Postpartum
Uterine Prolapse in
A Non - descript Doe**



INDIAN IMMUNOLOGICALS LIMITED

Reader's Desk



Excellent Book.

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Faridabad, Haryana



Very informative with full information.

Dr Pritam Chakravarthy
Dwaraka, Delhi



Excellent Journal.

Dr Ganesh Narian Dadhich
Kota, Rajasthan



Needs more content regarding small animal practice.

Dr Karthik Iyer
Mumbai, Maharashtra



Excellent Magazine.

Dr Ruby
Kota, Rajasthan



Magazine is informative.

Dr Aparna Gopinadhan
Chennai, Tamil Nadu



It is all good.

Dr Anuja Tiwari
Kota, Rajasthan



RTR has articles on current issues that imparts knowledge.

Dr AP Theurkar
Pune, Maharashtra



It is excellent magazine.

Dr Vikram Dave
Mumbai, Maharashtra



Include articles related to only one species in one issue.

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Prayagraj, Uttar Pradesh



Nice Journal. Being a private small animal practitioner, a separate journal should be there for them.

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New Delhi, Delhi



I am Dy Director of AH dept. of Gujarat State hereby requesting to send RTR to my address regularly, as it helps me to educate farmers and policy making decision.

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RTR journal is very good.



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Corrigendum

Article, *Traumatic Hyphema in A Kathiawari Foal: A Case Report*, printed vide Volume IX Issue 1 (Septemeber 2019 page no. 33) was credited to different set of authors. The same article is now printed in page no. 69 with correct set of author. Inadvertent error is deeply regretted.

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From the Editor's desk

COVID19 has almost debilitated several parts of the world over the past few months. For most part of 2020, the focus of Governments and people was towards managing the pandemic.

While the efforts to develop vaccine against COVID19 are ongoing, there was a silent outbreak in the form of Lumpy Skin Disease in cattle in many parts of the world. Also, there were reports about African Swine Flu outbreaks in the North-Eastern region of India. The current edition of RTR covers an article on Lumpy Skin Disease diagnosis and treatment.

In the large animal section, this issue covers few gynecology complications. Articles on *Postpartum Uterine Disorders in Bovine - A Review*, *Summer Mastitis - An Overview*, and *Successful Management of Partially Macerated Fetus in Murrah Buffalo: A case Report* discuss the critical aspects of cattle.

Aspects in control of worm infestations are presented via articles *Management Approaches to Control Parasitic Infection in Livestock* and *Parasitic Zoonotic Diseases in Livestock and Role of Management Practices to Reduce or Control of Their Emergence*.

In Grazers&Browsers section of this issue, *Contagious Caprine Pleuro Pneumonia(CCPP):A Transboundary Disease in Small Ruminants* has focused on the need for identification, treatment and its control. *Management of Postpartum Uterine Prolapse in A Non-descript Doe* has explained the treatment and management.

Focus on indigenous breeds is increasing recently. In this edition we have covered *Marathwadi Buffalo and Himalayan Yak* under large animal section. Similarly, in the companion animal section *Indian Gaddi Kutta or Pahari Leopard Hound*, a mastiff type mountain dog found in northern and western Himalaya regions in India is presented.

We are happy to publish few papers dealing with rare clinical conditions in canines. Cases relevant to diet-related Recurrent Hematuria in puppies, Cardiomyopathy, Pericardial Lipoma, Masticatory muscle Myositis and Urolithiasis are the relevant aspects discussed in detail.

Also, this edition includes the coverage of feline related cases dealing with *Clinical Management of Necrotic Right Eye in A Domestic Short Hair Cat* and *Congenital Deafness in Cat*.

We wish you a healthy environment and hope to gain normalcy very soon.

“Stay Safe, Wear Mask, Wash Hands and Maintain Social Distance”
to protect self, family and society.

Regards,

Dr Prasanna A Deshpande



Managing Director's Message

Dear Patrons,

Greetings!!

Our World is toiling hard to come to terms with the pandemic of the 21st Century, COVID19. No country could escape the wrath of this dreaded virus. Scientific communities across the globe are stretching beyond the limits to develop safe and authentic vaccines.

We, at IIL, are approaching the COVID vaccine development with an objective of "Single shot administration and long term protection". For this, we are working with Griffith University, Australia. Simultaneously, we are offering our manufacturing facilities to other vaccine researchers in advanced stages of development for formulation development and distribution of vaccine. IIL has the necessary scale up and downstream process capabilities to work in this direction. It is our belief that this approach enables the faster reach of vaccine to the public in these testing times.

On the operations front, being under the "Essential Services" category, IIL could continue the manufacturing and supply chain activities to provide valuable vaccines/formulations to all our customers. Several measures were taken and will continue further to protect the health of our employees and their families.

Economic compulsions and better understanding of the disease have led to the reopening of many establishments and the movement of people has increased significantly. Until a safe vaccine for COVID19 is developed, I urge everyone to take all precautions, including wearing of PPE, at all times.

We hope to see the Government's efforts in systematic easing of lockdown restrictions bring the economy back to the growth path.

Warm Regards

Dr K Anand Kumar

Lumpy Skin Disease (LSD) in Cattle

Srinivas K, Santosh B Ghadage, Adhir Chaubal, Surya Prasad V, Swapna Reddy E and Madhanmohan M
Indian Immunologicals Limited, Hyderabad, Telangana and V U T D C, TANUVAS, Madurai, Tamil Nadu.

Introduction

Lumpy Skin Disease (LSD) caused by Lumpy Skin Disease Virus belongs to the *Pox viridae* family from the genus *Capripox virus* principally affecting the cattle (*Bos Taurus and Bos indicus*) and water buffaloes (*Bubalis bubalis*) (1). The first case of the disease was reported in 1929 from Zambia (2). The LSD virus shares a close antigenic resemblance with *Sheep Pox Virus* (SPV) and *Goat Pox Virus* (GPV) on the basis of morphology, serology, and the cytopathic effects in tissue cultures. Lumpy skin disease is an infectious, eruptive, occasionally fatal disease of cattle and water buffaloes characterized by nodules on the skin and other parts of the body. Secondary bacterial infections further complicate the condition. The incubation period is approximately 4 to 14 days and can be longer in natural infections. Other synonyms for the LSD are *Neethling Virus Disease*, *Exanthema Nodularis Bovis* and *Pseudo-Urticaria*. General provisions on the disease are defined in chapter 11.9 in OIE (3). There is only one serotype of LSD virus.

Transmission

Biting arthropod insect vectors are implicated in the transmission of virus. Grazing on shared pasture lands, close proximity to water sources, temperature, rainfall and wet humid summer months have been associated with outbreaks of LSD and are consistent with vector-borne transmission of virus. It is also possible to witness outbreaks of LSD throughout the year. Experimental studies found no evidence for direct transmission of the virus between animals, instead transmission is strongly believed to be via arthropod vectors (4). It is not clear whether vector borne transmission is biological or mechanical and movement of unvaccinated cattle from infected areas poses a major risk. LSD has been successfully transmitted experimentally via *Aedes aegypti* mosquitoes and common stable fly, *Stomoxys calcitrans*. The arthropod vectors, flies and ticks are thought to inoculate the virus into the skin of the cattle with virus circulating in the blood stream producing viremia and then further disseminating into distant cutaneous sites, GI tract, respiratory tract, reproductive tract and other internal organs. LSD is host specific and evidence of infection in species other than cattle and buffaloes is not reported. Skin lesions are the major source of infection: although the virus is released

from body secretions and excretions including semen. Direct contact between animals does not play a major role in spread of the disease. Iatrogenic intra or inter herd transmission occur via contaminated needles during vaccination or if needles are not changed between animals. Eventually, affected animals clear the infection and there is no known carrier state for LSDV.

Disease distribution

In India, it was first reported from Odisha (Khairbani, Betnoti and Mayurbhanj) on 12th August 2019. Subsequently, the outbreaks were reported from other parts of Mayurbhanj, Bhadrak, Odisha. ICAR-National Institute of High Security Animal Diseases (ICAR-NIHSAD), Bhopal confirmed the virus identity by 16th November 2019. Epidemiological analysis and genetic characterization of Indian isolates revealed the closeness to South African isolates than to European isolates. During these outbreak episodes from Odisha, the researchers could detect the LSD virus genome by real time PCR from semen of naturally infected cattle (5). The disease is notifiable and classified under category A by OIE. The reasons for the sudden appearance of LSD in India could be due to climatic conditions conducive from globalization, importation of animals, disrupted livestock movement patterns associated with high insect activity and movement of people and goods. LSD cases are also reported from Chhattisgarh, Madhya Pradesh, Jharkhand, West Bengal, Assam, Andhra Pradesh, Telangana, Tamil Nadu, Maharashtra, Karnataka and Kerala. The disease is a classic example of a transboundary emerging disease and is not known to Europe till 2014 when it was first reported in Cyprus spreading further to different Balkan countries. Since 2019, several outbreaks of LSD have been reported from South Asia and East Asia. Outbreaks of LSD were reported in Africa till the year 2012 from where it began to spread to other parts of the world like Iran, Iraq, Jordan, Turkey, Russia, Balkan & Caucasus regions, Russia, Kazakhstan, Cyprus, Greece to France, middle east, west Asia, south and south east Asia (6). Recent outbreaks were reported from Bangladesh (July 2019) which was closely followed by Odisha outbreaks in the month of August 2019, China in August 2019 and Taipei in July 2020. The disease is endemic in most of Africa and few parts of Middle east and Turkey. The risk of transmission is very

high in hitherto unaffected countries. Cases of LSD are reported from Israel, Kuwait, Lebanon, Yemen, UAE, Bahrain, Oman, Saudi Arabia, Armenia, Azerbaijan, Bulgaria, Serbia, Kosovo, Macedonia and Montenegro (6, 7).

Clinical signs

Incubation period ranges from 2 to 5 weeks. The first sign of the disease is rise in temperature up to 41°C followed by appearance of nodules over the skin in next 3 days. The disease is an acute infectious viral skin disease of cattle characterized by fever, various lumps or nodules on the skin covering all parts of the body, lymphadenitis and edema of the legs, udder, genitalia and brisket (8, 9). A subcutaneous injection of infected material produces a painful swelling, fever, lacrimation, nasal discharge and hyper salivation followed by the appearance of characteristic and distinctive circumscribed eruptions/nodules of 0.5 to 5 cms in diameter on the skin from a mild to severe form in susceptible cattle in 3 to 5 days. Their numbers may range from a few to several hundreds in a single affected animal.

The skin nodules contain a firm, creamy-gray or yellow mass of tissue. The nodules or lumps are painful, involve the epidermis, dermis, and subcutaneous tissue. They are firm in nature, raised, round or circumscribed, may disappear and start again in other parts of the body or persist, the overlying skin sometimes necrosed, and sloughs to leave large open sores. The virus is present in blood, nasal, lachrymal secretions, saliva and semen. The skin lesions are often accompanied by oral, nasal and ocular discharge, lethargy, anorexia, a rapid drop in milk production. After 1–2 weeks the skin nodules become necrotic and slough off leaving ulcers and scars over the skin. The course of the disease lasts from 5 to 8 weeks.

In rare cases like in Foot and Mouth Disease (FMD) affected cattle, nodules may be found on muzzle, nasal and oral mucus membranes. Though infection rates are higher in affected cattle, the mortality is less. There is no evidence of human infection. Experimentally, buffalo, sheep, goats, and giraffes can be infected with mild clinical signs and are believed to be the reservoirs. The disease is more severe in young cattle though it is known to occur in all age groups of cattle. Pneumonia caused by the virus itself or secondary bacterial infections, and mastitis are common complications. Skin lesions in the legs and on top of the joints lead to deep subcutaneous infections complicated by lameness. When an animal with multiple skin lesions is sent to a slaughterhouse, subcutaneous lesions are clearly visible after the animal is skinned. In a postmortem examination, pox lesions can be found in digestive and respiratory tracts and

on the surfaces of internal organs. A few pictures of animals affected with LSD are shown in **figures 1 to 9**.

Economic losses

The disease inflicts significant economic losses due to reduction in body condition, milk loss, poor quality of hides, infertility problems in both males and females, abortions and serious trade restrictions and controls. Course of the disease is usually between 5 to 8 weeks. Full recovery of affected cattle takes several months and may prolong where secondary bacterial infection occurs and obstruction of respiratory passages by the lesions may lead to deaths in some cases especially in calves.

Morbidity and mortality rates

Morbidity and mortality in LSD outbreaks are lower than sheep pox and goat pox. Morbidity varies between 2 to 45% while the mortality is usually less than 10% (8). The resistance to the disease is influenced by immune status, age and breed rather than the virulence of the virus. Reports from LSD outbreaks in Middle East and Europe reveal a morbidity of 9%–26%, and mortality from 0.5% to 2%. Fifteen years' data from Uganda shows a morbidity of 4.77% and a mortality of 0.03 % indicating a lower prevalence. Pure bred and cross bred animals are more prone to the infection and younger ones suffer more severe form of the disease (9, 10).

Diagnosis

The disease can be diagnosed by the appearance of distinct cutaneous lesions which can also be seen in bovine herpes virus known as pseudo lumpy skin disease. In bovines, skin nodules are also caused by *Dermatophilus congolensis*. LSD virus shares close resemblance with SPV and GPV and grows on tissue cultures of ovine, bovine, monkey and rabbit origin producing cytopathic changes and intracytoplasmic inclusion bodies. The virus can also be propagated in chick embryos and eggs. Immunofluorescent or immunoperoxidase staining are largely helpful in specific identification of the agent. Virus isolation can also be attempted in skin biopsy samples. Virus Neutralization Test, ELISA, PCR and real time PCR techniques are helpful in detection of the antigens. International reference laboratories for LSD are located in Onderstepoort, South Africa and The Pirbright Institute, U. K (6). In India, suspected clinical samples are advised to be forwarded to ICAR- NIHSAD, Bhopal for further diagnosis.

Treatment

Antibiotics are advocated for the control of secondary bacterial infections and symptomatic

treatment with help from Veterinarian is useful in reducing the clinical signs. Administration of anti-pyretics, anti-inflammatory and anti-histamines to reduce the suffering coupled with administration of multi vitamins, antiseptics and fly repellants. Ethnoveterinary medicine is also useful to treat the infected animals. Feeding soft gruel, feed and fodder is advised in affected animals. LSDV is very resistant to physical and chemical agents, can persist in necrotic skin lesions for more than 30 days. LSDV is killed by 2% sodium hydroxide, 4% sodium carbonate, 2% chloroform and formalin, 2% phenol, iodine and quaternary ammonium compounds at 1: 33 dilutions and 0.5% respectively. Isolation and quarantine measures are helpful but to a lesser extent. The virus is susceptible to higher temperatures and gets killed at 55°C for 2 hours and at 65°C in 30 minutes.

Control by Vaccination

LSD is a vector borne disease which makes it difficult to predict and manage the spread and the best solution for the protection of animals is vaccination. Prophylactic vaccination using a live attenuated homologous Neethling strain vaccine administered subcutaneously has been in use in many countries and provides a robust immunity. The minimum recommended field dose (11) of the South African Neethling strain vaccines is $10^{3.5}$ TCID₅₀ although the minimum protective dose is $10^{2.0}$ TCID₅₀.

Heterologous protection by Sheep pox vaccine @ 0.5 ml by intradermal route is used in Egypt with a Romanian strain. A vaccine strain was developed in Kenya using a strain of sheep pox virus which had been passaged 16 times in lamb testis cells. A complete cross immunity had been demonstrated to exist between strains of sheep pox and LSD. This strain was used for many years at an immunizing dose of $10^{3.5}$ TCID₅₀. Capripox viruses are cross-reactive within the genus. Hence, it is possible to protect cattle against LSD using strains of Capripox virus derived from sheep or goats (12). However, it is recommended to carry out clinical trials and suitability studies and challenge studies using the most susceptible breeds, prior to introducing vaccine strains not usually used in cattle. Vaccination of cattle over 6 months of age offer protection. Goat pox vaccine is also used in a few countries as some cross protection is observed. Large scale vaccination combined with other appropriate control measures are the most effective way of limiting the spread and economic impact due to LSD. We as a country are fully braced up to face such emerging challenges as transboundary animal diseases are likely to hit in future too as recently we witnessed emergence of African Swine Fever (ASF) outbreaks in Assam, Arunachal

Pradesh and Meghalaya in 2020. The ASF disease was also confirmed by ICAR-NIHSAD, Bhopal in May, 2020.

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Fig.1 : Exposed skin lesion in cattle



Fig. 2 : Nodules on the head of a calf.



Fig. 3 : Numerous nodules over skin of a cattle



Fig. 4 : Nodules all over the body of a cattle.



Fig.5 : Cutaneous nodules on the muzzle of a cattle.



Fig. 6 : Nodules on the dewlap region with swellings.



Fig.7 : Extensive nodules all over the body in a calf.



Fig.8 : Skin nodules in a cow



Fig.9 : Skin nodules in a bullock

Postpartum Uterine Disorders in Bovine - A Review

C Pugazharasi, N Arunmozhi, T Sarath and R Suresh Kumar

Dept. of Veterinary Gynaecology & Obstetrics and Dept. of Clinics, MVC, TANUVAS, Chennai, Tamil Nadu.

Introduction

Parturition is a period of high risk for mother and young ones in all species, and cattle are no exception. As well as the risks of physical damage during the birth process or failure to release the placenta after parturition, there is often an upsurge of microbial infections through the reproductive tract in a cow. Some animals acquire infections of the uterus or mammary gland during late gestation, which may lead to premature parturition, or compromise fetal or calf health. However, the greatest impact on health and productivity is associated with microbial contamination of the uterine lumen after parturition. Amongst the mammals, *Bos taurus*, and particularly dairy cattle farmed in intensive systems, commonly acquire microbial contamination of the uterus (1). Indeed, 80–100% of animals have bacteria in their uterine lumen within the first 2 weeks after calving. Although immune responses progressively eliminate the microbes, up to 40% of animals still have a bacterial infection 3 weeks after calving (2). Of course, bacterial contamination does not always imply disease. The aim of the present review is to highlight the incidence, causes and consequences of uterine disease.

Normal postpartum events include

1. Uterine involution
2. Regeneration of the endometrium
3. Elimination of bacterial contamination from the uterus
4. Resumption of ovarian cyclical activity

The initial stimulus for these changes to occur is the expulsion of the fetus along with the associated membranes and fluids at calving. Uterine involution involves physical shrinkage, necrosis and sloughing of caruncles, and the regeneration of the endometrium (1). Following the loss of the allanto-chorion, there is necrosis of the uterine caruncles, which are usually sloughed by 12 days after parturition. Sloughing of the uterine caruncles contributes significantly to the rapid reduction in weight of the involuting uterus from 13 kg at parturition to about 1 kg 3 weeks later, because the caruncles account for over half of the weight of the uterus. There is initially regeneration of the endometrium in the inter-caruncular areas and then by centripetal growth of the cells over the caruncle.

Epithelial regeneration is complete by about 25 days after parturition, but the deeper layers of tissues are not fully restored until 6–8 weeks after calving.

The postpartum environment of the uterine lumen is in anaerobic environment which supports the growth of a variety of aerobic and anaerobic bacteria. Many of these bacteria are contaminants in the uterine lumen and are removed by a range of uterine defence mechanisms. However, uterine disease is commonly associated with microbial organisms like *Escherichia coli*, *Arcanobacterium pyogenes*, *Fusobacterium necrophorum* and *Prevotella species*. Indeed, *A. pyogenes*, *F. necrophorum* and *Prevotella species* have been shown to act synergistically to enhance the likelihood of uterine disease, and increase the risk of clinical endometritis, metritis, septic and puerperal metritis (3,4). Numerically the most prevalent pathogens are *E. coli* (37% of pathogenic bacteria isolated) and *A. pyogenes* (49%) (5). Furthermore, the *E. coli* infections appear to precede and pave the way for the *A. pyogenes* infection.

After parturition, steroid hormone concentrations decrease to basal values, and there is an increase in plasma follicle stimulating hormone (FSH) concentration within days of calving that stimulates the emergence of the first postpartum follicular wave. Subsequently, the first dominant follicle is selected around 10–12 days after calving (6,7). These events occur in all postpartum cows irrespective of periparturient disease, environment or dietary deficiencies. However, the first dominant follicle has three possible fates: ovulation and formation of the first postpartum corpus luteum (return of ovarian cyclical activity), atresia with the emergence of one or more follicular waves without ovulation (anoestrus), or formation of an ovarian follicular cyst (7). Early return of ovarian cyclical activity is generally accepted to be beneficial for subsequent fertility (8). However, it is suggested that an early postpartum first ovulation in the presence of uterine infection can lead to pyometra with persistence of a corpus luteum in the presence of pus within the uterine lumen (4).

Mechanism of Uterine Diseases

As the uterus is usually sterile, the presence of microbes or pathogen-associated molecules appears to provoke a substantial immune response.

The uterine immune response is generated by immune cells within the endometrium and by the endometrial stromal and epithelial cells. Indeed, it is the epithelial cells that are the first line of defence against microbes in the uterine lumen. Innate immunity in the genital tract is highly dependent on the expression of pattern recognition receptors (PRRs) to detect pathogen-associated molecular patterns (PAMPs). These PRRs, such as the family of Toll-like Receptors (TLRs), are highly conserved across phyla and detect a range of PAMPs associated with fungi, viruses and bacteria.

Binding of PAMPs to PRRs activates signal transduction pathways for mitogen-activated protein kinase (MAPK) and the nuclear factor-kappa B (NFκB) transcription factors, leading to secretion of prostaglandins, cytokines and chemokines. Epithelial and stromal cells express toll-like receptor 4 (TLR4), the innate immune receptor for lipopolysaccharide (endotoxin, LPS), which is the key PAMP of the common uterine pathogen *E. coli*. This concept of endometrial cell expression of PRRs is supported in other species by expression of other toll-like receptors for bacteria and viruses (9,10,11).

The principal hormones secreted by the endometrium are PGF2a and PGE2, respectively, and the secretion of these hormones is modulated by *E. coli* or LPS. The prediction from the in vitro work is that uterine disease would extend the luteal phase, which is what is observed clinically. Furthermore, exogenous PGF2a is an effective treatment for uterine disease and eicosanoids may modulate uterine immunity directly (12). In vitro, LPS stimulates progesterone secretion from mixed populations of luteal cells (including steroidogenic, endothelial and immune cell types) to a level similar to that seen with luteinizing hormone (LH), but at higher concentrations LPS kills the cells (13).

Incidence of Uterine Diseases

The placenta is normally expelled within 6 h of expulsion of the calf but if still present by 24 h, it is defined as a retained placenta. The incidence of retained placenta is between 2% and 5% of animals in a herd, but can be increased in cows with twins, after dystocia and where infectious agents are endemic. The expression of clinical uterine infection depends on the balance between factors such as the animal, immunity, the number and pathogenicity of the microbes, and the uterine environment. Typically, 25–40% of animals have clinical metritis in the first 2 weeks after calving, and disease persists in up to 20% of animals as clinical endometritis.

Although the clinical signs of uterine disease such as purulent material discharging from the uterus

into the vagina are readily detected, the role of subclinical uterine disease is less well characterized but is an emerging issue. Up to 50% of cows 40–60 days after calving had neutrophils in the uterine lumen or endometrium, concomitant with inflammation of the tissues, and subclinical endometritis reduces conception rates. Chronic endometrial scarring, obstruction of the uterine fallopian tubes and adhesions between the ovary and the bursa are other consequences of uterine bacterial infection. However, these are less of a problem in cattle than other mammals including humans, with the incidence of ovaro-bursal adhesions affecting about 2% of cows.

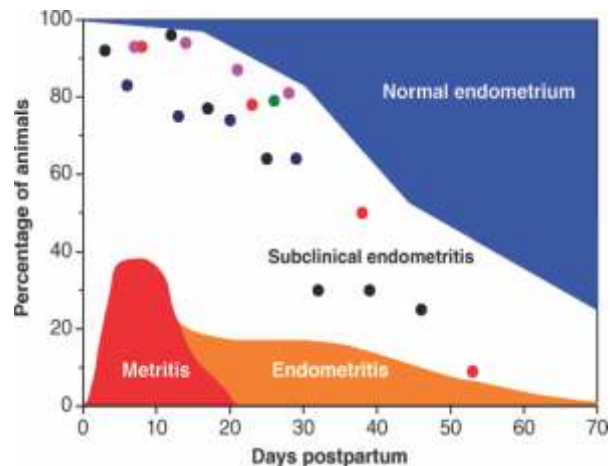


Fig.1. Incidence of uterine bacterial infection and disease in postpartum dairy cattle (14)

Bacteria can be isolated from the uterus of most cows during the postpartum period; each marker (●) indicates the percent of animals with bacteria isolated from the uterine lumen. The shaded areas represent estimates of the proportion of animals with metritis (■), clinical endometritis (■) or a normal uterus (■); the remainder of animals have subclinical endometritis.

Uterine Diseases

1. Retained fetal membrane (RFM)

The retained foetal membrane is main reproductive disorder in dairy cattle. It causes considerable economic losses in a herd due to decreased milk production, illness, and treatment cost, besides a decreased market value of the animal. Incidence of RFM varies from 5-10 % in cattle. Most cows will pass the afterbirth (placenta, cleansing or calf bed) within 6-8 hours of calving. Some cows take up to 24 hours. If the placenta is retained longer than this, the condition is classified as retained placenta or retained foetal membranes (RFM).

The condition is more common in dairy than beef herds and incidence and prevalence of the

condition varies from farm to farm and field. It is, however, well recognised that the effects of retained placenta afterbirth on individual cows vary from severe symptoms of fever and illness, such as ketosis, to non clinical signs at all, and the target levels in each herd have to be adjusted according to the significance of the condition within the herd.

Longer term effects of RFM are an increase in time to first service, lower first service conception rate and thus an increase in time to conception. Cows that abort, calve prematurely, have twins, have a difficult calving or any other debilitating illness during calving are more likely to cleanse poorly than full term, uncomplicated calving. Unhygienic calving conditions are an obvious risk for both retained placenta and metritis.

A variety of nutritional risk factors are associated with RFM. Milk fever and even sub-clinical calcium deficiency can be associated with an increased risk of RFM with older cows more at risk of lower blood calcium.

Overfat cows and vitamin E / selenium deficiencies have also been associated with increased incidence of retained afterbirth, although micronutrient imbalances are unlikely to be the most important cause of RFM. Cows that have an induced calving are more at risk of RFM but this procedure should not be routinely undertaken in organic herds.

2. Clinical Endometritis

Endometritis is the inflammation of endometrium and underlying glandular tissue. Clinical endometritis is characterized by the presence of purulent (>50% pus) uterine discharge detectable in the vagina 21 days or more after parturition, or mucopurulent (approximately 50% pus, 50% mucus) discharge detectable in the vagina after 26 days (14).

- Score 0 = clear or translucent
- 1= mucus containing flecks of white or off-white pus
- 2= < 50 mL exudate containing < 50% white or off-white mucopurulent material
- 3= > 50 mL exudate containing purulent material, usually white or yellow, but occasionally sanguineous

3. Clinical Metritis

Metritis is the inflammation of endometrium and underlying glandular tissue and muscular layer. Animals that are not systemically ill, but have an abnormally enlarged uterus and a purulent uterine discharge detectable in the vagina, within 21 days after calving, may be classified as clinical metritis (14).

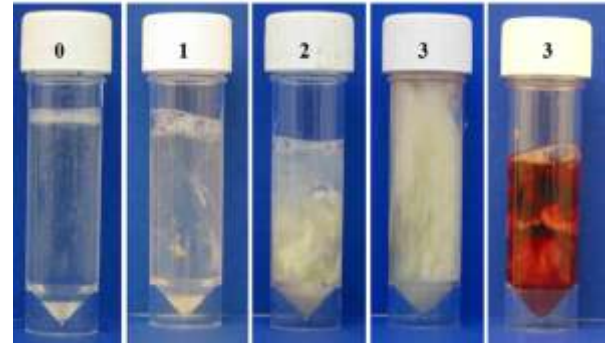


Fig.2. Vaginal Mucus Character (6)

4. Subclinical Endometritis

In the absence of clinical endometritis, a cow with subclinical endometritis is defined by >18% neutrophils in uterine cytology samples collected 21–33 days after calving, or >10% neutrophils at 34–47 days (14).

5. Pyometra

Pyometra is defined as the accumulation of purulent material within the uterine lumen in the presence of a persistent corpus luteum and a closed cervix (14).

6. Metritis & Endometritis

It is important to differentiate animals with metritis from those with endometritis. Metritis is infection of the cavity, lining and deeper layers of the uterus. On the other hand, endometritis is a localised infection of the lining of the uterus, which is inflamed with white pus mixed with mucus discharging from the uterus into the vagina. The deeper layers of the uterus are not affected by endometritis, so the uterus is not much bigger than that of a normal animal. Clearly, metritis is a much more severe disease than endometritis, requiring a different therapeutic approach.

7. Pyometra & Endometritis

Pyometra implies accumulation of pus within the uterine lumen associated with a closed cervix and a corpus luteum. There is often a corpus luteum present in animals with endometritis but the cervix is patent, often with pus discharging from the uterus into the vagina (14). In our experience, clinical endometritis is common whilst pyometra is relatively rare, comprising <5% of clinical cases of uterine disease. Fortunately, treatment with prostaglandin (PG) F2a is equally effective in both cases.

8. Puerperal Metritis

Puerperal metritis is defined as an animal with an abnormally enlarged uterus and a fetid watery red-brown uterine discharge, associated with signs of systemic illness (decreased milk yield, dullness or

other signs of toxæmia) and fever $>39.5^{\circ}\text{C}$, within 21 days after parturition.

9. Septic Metritis

In Septic metritis, the inflammation occurs within 1-10 days postpartum characterized by septicemia, toxemia and pyaemia.

10. Sclerotic Metritis

Sclerotic metritis is the chronic inflammation of uterus characterized by fibrotic changes in uterine wall.

Conclusion

Retained placenta, uterine bacterial infection and uterine disease are common after parturition in cattle and cause considerable infertility. Uterine bacterial infection stimulates a robust immune response but also modulates normal reproductive physiology. Increasing knowledge about the interaction between the environment, uterine infection, immunity and reproduction should lead to improved control and treatment strategies for cattle infertility.

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Summer Mastitis-An Overview

Shruti Bhatt, Shashi Pradhan, Kabita Roy, Mitali Singh and Pooja Dehariya
Dept. of Veterinary Medicine, C V S and A H, NDVSU, Jabalpur, Madhya Pradesh.

Introduction

Mastitis, in simple words is the inflammation of mammary glands and is derived from Greek word “mastos” (mid – 19th century) meaning breast. Animals acquire the disease due to a complex interplay between infectious agents, certain environmental factors and faulty managerial practices (1). Causative organisms can be either bacteria, viruses or fungi leading to significant physical, chemical and bacteriological changes in the milk and glandular tissue of the animal (1).

Etiology

The main causal organism is *Corynebacterium (Actinomyces) pyogenes*, along with others that may either allow the infection to develop or enhance the activity of the primary organism (2). It is most commonly observed during summer months, as is evident by the name, and is usually a disease of non – lactating cows and heifers (3).

Pregnant cows are found to be more susceptible than non – pregnant heifers, especially during the last 6 weeks of gestation (4). It is quite a severe form of mastitis that causes high temperature, udder damage as well as toxemia (2).

C. pyogenes is distributed over a wide geographic range and principally inhabits the mucous membrane of domestic animals (5). This is an agent that can remain in fomites and flies act as mechanical vectors (5).

Transmission

Transmission of infection is caused by the head fly, *Hydrotaea irritans*, that is regarded as the most important vector of summer mastitis. There is annual as well as monthly variation in the incidence of summer mastitis (6).

The disease is reported particularly on sandy soil, less on peat and rare on clay types of soil. This is in accordance with the habitat of *Hydrotaea irritans* (6). A huge number of flies are seen clustering around the affected teat orifice causing considerable irritation to the animal witnessed by frequent kicking.

Pathogenesis

The bacteria invade the teat canal and mammary glands when the udder gets inflamed and multiply and produce toxins causing injury to the milk

secreting tissues. This leads to leukocytosis or increase in somatic cells in milk thus adversely affecting both quantity and quality of milk and milk byproducts (7).

The first line of defense is the teat end. Teat canal is lined with keratin from inside and a sphincter of smooth muscles surround the teat canal from outside keeping the teat canal closed. This prevents the bacteria from entering and milk from escaping the teat (7).

Damage to the internal keratin causes increased susceptibility to bacterial invasion and colonization. At the time of milking, bacteria that are present near the teat opening can gain access to the teat canal, resulting in trauma and damage to the keratin or mucous membranes that lines the teat sinus (8). The teat canal may remain partially open after milking for 1-2 hours and during this span of time the pathogens may enter freely into the teat canal (9).

Clinical Signs

Initially the affected quarter is swollen, hard, painful and hot with a grossly enlarged teat. Flies are attracted to the unpleasant smelly secretions. Development of abscess occur in the affected part of the udder which ripen and break open discharging green/yellow pus, causing destruction of the quarter (1).

Isolation from the group, stiffness and reluctance to walk, lack of grazing giving a gaunt appearance, fetlock and hock joints distension and rapid loss of body condition is observed (5). If treatment is not administered, the animal may abort or even die. Usually the milk production does not come back to normal after recovery, so such animals should be culled (1).

Diagnosis

Diagnosis can be done on the basis of -

- History of flies, season etc.
- Via physical examination of udder and examination of milk
- Putrid odour in milk signifies Corynebacterial infection
- White Side Test – If the contents after mixing gets thick, viscous and appears like strand that comes along with the stick, as it is slowly moved

away from the contents, then it is positive for acute mastitis (1).

- California mastitis test and somatic cell count (10) can also be performed.

Treatment

Treatment aims at saving the life of the animal, saving the pregnancy, to produce a viable calf or at least a cow that can milk to some extent on the remaining quarters (11).

Regular and repeated stripping of the affected quarter should be undertaken to remove the affected material as much as possible (7). However due to the painful and edematous teat / gland animal may resent this by kicking.

This is followed by application of various drugs. This includes parenteral antibiotic injections such as procaine penicillin, sulphonamides or tylosin. Along with this, intramammary antibiotics (penicillin or intramammary erythromycin tubes) is also a must.

Non - steroidal anti-inflammatory drugs are used to counter the systemic effects of bacterial toxins like pain, swelling and fever. Corticosteroids should not be used in pregnant cattle despite the fact that they reduce joint effusions much more effectively. Infected cows and heifers should be put in isolation to prevent the spread of the illness to other animals(12).

Prevention & Control

- Grazing conditions – Preventing the animals to graze in susceptible fields.
- Fly control measures (like synthetic pyrethroids – Deltamethrin, Permethrin) include methods such as impregnated fly tags, pour – on preparations and sprays.
- The most effective means for prevention of summer mastitis is dry cow therapy, both in cows at drying – off and in susceptible pregnant heifers during the summer months (3).
- Sealing of the teat canal, using external teat sealants and micropore / adhesive tapes as physical barriers can help in prevention.
- Isolation of the affected animal from healthy ones is necessary along with proper discarding of infected milk strippings.
- Maintaining hygienic conditions at the farm, regular cleaning and disinfection of milking equipments etc. will ensure good teat end condition.

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Successful Management of Partially Macerated Fetus in Murrah Buffalo: A Case Report

Pramod Kumar, Satish, Bhanu Prakash, Tipu Sultan, Amit Narwal, Lakhan Ram Yadav and Amar Singh
Dept. of Veterinary Gynecology and Obstetrics, CVAS, RAJUVAS, Bikaner, Rajasthan.

Introduction

Fetal maceration is a pre-partum obstetrical disorder in which disintegration of fetus occurs that has died after formation of the fetal bones (beyond 4 months of pregnancy in cattle). Its occurrence is almost in all species but mostly described in cattle. It results due to failure of an aborting fetus to be expelled out, which may occur due to uterine inertia or partially dilated cervix, or the abnormal presentation of a fairly dry fetus which causes it to be retained in the uterus (1). The retention of the dead fetus in-utero followed by the invasion of microbes from the partially dilated cervix leads to putrefaction and autolysis of the fetal mass leaving bones within the uterus (2). It usually follows fetal emphysema and mummification and found to occur usually after 4 months of gestation when fetal bones have been developed normally (3). Usually these cases have a history of intermittent/frequent straining for several days accompanied by fetid reddish grey vulval discharge. This condition is noticed by the owners when foul smelling pus is discharged by a pregnant animal, some times pyrexia and anorexia but rarely, it may be diagnosed during pregnancy examination(4) this condition can also be diagnosed by the history, finding of a piece of bone lodged in the cervix, rectal palpation (free fetal bones palpable in crepitating pus and doughy thick uterine wall) radiography (especially in small animals) and ultrasonography (finding of hyperechogenic scattered bones in an echogenic or non-echogenic fluid with echogenic floating pus) (5, 6, 7). The condition is commonly found in cattle and buffaloes (6,8) but rarely may be encountered in mares (9), small ruminants (10,11,12) and companion animals. This paper aims to report a case of maceration of fetus delivered per vaginally after therapeutic medicinal management in a Murrah buffalo.

History and Clinical Observation

A nine years old buffalo in its third parity presented to clinics of Department of Veterinary Gynaecology and Obstetrics, Teaching Veterinary and Clinical Complex, College of Veterinary Animal Science, Bikaner with the complaint of extended gestation period more than fourteen months. The owner reported that at around 8.5 months pregnancy it developed abdominal discomfort and voided little bloody discharge and had been treated by the local area practitioner. The treatment details were

unknown. The udder was regressed and in the last three days the animal was observed with some foul smelling discharges when straining for urine. The animal was having normal rectal temperature (100.8°F) and plus, respiration as well as normal body condition. The per-vaginal examination of animal with well lubricated gloved hand revealed that cervix was partially dilated. Per-rectal examination revealed palpation of bony part with crepitating sound, uterine wall was exceptionally thickened and doughy in consistency. The placentomes were not palpable and fremitus was absent. During vaginal palpation foul smelling, red-chocolate colored watery fluid came out from the uterus. On the basis of history and clinical examination, this case was diagnosed as fetal maceration.

Treatment and Discussion

It was macerated fetus with incomplete cervical dilation (five fingers). Cervical dilatation therapy consisting of Cloprostenol sodium (Repregna®- Vet Mankind, India) 500 ug IM (intramuscular), Estradiol benzoate (Pregheat®- Virbac, India) 2 mg IM, Calcium magnesium borogluconate (Mifex®- Novartis, India) 450 ml slow IV (intravenous) along with the supportive fluid therapy and Amoxicillin and Sulbactam (Sodamox, Boehinger Ingelheim, India) 4.5 gm IV, Pheniramine maleate (Avilinet®- MSD animal health, India) @1 mg/kg b.wt. IM were given to the animal.

The animal was examined at 12 hours of interval for cervical dilation and after about 28 hours of the treatment and a bony mass consisting fetal head and other parts was found engaged in birth canal with foul smelling fluid draining out. One liter of liquid paraffin was drenched into the uterus with the help of irrigator for manual handling of the case. A few bones of the fetal head came first followed by a partially macerated fetus along with autolyzed fetal membranes by applying force through traction (**Fig.1**). On examination of the fetal mass it was found that some muscle mass was still there but mostly putrefied and autolysed so it was confirmed as a case of the partial fetal maceration. The uterus was lavaged with 3 liters warm normal saline and 1% povidine iodine solution. Then the rectal massage of the uterus was done to remove excess fluid. Symptomatically, the animal was treated with antibiotic, antihistamine, anti-inflammatory drugs and intravenous fluids which were continued for five

days. Oral herbal uterine cleanser (Utrasafe®-Vet Mankind, India) @ 100 ml PO (per orally) BD (twice a day) for 5 days. After about six hours of the removal of macerated fetus, animal started eating grass and drinking water.

Fetal maceration is one of the accidents of pregnancy where fetal death occurs more commonly from mid to late gestation (13). Fetal maceration in a non-descript buffalo has been reported earlier by (14, 15, 16) with successful use of only PGF₂α for cervical dilatation and per vaginal delivery of the macerated fetus. Saurabh *et al.* (2018) (4) used a combination of valethamate bromide and dinoprost (PG analogue) for the successful management of fetal maceration in a buffalo. But in present case history of extended gestation so use combination of therapy cloprostenol sodium and estradiol benzoate for cervical dilation. Bisla *et al.* (2018) (17) used a combination of cloprostenol sodium, estradiol benzoate, valethamate bromide and dexamethasone for the successful management of fetal maceration in a buffalo. Laparohysterotomy to remove the macerated fetus is potentially dangerous and must be considered as a last resort (18). Dutt *et al.* (2018) (8) reported two cases of fetal maceration in crossbred cattle and opted left flank laparotomy as therapeutic measure due to failure of cervical dilatation therapy.

Summary

A Murrah buffalo affected with fetal maceration was successfully treated with by prostaglandin F₂ α and estradiol benzoate combination therapy.

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Fig.1: Partially macerated fetus of buffalo delivered through vaginal route

Management Approaches to Control Parasitic Infection in Livestock

V Singh, P Choudhary and A K Jhirwal

Dept. of Livestock Production Management and Dept. of Veterinary Parasitology,
College of Veterinary and Animal Science, RAJUVAS, Bikaner, Rajasthan.

Abstract

Parasitic zoonotic diseases are prevalent throughout India and are major public health problems in different parts of world. These diseases adversely affect animal's health and threaten profitable animal production, resulting in huge economic losses in our country. Poor diagnosis of zoonotic parasites is due to lack of proper surveillance and the shortage of information about the existence of asymptomatic animal carriers. Emergence of diseases such as neurocysticercosis, cryptosporidiosis, human echinococcosis / hydatidosis and toxoplasmosis in those are immunosuppressed, together with the re-emergence of cutaneous leishmaniosis, poses a serious threat in India and the prevention and control of these parasitic zoonoses, and others, and is a great challenge. In recent decades zoonotic parasites and new emerging diseases have been recorded in both the human and animal populations resulting from ecological changes such as urbanization and industrialization. Although early diagnosis and treatment of zoonotic diseases is of paramount importance, besides it the feasibility of integrating human, Animal, and environmental health also needs to be emphasized so that better management and integrated approaches are becoming major elements of disease control and prevention strategies based on reliable information from systematic surveillance.

Introduction

Parasites are a major threat frequently causing significant economic fall and impacting animal welfare. Infection with gastro-intestinal helminths have destructive effect on animal health [1]. The parasitic population can be controlled by the means of chemicals but due to irregular use and dosing, resistance is created among the parasites. Drug resistant worms are increasing and new products are not available. As a result, there is a need to effectively control worms in animals. No longer can we recommend control programs based on drug treatment solitary that will be satisfactory. It must design a management control as an integrated parasite control program because the numbers of worms, their impact on herd and their level of resistance to drugs will vary from farm to farm. Parasite control programs aim to provide highest parasite control and are looking greater impact at primary level in controlling parasitic infection and infestation also. Based on the probability of incidence of parasitic infection as well as increase in value of milk production, the possible economic gain at state level has been appraised to be Rs. 46 million, Rs. 35 million, and Rs. 14 million only in the north-eastern states of India, depending upon the different strategic treatments [2]. In spite of significant production fall, which may run into millions of rupees, the problem is persisting because of chronic and stealthy nature [3]. Although specific estimates are not available, economic fall from worm parasite infections of cattle can be

significant. But the extent of internal and external parasite problems is usually related to management practices that increase vulnerability, whereas ongoing preventive management practices will minimize losses caused by parasitic infections[4].

Management is necessary to control parasitism; a good management will keep away majority of parasites from livestock. Management practices that were discussed here include grazing management, pasture management and nutritional management.

Grazing Management A successful grazing management needs to be based on appropriate knowledge of the epidemiological conditions for the parasitic infections. The general objective of the methods of grazing management is to limit the contact between susceptible hosts and the parasite infective stages (the third stage larvae or L3) [5]. Animals must be prevented from the ingress to parasite infected water bodies and there should be proper drainage in the animal shed. The newly introduced animals should be quarantined. The bedding material should be allowed to deteriorate along with manure for better control of parasites as it acts as important source of diverse parasitic infections like winter coccidiosis. Mixed species grazing proved effective in certain areas for small ruminants to limit the populations of *H. contortus* [6].

Pasture Management The basic principle of pasture management is restricting the intake of infective stages of pasture-borne parasite infections

in livestock. Many of the grazing lands have been invaded by non-palatable encroaching alien species like Lantana, Eupatorium, Parthenium, Prosopis juliflora, Leucaena etc. severely impacting their productivity by providing low quality fodder, shortage of adequate nutrition, fibrous nature of fodder and deficiency of micronutrients [7]. Clean pasture is a pasture with a nil or very low possibility infection when animals are firstly grazed on it [8]. It can be produced by rotation between a susceptible species and unsusceptible species and the use of land for forage or crops [9].

Nutritional Management Well-nourished animals manage better and overcome infection with parasites promptly than mal-nourished ones [10]. Two factors that play key roles in refuge from nematode infection are protein availability and balanced mineral supply [11]. Types of diet and accessibility of vitamins, minerals and other nutrients are directly related with susceptibility of animal to the parasites. Vitamin A, D and B complexes are requisite in developing the immunity against parasites. Minerals like zinc, iron, cobalt, sodium, potassium, phosphorus, etc. are very requisite for proper functioning of immunological phenomenon going inside the animal's body to thrive functional immunity against the parasites [12]. Iron supplements are also very prime where animals are affected by worms that drain blood, like Haemonchus (worms in the abomasum) and Ancylostoma (intestinal worms) [13].

Conclusion

The general opinion is that better managerial practices combined with integration of more than one compute like best breeding strategies, appropriate biological control measures, scientific exertion of biotechnological tools and techniques and appropriate chemical control measures will help in reducing parasitic burdens to considerable levels which in turn will reduce mortality and production losses to a greater extent.

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Parasitic Zoonotic Diseases in Livestock and Role of Management Practices to Reduce or Control of Their Emergence

P Choudhary, V Singh and A K Jhirwal

Dept. of Livestock Production Management and Dept. of Veterinary Parasitology,
College of V and A S, RAJUVAS, Bikaner, Rajasthan.

Abstract

Zoonotic parasitic infections are playing a significant role in health of humans as well as in livestock animals. During the last decades there have been changes in food preferences and eating habits, there is a growing market for more ready-to-eat fresh and healthy food, which may have created new situations where pathogens may be introduced into food and then to populations [1]. Changes in dietary practice such as the consumption of raw or undercooked meat and fish have been recently implicated as a reason for the emergence of several helminth zoonoses [2]. Therefore, proper coordination between various medical specialties, including veterinary services and other governing bodies, is needed for better and more effective strategic planning to control zoonoses in addition to better managerial practices to hamper the spread or transmission of so called parasitic zoonoses.

Introduction

Most parasitic zoonoses are neglected diseases despite causing a considerable global burden of health hazards in both humans and livestock animals. Worldwide, Neglected Tropical Diseases (NTDs) predominantly affect the poor with more than 40 million people infected and 750 million at risk [3, 4], furthermore, zoonotic neglected diseases make a significant contribution to the entrenchment of poverty in poor rural communities especially who derive income from livestock production [5].

Most parasitic zoonoses are neglected diseases despite causing a considerable global burden of human health and having a substantial financial burden on livestock industries.

Parasitic species which infect and reproduce in food producing (livestock) and companion animals, can be transmitted to human hosts, frequently resulting in disease. These zoonotic parasites can result in human morbidity and in some incidences mortality, thus posing a high risk to public health safety.

How they are transmitted

Zoonoses can be transmitted in various ways:

- through the air
- by eating contaminated meat or produce
- through close contact with an infected animal
- by touching an area or surface that an infected animal touched
- through insect bites like mosquitos or ticks

Livestock and dairy animals are common carrier of many zoonoses. People, who live and work on farms are in close contact with many types of livestock. Outdoor activities, family pets, petting zoos etc are the way of zoonotic diseases transmission.

Here we discussed certain zoonotic diseases that are important in present aspect and required an urgent attention to check their transmission.

Zoonotic Helminthiasis

a) Cysticercosis (*Taenia solium*): Cysticercosis caused by *T. solium* has been the subject of a number of recently published reviews with an Asian focus [6, 7]. There are several reports of porcine cysticercosis in neighboring countries: 3-26% in India [8], 13.7-30% in China [9] and 0.4% in Thailand [10].

b) Trichinellosis: Trichinellosis is a direct zoonosis caused by infection with nematodes of the genus *Trichinella* and is one of the most widely distributed parasitic zoonoses worldwide [11, 12].

Zoonotic Protozoal Diseases

Giardiasis *Giardia* infections are common in humans and domesticated animals, especially livestock, but also occur in pets (dogs, cats), numerous species of wild mammals and birds [13]. In young livestock, *Giardia* infections may adversely impact on production [14]. Giardiasis in domestic ruminants has a negative effect on performance, resulting in decreased rate of weight gain, impaired feed efficiency, lower carcass weight and increased

time to slaughter [15].

Cryptosporidiosis It has been reported worldwide. There are many documents that proved the main reservoir of zoonotic *Cryptosporidium* in livestock, with the potential transmission of *Cryptosporidium parvum*, to humans via contaminated water or through direct contact with livestock [16]. Farm animals play significant role in contributing parasite cysts in large proportion because of their high abundance on farms [17] and can act as the causal agents of human cryptosporidiosis. *Giardia* and *Cryptosporidium* have emerged as important parasites of dairy cattle because of their proven pathogenicity, economic losses and the potential public health significance of zoonotic transmission [14]. Cows can serve as a major host of *Cryptosporidium* worldwide, causing potentially high risk to the human population [18]. Economic loss in the cattle farming industry is due to neonatal diarrhea which causes dehydration, inhibition of normal development and even death [19]. There has also been considerable interest and discussed regarding potential of zoonotic transmission of this pathogen, particularly from livestock. Farm animals are believed to play the most significant role in contributing parasite oocysts in large proportion because of their high abundance on farms [17, 20].

Toxoplasmosis The disease is caused by the obligate intracellular protozoan *Toxoplasma gondii*. Intermediate hosts include sheep, goats, pigs, and humans. Toxoplasmosis in the central nervous system is nowadays seen in acquired immunodeficiency syndrome (AIDS) patients in India [21]. Toxoplasmosis is not only a problem for the unborn child through congenital transmission. It is also a serious problem in immunosuppressed individuals such as HIV patients and transplant patients [22, 23]. In Southeast Asian countries, culinary habits (e.g., eating under cooked meat) and low water quality may be a more significant risk factors for *T. gondii* than cat ownership [24]. In India, recent reports showed that prevalence of *T. gondii* infection was 50% in sheep, 41.2% in goat, 64.4% in cattle and 0.5% in Mithun (*Bos frontalis*) serum samples [25, 26]. Zoonotic parasitic diseases that are spread by mosquitos and ticks are scabies and myiasis.

How to prevent spread or transmission?

Management is an effective way to help prevent the spread of zoonotic diseases in addition to certain integrated approaches. Management practices includes washing of hands properly, practice safe food handling includes washing off all produce before eating it, avoiding bitten or scratched by an animal, to avoid eat, drink, or touch your eyes or mouth while you're handling or in close contact with

animals, using insect repellent or other methods to keep mosquitos, fleas, and ticks away, using gloves during the handling of a sick animal, animal houses should be kept clean, proper disposal of dead animal carcass. Despite these several integrated approaches include biological control, grazing management for the prevention of zoonotic diseases. Livestock should be allowed to graze in a clean and parasitic worm free grasslands by implication of certain grazing techniques such as rotational grazing.

Conclusion

In this review, we have discussed certain parasitic zoonotic diseases, among which, some are showing rising trends such as toxoplasmosis. As various factors favor parasitic zoonoses, there needs to be a permanent solution in terms of certain approaches like grazing management that reduces or alters the transmission of parasites which are responsible for various zoonoses. Therefore, a proper management and other integrated approaches are helpful in prevention of zoonotic diseases. In addition to these practices a proper coordination between veterinary services and other governing bodies is needed for better and more effective strategic plans to control zoonoses.

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Clinical Application of Gene Therapy

Akhilesh Pandey, S K Joshi, M S Thakur, R K Vandre, Vashali Khare and Y Pandey

Dept. of AGB and Dept. of VAN, College of Vety. Sc. and A. H., N.D.V.S.U., Jabalpur, Madhya Pradesh.

Introduction

Gene therapy offers great opportunities to treat or cure disease and alleviate human as well as animal suffering. The majority of the infants that were afflicted by the devastating disease and that were subsequently treated by gene therapy can now essentially lead normal lives. Now this advanced technology is showing great achievement in the treatment of various type of genetic or non-genetic diseases in human or animals. Most importantly, these successes offer new therapeutic options for patients who are currently untreatable. Convincing evidence continues to emerge that gene therapy is effective in patients suffering from other hereditary diseases besides the congenital immune deficiencies (e.g. β -thalassemia, hemophilia and *epidermolysis bullosa*) but also from more common disorders like cancer, neurodegenerative or cardiovascular disorders. Even patients (including animals) suffering from an inborn genetic disease that is not life-threatening but causes blindness can finally start to see by gene therapy. These few selected recent examples of clinical advances in gene therapy clearly indicate that the momentum in this new approach of treatment is building up. In the absence of effective drugs or alternative therapies, gene therapy technology may play a significant role and also best hope for the many patients suffering from various diseases. In the present scenario when there are global economic challenges, it is even more important than ever to find sustainable solutions to treat diseases of high unmet medical needs. The potential for a one-time curative treatment by gene therapy permanently solve continuous therapeutic interventions for the treatment of chronic diseases. So to reduce the economic burden it is absolutely essential to further consolidate and exert sustainable effort towards the gene therapy.

Objective

- To compensate for a missing protein in a genetic disease.
- To inhibit the production of an undesirable protein (by generating an antisense mRNA).
- To express a trophic factor or an anti-inflammatory cytokine.
- To introduce a suicidal gene for the treatment of cancer.

History of Gene Therapy

Genetic engineering that was first presented at the Sixth International Congress of Genetics held in 1932, the concept of genetic correction arose after Avery, MacLeod and McCarty in 1944 suggested that "genes could be transferred within nucleic acids" (1,2).

Clyde E. Keeler in 1947 was probably the first to use the term gene therapy, although the process he was describing (the correction of gene-based deviations in plants and animals) was not envisaged as an effective therapeutic technique to treat genetic diseases in man (3, 4).

Lederberg, 1963 - The first real contribution to the field is attributed to Nobel Prize winner Joshua Lederberg, a pioneer in bacterial genetics and plasmid biology, and a visionary in gene therapy.

Obstacle in the Development of Gene Therapy

Short Life of Treatment The therapeutic genetic material introduced into target cells must remain functional. Naked DNA or certain viruses (e.g. AAVs) may remain episomal and allow sustained expression in stable tissues (e.g. neurons or skeletal muscles).

Toxicity and Inflammatory Responses In the OTCD trial, the patient J. Gelsinger died from fulminant hepatitis four days after beginning treatment with an adenovirus vector. Since then, work using adenovirus vectors has focused on genetically crippled versions of the virus, safer production standards and clinical protocols.

Insertional Mutagenesis This has occurred in clinical trials for X-linked severe combined immunodeficiency (SCID-X1) patients, in which hematopoietic stem cells were transduced with the gamma-chain interleukin 2 (C IL2) receptor gene using a retrovirus, and this led to the development of T-cell leukemia in 5 out of 20 patients. All but one of these children responded well to conventional anti-leukemia treatment.

Significance of Recent Progress of Gene Therapy

Deeper Understanding of the Biology of the Transduced Cells

For instance, for treatment of SCID patients, using myeloablative preparative treatment which was not

included in the early trials.

Improvements in Our Understanding of the Vectors

The issue of insertional mutagenesis has been addressed by replacing retroviruses by lent viral vectors (first evaluated in 2006 in HIV using genetically modified CD4 T-cells, and now in other immunodeficiencies), or by including certain sequences such as the globin locus-control region to direct to specific chromosomal site. Other forms of safer genetic engineering include gene targeting and knocking-out specific genes via engineered nucleases.

Improved Delivery Procedures

The injection of naked DNA is a perfect example of this issue. Clinical trials of intramuscular injection of a naked DNA plasmid have occurred with some success; however, expression has been very low in comparison to other methods of transfection research efforts focusing on improving the efficiency of naked DNA uptake have yielded several novel methods, such as electroporation, sonoporation, and the use of a "gene gun" (ballistic gold, DNA-coated gold particles). The advanced current indications are DNA vaccines, and angiogenic cardiovascular applications, as they involve only short-term expression and local delivery.

Clinical Application of Gene Therapy

a) Oncolytic Adenoviruses for Cancer Gene Therapy

Relatively novel and promising therapeutic platform is virotherapy with oncolytic adenoviruses. Over the last two decades several engineered viral mutants have been evaluated in clinical trials targeting various tumor types and were demonstrated to be safe, with some efficacy. This approach has been applied to numerous viral species, including adenovirus, measles, herpes and poxviruses, to name a few. However, most of the work has focused on adenoviral vectors, especially serotype 5 (Ad5), because of the ease of genetically engineering its small, linear and well characterized 36 kb genome. Additionally, adenoviral mutants can be engineered to not only lyse cancer cells but also express therapeutic transgenes to promote elimination of tumors. Another advantage of adenovirus is the ability to infect both proliferating and non-proliferating tumor cells, an important consideration in many solid tumors with only sub-populations of cells that are actively dividing (e.g. prostate cancer).

Developmental Process

There are several approaches to engineering replication-selective, adenoviral mutants:

deletion of viral genes that are essential for the viral life-cycle to proceed in normal cells but are functionally complemented by the altered gene expression in cancer cells insertion of tumor-/tissue-specific promoters to control expression of early viral genes that drive replication modification of viral tropism to specifically target tumor antigens and infect cancer cells only insertion of microRNA (miRNA) target sequences to suppress expression in normal cells and combining any or all of the approaches above, with or without expression of therapeutic genes e.g. small RNAs, anti-angiogenesis factors.

b) Gene Therapy of the Beta (β) Hemoglobinopathies

Patients of beta hemoglobinopathies who inherit two different mutations may be severely affected whereas heterozygote carriers are generally asymptomatic. In some populations where malaria is endemic, the prevalence of Hb defects can be as high as 40%. Most affected patients in developing countries die before the age of five years, whereas most of the affected children born in high-income countries survive but live with a chronic and severe disorder. β -hemoglobin disorders fall into two large groups of hemoglobin (Hb) mutations: structural variants, in which amino acid changes produce abnormal Hb such as Hb S, E, D or C and β -thalassemia's, in which β -globin chain production is low or nonexistent. Patients who inherit two different mutations may be severely affected whereas heterozygote carriers are generally asymptomatic. In some populations, where malaria is endemic, the prevalence of Hb defects can be as high as 40% (5).

Beta-Thalassemias

There is very a wide range of clinical severity, from the severe transfusion- dependent thalassemia major to the highly variable no transfusion-dependent thalassemia intermedia. They can be caused by a number of mutations at the globin locus resulting in either no globin production or reduced levels of synthesis that lead to imbalanced α :non- α -globin chain ratios. Excess α -chain damages the cell membrane, leading to apoptosis and anemia. In β -thalassemia, the aim is to achieve cellular levels of β -globin expression that are sufficient to bind much of the unpaired α -chains, and this level varies whether there is complete absence of endogenous β -globin expression-thalassemia. Regular transfusions and a strict iron chelation program are warranted to assure patient survival. Without treatment, patients succumb within two years.

Sickle Cell Disease

James Herrick in 1910 reported the first case of a patient with a typical elongated red blood cells (RBCs) and these findings led to the description of sickle cell anemia in 1922. It was due to nucleotide change from GAG to GTG. This valine for glutamic acid substitution at codon 6 is responsible for HbS polymerization, which is the primary molecular event leading to RBC defects, sickling, hemolysis, increased blood viscosity, vaso-occlusion, painful crises, strokes and multi-organ damage.

Gene Therapy of Retina

- a. Strategies for Dominant Mutations in the case of dominant inherited retinal degeneration, the normal protein are not expressed as per desire of the body and one must also suppress expression of the mutant protein to obtain a therapeutic effect. The proposed gene therapy strategies are to remove the endogenous gene expression using molecular tools and express a normal gene copy artificially.
- b. Neuroprotection A large number of trophic factors, e.g. ciliary neurotrophic factor (CNTF), glial-cell-line-derived neurotrophic factor (GDNF) and rod-derived cone viability factor (RdCVF), have demonstrated their ability to protect retinal neurons. So gene therapy represents a priori, an ideal solution for delivery of these factors into the eye.
- c. Ocular Neovascularization is an essential symptom in the onset of blindness for eye disease representing a crucial public health issue such as wet-AMD or diabetic retinopathy. Vascular endothelial growth factor (VEGF) has been identified as a major player in the onset of neovascularization. Consequently, a new class of pharmacological agents, designed to block VEGF action, has been put into use, dramatically improving the management of wet-AMD patients.
- d. Optogenetics is a new emerging area of neuroscience based on targeted expression in neurons of bacterial ion channels whose opening is triggered by light and leads to depolarization of the neuronal cell. These tools have led to significant progress in our understanding of the neural circuits of the CNS.

Gene Therapy for Hemophilia A and B

Hemophilia A and B are congenital bleeding disorders caused by a deficiency of functional clotting factor, FVIII and FIX, respectively. Hemophilia results in an X-linked bleeding diathesis caused by a mutation in the corresponding clotting factor genes. It affects an estimated 400,000 individuals worldwide (according to the World Federation of Hemophilia). Hemophilia A affects nearly 80–85% of the patients, whereas the remaining 15% are afflicted by hemophilia B. As FVIII and FIX are essential cofactors in the blood coagulation cascade, patients suffer from recurrent bleeding and chronic damage to soft tissues, joints and muscles. This progresses towards chronic synovitis, crippling arthropathy and physical disability. The bleeding could also be fatal in the case of intracranial hemorrhage.

Hemophilia as A Target of Gene Therapy

The introduction of a functional FVIII or FIX gene copy into the target cells via gene therapy may provide a cure and eliminate the need for repeated clotting factor infusions.

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Equine Colic

Gurpal Singh, Vandana Sangwan and Arun Anand

Dept. of Veterinary Surgery and Radiology, College of Veterinary Science, GADVASU, Ludhiana, Punjab.

Abstract

India has a large number of equine populations kept in government organizations (patrolling, goods carrying and sports purpose), racecourses and by farmers (as a pleasure horse). Colic is the abdominal pain in equines. Colic is a life threatening condition of equines if not dealt correctly and timely. Along with the type and symptoms, the report describes the importance of treatment in equine colic in the region of Punjab, India.

Introduction

The 2019 livestock census indicates the equine population of India to be 0.5 million (including 0.3 million horses and ponies, 0.1 million mules and 0.1 million donkeys; Source: Livestock census, Department of Animal Husbandry, Dairying & Fisheries, Ministry of Agriculture & Farmers' Welfare, GOI) which had markedly reduced since independence (in 1951; 2.9 million numbers) due to mechanization.

Colic is defined as abdominal pain in equines and is the most common and dreadful condition affecting the horse, as in pain, the equine hurts itself causing multiple wounds. Colic can be broadly classified as gastrointestinal or non-gastrointestinal origin. Non gastrointestinal colic may be due to urolithiasis and disorders of reproductive, nervous, respiratory, or musculoskeletal systems while gastrointestinal colic is due to disorders in intestines or stomach.

Types of Gastrointestinal Colic

Gas Colic It usually results from ingestion of green pastures, infected feeds or grains. Excessive production of volatile acid causes gas distention and flatulence, leading to pain.

Spasmodic Colic it is the intestinal cramps or spasms. It is due to increased borborygmi or intestinal motility may lead to diarrhea and is usually not fatal.

Impactive Colic It is most common type of colic encountered in the local Marwari breed in Punjab. It usually occurs during winter season due to decreased availability of green pasture and water intake (although could occur in any season). Feeding of wheat straw to equines is described as the most important pre-disposing factor for impactive colic.

Sand Colic Grazing of equines on sandy areas could result in sand accumulation in the large intestine or stomach, leading to obstruction / impaction and results in pain. This type of colic is also accompanied with intestinal motility. It is usually reported in equines kept in the sandy areas of Punjab region in India.

Displacement Large intestine of equine may get displaced from its normal anatomical position resulting in either side of dorsal displacement. Left dorsal displacement results in entrapment of colon in nephrosplenic shelf and the equine show bouts of pain.

Volvulus It results from involvement of mesentery between the dorsal and ventral large intestines. The large colon gets rotated on its mesenteric axis due to distention leading to moderate to severe strangulating lesion. Early referral for surgery is the important factor for favorable prognosis in such equines.

Strangulation Rotation or twisting of intestine on its own axis in a small portion results in obstruction of outflow and blood supply resulting in ischemia and endotoxemia. Discoloration of affected intestine is very quick. The condition shows acute pain and need early referral for surgery for favorable prognosis.

Fecalith / Foreign Body Feeding of dry fodder or if the animal is allowed to graze in outdoor mechanized premises may develop colic due to obstruction with a foreign body (ropes/polythene bags) or a fecalith. The pain can be moderate to severe as the equine is not able to pass feces.

Types of Non - Gastrointestinal Colic

Non-gastrointestinal in origin or false colic refers to conditions resulting from other abdominal organs (including liver, spleen, ovaries, kidneys, urinary bladder, gall bladder, uterus etc). This discomfort can be anything from mild pain, causing the horse to paw, bite, and kick at its sides or restlessness, to excruciating pain that causes the horse to hurt itself.

It has been classified into 9 categories (1)

Musculoskeletal Acute laminitis, exertional rhabdomyolysis, atypical myoglobinuria, back pain, exhausted horse syndrome, rupture of the prepubic tendon / abdominal wall hernia, hyperkalemic periodic paralysis.

Urogenital Nephroliths, pyelonephritis, ureteral calculi, cystic calculi, cystitis, bladder neoplasia, urethral calculi, urethral diverticular concretions, uroperitoneumuterine torsion, rupture of the middle uterine and utero-ovarian arteries, uterine rupture, ovarian tumors and hematoma, ovulation pain, retained placenta/metritis, premature foaling, abortion and dystocia, spermatic cordtorsion, orchitis, seminal vasculitis.

Endocrine Pheochromocytoma, hyperlipaemia.

Cardiovascular Myocardial infarction, pericarditis,

aortic-iliac thrombosis, vascular accident (rupture of the aortic ring, aorticopulmonary fistula, hemoabdomen).

Hepatic Acute hepatic necrosis (serum hepatitis), pyrrolizidine alkaloid toxicity, choledocholithiasis, biliary, atresia in foals, hepatic neoplasia.

Respiratory Pleuritis, pleuropneumonia, pulmonary abscessation.

Haemolympathic Splenic tumor, splenic hematoma / rupture, splenic abscess.

Nervous Hypocalcemia, peripheral neuropathy (tetanus, botulism), behavioral problem, equine motor neuron disease, seizures.

Other Pancreatitis, peritonitis, intra-abdominal abscessation, intra-abdominal neoplasia.

Symptoms of colic

Mild colic It includes inappetence, intermittent flank staring and pawing.

Moderate Colic It includes frequent sitting and standing on ground, kicking at the abdomen and sometimes rolling (if brought on soft floor) so may be kept standing by making it walk.

Severe Colic It includes persistent rolling and thrashing with difficult to be kept standing. The equine can sweat profusely and may get itself injured at bony protuberances due to rolling and lying down.

Approach to a colicky equine with gastro-intestinal lesion

1. **Proper Signalment** It is pre-requisite as colic etiologies are specific to different ages, breeds and gender. Neonate foals are more susceptible to meconium impaction, young horses upto 2 years are more likely to get affected with intussusception while geriatric equines are more prone to large colon impaction or fecaliths (2). Arabian equines are more susceptible to enterolithiasis (3). Male stallions are reported to be affected with inguinal hernia while uterine torsion induced colic has been found in mares (2).
2. **Detailed History** It includes; any recent change in diet, deworming history, recent diarrhea/fever/laminitis history, duration from 1st sign of colic and any previous colic history. Recent history of deworming with anthelmintic with higher efficacy towards ascarids such as oral ivermectin has been reported to paralyze ascarids resulting in excessive accumulation of ascarids in small intestines resulting in impaction (4).
3. **Physical Examination** It includes per-rectal examination, intestinal motility / borborygmi and nasogastric intubation. Per-rectal examination can help in diagnosing the large colon lesions of impaction, fecalith, enterolith or any foreign body. Nasogastric intubation with reflux >4 liters can help in concentrating on anterior gastrointestinal affection while the reflux of <4 liters usually associated with the affection of large intestine(2,5).

Hypermotile intestines are commonly found in sand or spasmodic colic. Per vaginal / rectal examination can also help in diagnosing the uterine torsion in advanced pregnant mares, which is the major cause of mild to moderate non-gastrointestinal colic in mares.

4. **Clinical Examination** It includes heart rate, respiration rate, temperature, mucous membrane color and capillary refill time (CRT). Heart rate gets elevated due to colic and more than 70 bpm is usually indicative of cardiovascular deterioration and indicative for surgical exploration. Respiratory rate gets elevated due to pain and pressure on diaphragm associated with abdominal distension. Prolonged CRT of more than 2 secs and dry / congested mucous membranes are also indicators for early referral for surgery.
5. **Hematology and Serum Biochemistry** If available, Hb, PCV, serum lactate, glucose and creatine kinase should be assessed as they are key prognostic indicators. Increased Hb with increased PCV is usually indicative of dehydration / strangulating lesion and excessive fluid therapy should be promptly initiated along with referral for surgery. Serum lactate > 2m mol/L is associated with colic and if it doesn't improve with medicinal therapy, decision for early referral for surgery should be taken. Increased glucose of more than 300mg/dl is also a poor prognostic indicator (6).
6. **Radiography and Ultrasonography** Radiography of mid to caudal ventral abdomen may help in diagnosing sand in colon. Ultrasonography is helpful in diagnosing ileus, nephrosplenic entrapment of left large colon, small intestinal distention and increased peritoneal fluid.

Treatment

Daily / hourly physical and clinical examinations can formulate a decision for medicinal or surgical treatment.

Medicinal treatment usually includes

1. **Analgesic-** Inj Flunixin meglumine @ 1.1mg/kg, IV is the most commonly used NSAID in colicky equines. If the equine is non-responsive to this, α -2 agonists, like inj. xylazine HCl and Opioid analgesic butorphanol can be used alone or in combination and if still non-responsive may be associated with strangulating lesion and require early surgical referral.
2. **Fluid therapy** It is an important part of colic treatment and is usually lacking in field conditions. Both parenteral and oral routes can be used for fluid administration. If the horse shows positive nasogastric reflux, then oral route should not be used. The amount of parenteral fluid requirement includes determination of fluid deficit, maintenance requirement and other losses such as reflux or diarrhea which can be calculated using formula(7):

Fluid deficit (L)= % Dehydration (4%-10%) * body weight (kg) with maintenance= 50 ml/Kg/day.

Total calculated amount of whole day should be administered with half of the calculated fluid administration within first 2 hours while rest amount to be administer throughout the day.

3. **Antibiotics protection against enteric bacteria** Horses are susceptible to endotoxemia. Polymyxin B @ 1000-5000 U/kg bid is widely used to protect equines from endotoxin induced inflammatory reactions (8). If surgery is performed or in infectious diarrhea, antibiotics may be prescribed appropriately.
4. **Laxatives and lubricants** Enteral administration of liquid paraffin or mineral oil in impactive cases can be used as laxatives in order to coat the internal linings of intestine and help in increasing the motility. Dioctyl sulfosuccinate (DSS) @10–50 mg / kg in 2 L water via nasogastric intubation is an effective lubricant but with toxic effects, so should be used with caution (8). Psyllium husk @1 g/kg PO may be given for 7 days to equines diagnosed with sand colic (4, 8).
5. **Deworming therapy** Parasites such as *Strongylus vulgaris* had been reported to cause colic in equines (7, 8). Deworming with antihelminth such as ivermectin or fenbendazole is recommended. Fenbendazole at twice the dose rate for 5 consecutive days is reported to be effective(7, 8).

Prevention

1. Winter months are predisposed to impactive colic due to dry fodder (wheat straw) and cold water resulting in decreased water intake. Adequate availability of green pastures and lukewarm water may help in lowering chances of impactive colic.
2. In certain regions, it is a falsehood trend of feeding wheat straw to equines which is then a major cause of impaction. Wheat straw is only recommended for ruminants and should not be used in equines.
3. Avoid abrupt change in diet schedule.
4. Various dental abnormalities predispose horses to colic. Regular dental checkups and floating of irregular teeth may help to lower the chances of colic.
5. Avoid horses to graze in sandy areas as it may cause sand colic.
6. Proper deworming schedule should be followed twice in a year. Preferable schedule should be alternative oral administration of ivermectin (25ml/100kg body weight) and fenbendazole (10 mg/kg body weight) at a gap of 6 months.
7. Avoid excessive access to fermentable ingredients such as beet pulp or grains as they may predispose to gas colic.
8. Proper exercise is recommended to equines.

Colic in Punjab, India

Punjab is geographically situated in the northern region of India and experiences all type of seasons including

extreme winter, extreme summer and high humidity. The equine surgery section at Guru Angad Dev Veterinary and Animal Sciences University, Ludhiana, Punjab, India has been involved in up-gradation of equine colic surgery from the last several years (9, 10, 11). The majority of cases presented here are of Marwari breed followed by Thoroughbred and mules. Marwari breed is kept by the farmers for pleasure and usually report impactive / non-strangulating type of colic, where wheat straw is the pre-disposing factor. The falsehood decision of feeding wheat straw to equines needs to be changed to avoid impactive colic. Equines must be fed a proper mixture of horse grams, oats, wheat bran, barley / maize and green fodder as per the season and physiological requirement of the equine. Early referral for surgery in non-responsive colicky is the key to favorable prognosis.

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Contagious Caprine Pleuro Pneumonia (CCPP): A Transboundary Disease in Small Ruminants

S Parthiban, N Sweetline Anne, G Balakrishnan and J Johnson Rajeswar

Dept. of Veterinary Microbiology, Veterinary College and Research Institute, TANUVAS, Tirunelveli, Tamil Nadu.

Abstract

Sheep and goat rearing is the root base of rural economy in India. It plays an important role in the upliftment of the economic status of rural as well as peri-urban people. Government of India also encourages sheep and goat farming towards achieving food security. Contagious Caprine Pleuro Pneumonia (CCPP) is a devastating disease of goats caused by *Mycoplasma capricolum* subsp. *Capripneumoniae* (*Mccp*) a member of the "*M. mycoides* cluster. CCPP is transmitted by close direct contact between animals, and thus movement of infected goats and congregation of animals is the key factor in its spread and serve as a potential threat to many disease-free countries. As a result, it is classified as a List B disease by the OIE. CCPP infected animals should be differentiated from several diseases presenting similar respiratory signs in small ruminants, such as Peste des Petits Ruminants, Sheep pox and Pasteurellosis. Since *Mycoplasma* is a fastidious organism it is very difficult to isolate on culture media *in vitro*. Consequently, isolation trials are usually failed and moreover negative result of cultivation of *Mycoplasma* doesn't indicate the absence of infection. To overcome on these constraints an accurate and reliable diagnostic technique is essentially required for rapid detection and confirmation. DNA amplification techniques provide an accurate identification of CCPP causative agent directly to clinical material, such as lung and pleural fluid.

Introduction

Goat and sheep farming is one of the important agro-based activities in India and plays an important role in income generation and improving the household nutrition. Sheep and goats are a potential source of meat, milk, fibre, hide, manure for landless rural small and marginal farmers and provides dependable source of income to 40 per cent of rural population below the poverty line in India. Over 94 per cent of the goat population of the world is distributed in the continents of Asia and Africa. According to the 20th livestock census, 2019; India possesses 148.8 million goats and 74.26 million sheep. They contribute a significant level of income to the total national economy through various products and byproducts. Pneumonia has been noticed as one of the most frequently encountered condition and is responsible for around 28.7 per cent mortality. Among the important sheep and goat diseases, mycoplasmal infections result in significant losses [1].

Etiology

The most important mycoplasmas of small ruminants are the pathogens of the *Mycoplasma mycoides* cluster [2]. The cluster is composed of six genetically and immunologically similar *Mycoplasma* species: *Mycoplasma mycoides* subsp. *mycoides large colony* (*Mmm LC*), *Mycoplasma mycoides* subsp. *mycoides small colony* (*Mmm SC*), *Mycoplasma capricolum* subsp. *Capricolum* (*Mcc*), *Mycoplasma capricolum* subsp.

capripneumoniae, *Mycoplasma mycoides* subsp. *capri* and *Mycoplasma bovine* serogroup 7. The most important of these diseases are Contagious Caprine Pleuro Pneumonia (CCPP) is caused by *Mycoplasma capricolum* subsp. *Capripneumoniae* (*Mccp*) (previously *Mycoplasma biotype F38*) designated by the Office of International Epizootics as List B diseases because of their economic impact on livestock. Mastitis, arthritis, pleurisy, pneumonia, and keratoconjunctivitis may also result from infection. The direct losses of the disease result from its high mortality, reduced milk production and meat yield, cost of treatment, control, disease diagnosis and surveillance. In addition to these, there are indirect losses due to the imposition of trade restrictions. *Contagious agalactia* also contributes to significant losses in small ruminants [3]. In India, the first record on the existence of CCPP was made by Steel in the year 1889 from Khandesh, Bombay. The disease has been reported in many states of the country including Andhra Pradesh, Assam, Goa, Gujarat, Himachal Pradesh, Kerala, Maharashtra, Manipur, Rajasthan, Tamil Nadu and Meghalaya [4]. Although the significance of mycoplasmosis is well known, very little work has been carried out to establish its prevalence in India which has more small ruminant population.

Epidemiology

CCPP is reported to affect only goats although there has been evidence of the infection in sheep and cattle [5]. Clinical disease and sero-positivity have

been reported in sheep in contact with affected goats, but the disease is not zoonotic. Isolation of *M. capricolum subsp. capripneumoniae* has been reported from healthy sheep that had been in contact with goat herds affected by CCPP. The isolation of *M. capricolum subsp. capripneumoniae* from cattle with mastitis has also been reported [6]. The disease has also been shown recently to affect wild ruminants such as the wild goats (*Capra aegagru*), Nubian Ibex (*Capra ibex Nubian*) and Laristan Mouflon (*Ovis orientalis laristanica*) and Gerenuk (*Litocranius walleri*). The disease caused by Mccp is readily contagious and fatal to susceptible goats of all ages and both sexes, rarely affects sheep and does not affect cattle.

Disease Transmission

Susceptible goats get infected through inhalation of contaminated droplets from infected goats. For the transmission to take place close contact between infected and naive animals is of essence due to the high sensitivity of the mycoplasma to the external environment. Stress factors occasioned by malnutrition and movement over long distances predispose the animals to the disease. The extensive and traditional livestock production system practiced in most African countries enhances the spread of the disease as animals congregate during grazing and watering. Breed and sex do not appear to affect the epidemiology of CCPP, but age is an important factor. All age groups

are susceptible but mortality is higher among the young. Chronically infected goats are responsible for perpetuation of the disease in a herd [5, 7] Concurrent infections with viral diseases such as *Peste des Petits Ruminants* (PPR) and Capripox predispose lung tissue to invasion by mycoplasmas. The role of sheep as reservoirs of infection is not very clear [8].

Pathogenesis

The mechanisms of the pathogenesis of CCPP are exactly unknown, but it is clear that most of its species adopted complex strategies to enter the host tissues. In its pathogenesis, the prime and key factor of the organism is to attach and adhere to the polymorph nucleated cells and consequently phagocytic activity of these cells is completely altered. CCPP is typical example of multi-factorial diseases, where factors such as intercurrent infections, crowding, inclement climatic conditions, age, genetic constitution, and stress from transportation, handling, and experimentation are important determinants of the final outcome of infection. An essential part of the pathogenesis of the disease is thrombosis in the pulmonary vessels, probably prior to the development of pneumonic lesions. Bronchitis, bronchiolitis, and alveolitis with predominantly neutrophils and mono nuclear cellular response constitute the very early inflammation in Mycoplasma pneumonia. There are various substances produced by the Mollicutes,

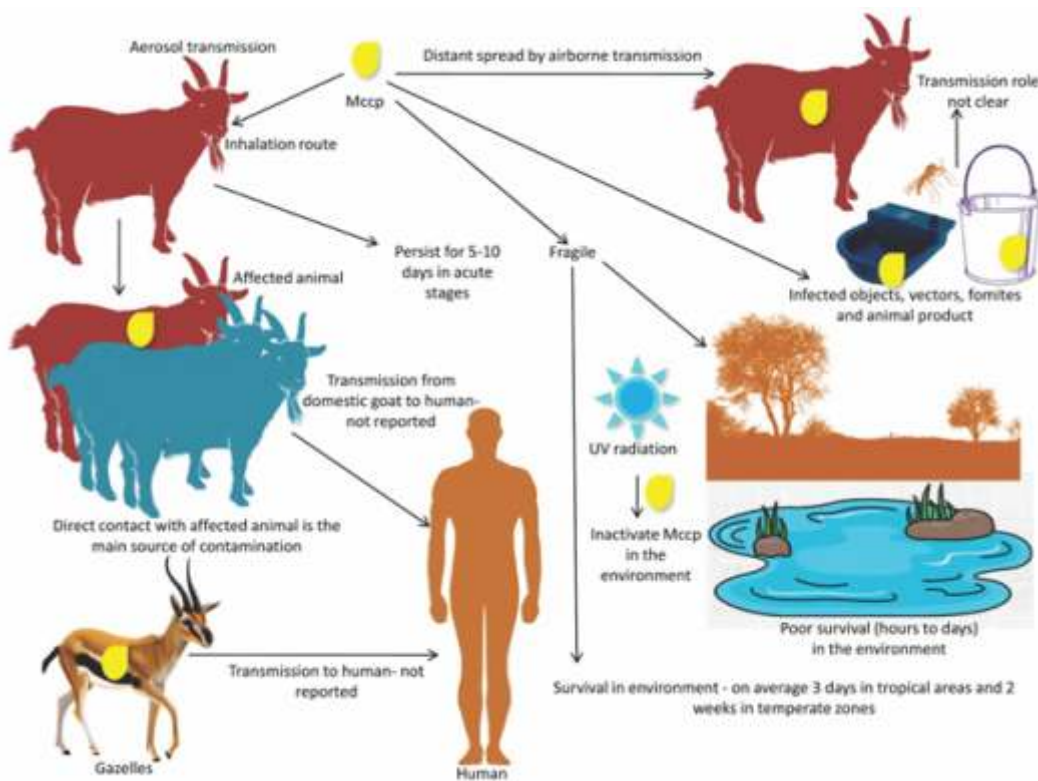


Figure depicts Transmission of *Mycoplasma capricolum subsp. Capripneumoniae* (Adopted from 12).

which are potentially important in disease pathogenesis. Peroxide and super-oxide production may be important in disruption of host cell integrity [9].

Contagious Caprine Pleuro Pneumonia (CCPP) is a highly infectious and serious respiratory disease of goats clinically characterized by coughing, respiratory distress and extremely high morbidity and mortality rates [5]. The disease is included in the list of notifiable disease by World Organization of Animal Health (OIE) because of remarkably high morbidity and mortality rates causing significant socio-economic impact once declared in a country. The gross pathological lesions are localized exclusively to lung and pleura and are often unilateral. There is fibrinous pleuropneumonia with massive lung hepatization and pleurisy. Affected lungs can be totally hepatized with accumulation of straw-coloured pleural fluid. A lung section shows a fine granular texture with various colours, but usually without any thickening of the interlobular septa. There is often an abundant pleural exudate and conspicuous pleuritis. The pleural exudates can solidify and form a gelatinous covering sometimes over the whole lung. In acute cases, the pleural cavity contains an excess of straw-coloured fluid with fibrin flocculations. In chronic cases there is a black discolouration of the lung tissue and sequestration of the necrotic lung areas. Adhesions between the lung and the pleura are very common and often very thick.

Clinical Signs

CCPP causes severe respiratory disease in affected goats initially characterized by a high fever (41-43°C), high morbidity and mortality rates affecting all ages and both sexes. Other clinical signs that may be noted include lethargy, anorexia and abortions in pregnant goats. After 2-3 days of high fever, the respiratory symptoms become more prominent. This may include accelerated and laboured breathing with painful grunting, frequent, violent and productive cough. In the terminal stages, the animals are unable to move. They stand with their front legs wide apart, the neck is stiff and extended downward, stringy saliva continuously drips from their mouth and mucopurulent discharge obstruct their nostrils. The tongue may protrude and bleat distressingly. Death generally occurs within 7 to 10 days after the onset of the signs, but can be as fast as 2 days. Some animals may have long term infection whereby symptoms are milder but dominated by intermittent cough, nasal discharge and debilitation [3].

Diagnosis

The diagnosis of outbreaks of respiratory disease in goats, and of *CCPP*, is complicated, especially

where it is enzootic. It must be differentiated from other similar clinico-pathological syndromes such as *peste des petits ruminants*, to which sheep are also susceptible; pasteurellosis, which can be differentiated on the basis of distribution of gross lung lesions; and *contagious agalactia* syndrome [5, 10]. It is not possible to establish a *CCPP* infection in the field based on clinical signs or on postmortem examinations alone. The diagnosis is more complicated where it is endemic.

However, high mortality and typical early thoracic lesions in goats during the acute phase are highly indicative of *M. capricolum subsp capripneumoniae* infection. All cases of caprine mycoplasmosis need additional laboratory tests to establish a presumptive diagnosis. It may be difficult to distinguish *CCPP* from an infection by *M. mycoides subsp mycoides LC* or *M. mycoides subsp. mycoides SC*, which have a pulmonary location. In the case of *M. mycoides subsp. mycoides LC* infection, thickening of the interlobular septa may be evident. These lesions are similar to those observed in the case of CBPP. Sometimes the thickening is absent or inconspicuous and hence laboratory confirmation is needed [3]. The surface of each lung tissue with the lesions is sterilized with a hot spatula and deep tissue is minced with sterilized scissors. One gram of minced tissue is mixed with 9 ml of modified pleuropneumonia-like organisms (PPLO) medium and stored for further analysis.

Isolation and Identification

Even though lot of serological and biochemical tests are available isolation of *Mccp* remains the confirmatory test. Samples to be taken from live animals are broncho-alveolar washings or pleural fluid obtained by puncture. Samples to be taken at necropsy are lung lesions, lymph nodes, and pleural fluid. For cultivation of the pathogen, the tissues are grounded in buffered solution and inoculated into the PPLO broth and on PPLO agar media or modified Hay Flick culture media following standard procedures. The inoculated media were incubated in a 5% CO₂ humidified incubator at 37°C. The broths were examined daily for signs of growth, such as change of pH indicated by a color change or turbidity change in the media. The plates were examined after 2-3 days under 100X magnification for the presence of a typical "fried egg" appearance of mycoplasma colonies [11]. Growth of *MCCP* requires rich media containing high percentages of serum. Isolation is hampered by the slow growth of *MCCP*, up to 15 days, and the presence of other mycoplasma species such as *M. ovipneumoniae*. In broth, growth is visible within 4–15 days but turbidity is always very faint. *MCCP* sometimes produces 'comets' in unshaken liquid cultures.

Serodiagnostic Assays

Latex agglutination test (LAT), Enzyme Linked Immunosorbent Assay (ELISA) and Complement Fixation Test (CFT) are routinely used serodiagnostic tests with current forms of improved LAT being suitable for application in the field with comparable sensitivity and specificity to ELISA-based tests and better than CFT, Counter Immune Electrophoresis (CIE) and Passive Hemagglutination Test (PHT). However, on laboratory platforms ELISA - based tests are usually more sensitive and specific than other tests [12].

Molecular techniques for the rapid and specific identification

Molecular techniques can be used for the rapid and specific identification. Two polymerase chain reaction (PCR) assays for the specific identification of Mccp have been published. The first one is based on the amplification of the 16S rRNA gene of the mycoides cluster [13]. The PCR product is then analyzed by restriction enzyme cleavage for the identification of the Mccp amplicon. The second one is based on a specific A DNA fragment of 7109 bp containing genes coding for the arginine deiminase pathway (ADI) was chosen as target and amplified with the primer sequences

(Mccp-spe-F/R) Mccp-spe-Forward: 5'-ATC-ATT-TTT-AAT-CCC-TTC-AAG-3'

Mccp-spe-Reverse: 5'-TAC-TAT-GAG-TAA-TTA-TAA-TAT-ATG-CAA-3' and sequenced [14].

Differential Diagnosis

There are other diseases with similar clinico-pathological syndromes which may be confused with CCPP. These include:

- *Peste des Petits Ruminants (PPR)* - to which sheep are also susceptible
- Pasteurellosis - which can be differentiated on the basis of distribution of gross lung lesions
- Mastitis, arthritis, keratitis, pneumonia and septicaemia syndrome (*contagious agalactia syndrome*) - the pneumonia is accompanied by prominent lesions in other organs and it does not affect sheep or cattle.

Treatment and Control

Antibiotics can be helpful in the treatment of CCPP; erythromycin, tylosin, tetracycline, or streptomycin is recommended but their success depends on early intervention and treatment. In countries that are newly infected, trade and movement restrictions and the slaughter of infected animals is recommended. Quarantine of affected flocks is desirable. Vaccines are available in some countries, and good to excellent protection has

been reported. Treatment with tylosin at 10 mg / kg / day, IM, for 3 days, has been effective, as has oxytetracycline (15 mg / kg). In most cases of treatment supporting therapy (Anti-inflammatory, vitamin supplementation etc.) is very much important it should be always coupled with therapy [5]. Taking account of the above epidemiological and other factors, three broad strategies for the control and eradication of CCPP will be applied in the event of an incursion, namely:

- Reduction of the number of infected and potentially infected animals in goat populations through stamping-out campaigns.
- Reduction of the rate of direct contact between infected and susceptible goats through surveillance programmes, zonation, quarantine and strict movement controls.
- Reduction of the number of susceptible animals in target populations through comprehensive vaccination campaigns.

Eradication

- Quarantine of infected flocks is desirable, and on-site testing and slaughter is sometimes necessary to control the spread of CCPP. The quarantine must be strictly imposed due to the high communicability of the disease. Sodium hypochlorite (bleach) containing a stock concentration of 5.25% available chlorine is effective for disinfection.
- Sufficient regulatory restrictions should be maintained to prevent introduction of CCPP into apparently healthy animals. Serologic testing of susceptible animals for importation is a recommended safeguard.
- Successful control of the spread of CCPP rests on removing susceptible animals from any possible contact with CCPP-infected animals, whether they are clinically affected or subclinical carriers only. On-farm quarantine of suspicious and contact animals would be very advantageous in stemming the spread of the disease. In an outbreak situation, testing, slaughter, and quarantine would be the methods of choice.

Conclusion

Responding to huge marketing signals, the small ruminant production system in India has been slowly moving from extensive to intensive system of management in densely populated herd to achieve high levels of economic efficiency. In addition to this, due to the impact of climate change, the animals face lots of stress, lowering of body defense mechanisms, which lead to emergence and reemergence of economically important

diseases from time to time in various ways. The emergence of a new disease may present considerable problems of diagnosis, surveillance and control. Contagious Caprine pleuropneumonia (CCPP) is one of the reemerging devastating disease of goats and should be differentiated from a number of diseases presenting similar respiratory signs in small ruminants which will help in selecting suitable therapeutic and control measures for CCPP and as well as for other diseases.

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Management of Postpartum Uterine Prolapse in A Non - descript Doe

C Pugazharasi, T Sarath, N Arunmozhi and R Suresh Kumar

Dept. of Veterinary Gynaecology and Obstetrics and Dept. of Clinics, MVC, TANUVAS, Chennai, Tamil Nadu.

Abstract

A post-partum total uterine prolapse in an old non-descript pluriparous goat was presented to large animal obstetrics outpatient ward, Madras Veterinary College Teaching Hospital. On examination, the prolapsed mass was soiled, inflamed, congested, edematous and fetal membranes intact. Epidural anaesthesia was achieved using 2 ml of 2% lignocaine solution at the first coccygeal space. The prolapsed uterus was repositioned and vulval retention suture pattern was also applied. Oxytocin, calcium borogluconate, broad-spectrum antibiotics and fluid therapy were administered and the animal had an uneventful recovery.

Introduction

Post-partum Uterine prolapse has been recorded in all animal species. It is most common in the cow and ewe, less common in the doe and rare in the mare (1). It is simply an eversion of the uterus which turns inside out as it passes through the vagina. Prolapse of the uterus generally occurs immediately after or a few hours of parturition when the cervix is open and the uterus lacks tone (2). The etiology of uterine prolapse is unknown. But many factors have been associated with these includes conditions such as poor uterine tone, increased straining caused by pain or discomfort after parturition, excessive traction at assisted parturition, the weight of retained fetal membranes, conditions that increased intra-abdominal pressure including tympany and excessive estrogen content in the feed (2, 3).

Prolapse that occur more than 24 hours post-partum is extremely rare and is complicated by partial closure of the cervix, making replacement difficult or even impossible (4). In the period immediately after prolapsed occurs the tissues appear almost normal, but within a few hours they become enlarged and edematous. Some animals will develop hypovolaemic shock secondary to internal blood loss, laceration of the prolapsed organ or incarceration of abdominal viscera. The prolapse is visible as a large mass protruding from the vulva, often hanging down below the animal's hock. The placenta may likely be retained during this period. Success of treatment depends on the type of case, the duration of the case, the degree of damage and contamination.

Case Details

A female doe weighing 22 kg body weight with the history of 2 kidding was presented to the Large Animal Obstetrics unit of Madras Veterinary College

Teaching Hospital with a protruding mass from the vulva. The doe kidded two live fetuses (1 female and 1 male) before 24 hours. A thorough physical examination was carried out and the vital parameters were within normal limits. On examination, the prolapsed mass was soiled, inflamed, congested, edematous and fetal membranes intact. The goat was apparently healthy and in standing position. Prolapsed mass was hanging out of vulva up to the level of hock. The case was diagnosed as **post parturient total uterine prolapse**.



Treatment and Discussion

The goat was administered 2 ml of 2% lignocaine at first coccygeal space to attain epidural anaesthesia. The prolapsed uterus was gently washed with luke warm water and the foetal membranes were separated manually from the maternal caruncles. The urine was relieved from bladder by catheter. The mass was washed with saturated salt solution to reduce the edema. The cetrimide cream was liberally applied over the mass. Portion of the animal was elevated by folding the hind limbs at the level of



hock joint. The vulval lips were pulled apart and the everted mass was reduced using the palm of both the hands. The proper replacement was ensured by introducing the hand through the cervix and vulval retention suture was applied (5).

Further, the animal was treated with 200ml DNS IV, 400mg of Amoxicillin+ Cloxacillin combination IV, 2 ml of Meloxicam IM, 2 ml of Chlorpheniramine maleate IM, 10 IU of Oxytocin IM and 2 ml of B-complex IM for 3 days. The vulval retention suture was removed after 5 days and animal had an uneventful recovery. Prolapse of the uterus normally occurs during the third stage of labour at a time when the fetus has been expelled and the fetal cotyledons has separated from the maternal caruncles (4). In small animals, complete prolapse of both uterine horns is usual (6, 7). In some instances, the bladder and intestines may prolapse into the everted uterus. These require careful replacement before the uterus is replaced. But in the present case prolapse of uterus was observed after forceful delivery of kid. The goal in the treatment of uterine prolapse is repositioning of the organ followed by a method to keep it in a retained position. Once the uterus is in its normal position, Oxytocin 10 I.U. intramuscularly should be administered to increase uterine tone.

A caudal epidural anaesthesia is essential before replacement of a uterine prolapse as it decreases straining and desensitizes the perineum (2). An injectable broad-spectrum antibiotic once administered for three to five days after replacement of the prolapsed will prevent secondary bacterial infection (8, 9). The prognosis depends on the amount of injury and contamination of the uterus. The prognosis is favorable when a clean, minimally traumatized uterus is promptly replaced.



Conclusion

In the present case, the prolapsed mass was soiled, congested, edematous and fetal membranes intact. So, after removing fetal membranes prolapsed mass cleaned properly and repositioned. Finally, vulval retention suture were applied to prevent recurrence.

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Marathwadi Buffalo

Alok K Wankar, S N Rindhe, B L Kumawat and P M Kekan

Dept. of Veterinary Physiology, Livestock Product Technology and Dept. of Animal Reproduction,
C V A & S, Parbhani, Maharashtra.

The Marathwadi buffalo is an ancient buffalo breed of marathwada region, in the river valley of the Purna, Dudhna and North banks of Godavari. It is distributed in the districts of Beed, Parbhani, Jalna, parts of Nanded, Latur and Osmanabad of Marathwada region. In the region it is also recognised by Ellichpuri and Dudhana Thadi names.

These animals are of medium built and very distinct from heavier western and northern buffalo breeds [1]. The buffaloes are mainly reared for their milk, for drought and also utilized for local transportation. It is very well adapted to native climate and resilient to adverse conditions, feed scarcity and droughts [2].

Marathwadi buffaloes are primarily maintained by small marginal farmers and landless labourers and not reared by any specific community. A semi-intensive management system is commonly adopted along with cattle. Grazing along with little sorghum, paddy straw, sugarcane leaves/tops, grasses and other farm waste mainly constitute the feed.

The population is constituted of both pure breed animals and grade up with Murrah breed by artificial insemination [3].

Color ranges from grayish to jet black (85.54-97.70 %) and some animals have white markings on their forehead and leg regions and tail switch (14.46%).

The animals are of medium built, a broad forehead and short neck [3, 4]. Horns are mostly round or flat, comparatively medium in length, slightly curved straight.

Horns run parallel to neck and never extend beyond the shoulder blade/hump with average length of 43-47 cm.

Tail length is about 65-66 cm reaching up to hock region and average body weight is 320-400 kg.

Males are slightly heavier as compared to females, with average height of 133 cm, total body length and heart girth of 187.1 cm.

While, females measure 124.3 cm high, with body length of 128.5 cm and heart girth of 181.6. Females are average but consistent milkers, giving 4-8 kg milk/day. Average lactation length is 300 days, total milk yield per lactation is 1118 kg. Females calve (parturition) for the 1st time at 52.3 months of age and calving interval is 501 days [5].

Marathwadi buffalo is only 0.35% of the total buffalo population in India. Current Marathwadi buffalo population is 376595, out of which pure animals are 278502 and graded are 98093 There is a steady

decline in the breed population from a 425000 in 1997 to 278502 in 2013[5,6].

The animal district wise in Marathwada region is as follows (7)

Integrated modern animal husbandry, supported by strong scientific advancements are essential to conserve the ancient indigenous breed of livestock, like marathwadi buffalo.

District	Population (in thousands)
Beed	24.6
Parbhani	10.5
Jalna	2.1
Parts of Nanded	13.9
Latur	83.6
Osmanabad	31.8

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Importance of Yak to Himalayan Culture: A Review

Rashmi Choudhary, Danveer Singh Yadav, Jitendra Yadav, M S Jamra and Mamta Singh
College of veterinary Science and A. H., MHOW, NDVSU, Jabalpur, Madhya Pradesh.

Introduction

Yak is also called as “**Snow Camel**”. Yak is in the economy of the so-called “roof of the world”, the Qinghai-Tibetan Plateau. A number of researchers (1, 2) have discussed the importance of yak for pastoralists in the Himalayas. Yak (*Bos grunniens*) is a long-haired, short-legged ox like mammal also called “Hairy cows” and was originally domesticated in Tibet thousands of years ago and has supplied the indigenous people of these mountainous regions with most of their daily needs including meat, milk, butter, cheese, wool, fiber, leather, fuel, and packing / trekking / travel requirements. This versatile animal is an integral part of the lives of the Tibetan natives and substantially adds to the renowned health and longevity of these people. Yaks are heavily fringed with long black hairs over a shorter blackish or brown undercoat that can keep them warm. Yaks can thrive in high elevation cold winters up to - 40 °C. They prefer to eat snow rather than drink water. They prefer to use shade shelters with open sides, or the shelter of trees. It is not known with certainty that when yaks were domesticated, although it is likely that they were first bred as beasts of burden for the caravans of Himalayan trade routes. Yaks’ lung capacity is about three times more than that of cattle, and they have more number of smaller red blood cells which improves the blood’s ability to transport oxygen. Yaks seasonally aggregate into larger groups and breeding occurs in September–October.

Calves are born about nine months later and nursed for a full year. The mother breeds again in the fall after the calf has been weaned. The wild Yak (*bos mutus*) is found in the Himalayan Mountains of Tibet and surroundings at elevations of 14,000 feet and cannot live below 12,000 feet elevation for any length of time. But the wild Yak bulls interbreed with various cattle breeds surrounding their native Himalayan Mountain terrain. These cross calf heifers crossed back several times to the wild Yak. These multi generational crosses became the domesticated Yak (*bos grunniens*). In the family *Bovidae*, the yak belongs to the same genus as cattle as well as the banteng, gaur, and kouprey of Southeast Asia.

Personality and Behaviour

The Tibetan Yak has a truly striking exotic appearance like handle bar horns, buffalo humped shoulders, horse like tail, and a long hairy skirt reaching almost to the ground, they are very pleasing to the eye. Yak babies are agile, athletic, playful, and leap and run like excited horses with their tails held high over their backs. Yaks do not bellow, bawl, or moo. Instead they communicate in quiet grunts, snorts and head shakes. Yaks are

extremely intelligent, curious, independent, and quiet animals that make them a pleasure to raise. If raised as a pasture pet, they will respond to you as a pet, always seeking attention and responding in turn with appreciation and with real personality. If raised on a ranch with minimum interaction, they quickly recognize and accept their care takers as friendly and are not aggressive, as long as the caretaker knows how to communicate with their Yaks, and establish their leadership position in the Yak’s pecking order.

Adaptation of Yak to High Altitude

Yak survives if mud mean temperature is below 5 °C and average in hottest month is not above 13 °C. This is ideal temperature range for optimum activity of yak for growth and production beyond this yak are under physiological stress. During long winter season when little feeds are available. It may be said that some ruminant species may help yak to sustain long winter months by producing additional bacterial mass. Another important finding of blood haemoglobin (Hb) revealed that fetal Hb in yak continue to persist in total hemoglobin make up the adult to extent of 42% even beyond 10 years of age. In other *bovidae*, it disappears in early stage. The fetal Hb has greater affinity for oxygen and might be considered as adaptive response in high altitudes where oxygen pressure is very low.

Crossbreeding

Yak can crossbreed with any cattle breed. The offspring females are extremely fertile and the males are sterile. Calving occurs easily and offspring born are having low birth weights (40-50 pounds). They got natural protection from the cold. Calves are more hardy at five days than their mother, they are naturally disease resistant and all yield high production rates. The half Yak bull is sterile, but castration is still necessary. Hybrid vigor growth rates yields an early optimum sized feeder earlier than with full blood yaks, while nearly maintaining the feed efficiency and meat characteristics of the Yak.

Yak and local cow are limited in Sikkim

Yak X Local Cow = Joe

Male Joe is sterile, female joe yield more milk (3-4 liters) is hardier and tolerate heat at lower temperature better than pure yak.

Female Joe X Yak = Kukuya

Female Joe X Cow Bull = Tey

Size, Growth and Maturity

Adult Yak cows range in weight from 600 to 700 pounds and stand 4.5 feet at the shoulders, while Yak bulls range from 1200 to 1400 pounds and stand 5.5 feet at

the shoulders. Full size is achieved in six to eight years. Yak heifers conceive at eighteen to twenty-four months of age and calving occurs at two and one-half years of age. Gestation period is 8.5 months. Calving of the twenty-five to thirty-five pound babies appears effortless. Scours are extremely rare, and only occur in extremely wet and muddy conditions. Yak udders are very small, yielding low quantities of extremely rich milk. Newborn babies are up and running in minutes, grow rapidly, and are exceptionally disease resistant and cold hardy due to their wool coat. Yak bulls are considered breeders at 3 to 4 years of age. Yaks breed and calve far longer than cattle since Yaks live 20-25 years. Your breeding animal replacement costs will drop 50% to 75%. Yak bulls must be raised with cattle if they are to become cattle breeders.

Products Obtained

Meat Yak's meat is very juicy, delicate, flavourful, and healthy. It is 95% fat-free; its delicious flavour comes from its unique distribution of fatty acid percentages. Yak meat is extremely low in palmitic acid that is bad for our health (30% less than beef as a percentage of fats and 120% less than beef as a percentage of meat.) Yak meat is also much lower in calories, saturated fats, cholesterol, and triglycerides. Simultaneously, Yak meat is much higher in stearic and oleic acids that are good for us (35% higher than beef as a percentage of fats.) Yak meat is also higher in protein and solids (less water) than beef. The meat from this hardy breed may be the healthiest even than skinless chicken, buffalo, elk, or any other meat. Yak is even lower in fat than salmon. All this is accomplished on a grass/forage diet alone, with no grain, hormone, steroid, or antibiotic feed supplements.

Wool and Hair Production Yak wool is comparable to angora or kashmere in its superiority and feel. Yaks will give an average of one pound of wool per year. Yak guard hairs are almost identical in texture to human hair and are used for wig production. Two types of wool are found in yak, coarse outer hair and the inner soft hair coat called Khullu in Bhutia language. During shearing Yak gives more coarse hair of about 2 kg and 1/2 kg of fine hair. The hair of Yak is highly water proof with great tensile strength. Fine wool or Khullu is used to make garments like mufflers, sweaters and blankets. The fine wool woven to make garments resists rain and cold winter. Coarse hair is used to make tents, caps, blankets, hand bags, door mats and hand woven carpets.

Milk It is believed that milk yield is higher in hybrids than in pure yak. Pheno refers to yak type in Bhutia language gives less milk compared to Hazi type which gives more milk. The consistency of yak milk is thick, fatty, cream in color and sweet in taste. Total yak milk yield is 147 - 487 kg during lactation period of 180 days has been reported (3). Yak milk and dairy products are popular foods in high-altitude regions. Yak milk contains 16.9 - 17.7% solids, 4.9 - 5.3% protein, 5.5 - 7.2% fat, 4.5 - 5.0% lactose, and 0.8 - 0.9% minerals. Yak milk is rich in fat, protein, essential minerals, and

healthy polyunsaturated fatty acids such as conjugated linoleic acid and omega-3 fatty acids (4, 5). In China, Yak x Holstein or Yak x Hereford cross cows are milked for their very rich milk which is used primarily for production of butter and cheese. The export demand for these products is greater than supply. In this country there is no Yak-cross milk or milk by-product market developed as yet. This could become a future production opportunity for American breeders. Indigenous cheese produced is locally termed "Churpi".

Yak Leather, Hides, Skulls and Tails

Yak leather can go to normal leather processing. Other specialty markets are currently being developed for this woolly, long haired hide, as well as processed skulls and tails.

Conclusion

The indigenous knowledge all together plays a pivotal role in maintaining yak sustainability and also their livelihood sustainability. Thus the documentation of traditional knowledge would provide the preservation of culture and indigenous practices of North and East Sikkim and used as a key for poverty mitigation and food security. The livelihood sustainability of the herdsman of the alpine Sikkim is mainly around the Yak, a "surefooted" multipurpose animal. Yak is their major income generating source for poor herdsman from its milk, meat, hair, skin to tail serving them to help their survival in this world away from transformation making them to value traditional values, religion, community and ethnic beliefs. Yak husbandry was found to be an indispensable part of mountain livelihood, equally linked with the social and cultural aspects of the people. Yaks have provided major income opportunities to many indigenous people living across higher altitude regions of Asian countries.

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Indian Gaddi Kutta or Pahari Leopard Hound

A GaddiKutta is a mastiff-type mountain dog found in Northern India, especially in states of the western Himalayan region: Himachal Pradesh, Uttarakhand, and Kashmir. They are also called Indian panther hounds and Mahidant mastiffs. Initially bred for hunting purposes, the multi-talented Gaddi Kutta is widely used by local shepherds, mostly Gaddis (from the tribe of the same name). They are said to be strong enough to repulse attacks by snow leopards and to have the intelligence to herd stray sheep and goats back to their pens.

Generally, found in the Himalayan ranges, Gaddi Kutta (dog) can be easily spotted herding flocks of sheep with shepherds who are commonly called 'Gaddis.' The dogs are so reliable with defense that a Gaddi (Himalayan Shepherd) has to barely worry about managing and protecting a large flock of sheep. Three to four gaddi dogs are necessary to protect a flock of nearly two thousand sheep. They keep the sheep safe from attacks by dangerous animals like the snow leopards or bears.

Although, the gaddi dog has always been an important component of the Himalayan life, sources about its origin are still ambiguous. Himalayan shepherds justify the origin of the gaddi dog as a cross breed between a tiger and a dog. But that's probably because the breed is as ferocious as a tiger and as loyal as a dog. Another legend says that the dogs are a cross between the wild dingo-like hounds from Himalayas and fighting line of Tibetan Mastiffs. Besides the two countering statements, there's a judicious answer to origin. Gaddi dogs are

basically sub-breed to Tibetan Mastiff and have naturally adapted themselves to survive in rough outdoor.

Superb Intelligent and Very Gorgeous

Known for its courageous temperament, a gaddi kutta (dog) has unmatched protective instincts. The breed has a heavily arched and massive neck acting as natural shield from attacks by not allowing predators to sink their teeth deep into flesh. Also, the dog has a massive skull, and his overall sharpness gets balanced with sturdy built. The figure is perfectly matched with a prominent occipital, straight back, deep low brisket and high hocks.

Strong and Ferocious

Rocky steep terrains and rapidly changing weather makes the Himalayan region a survival conflict. Moreover, things can get worse when protecting sheep from the predators in the nights and during the freezing winters. However, for a gaddi dog all such challenges are a child's play. With a thick, heavily-feathered tail and distinctive double coat around the neck & shoulders, the dog can easily stand a snow storm, and still not wither.

- **Weight:** 35 – 40 kg (female adult), 40 – 45 kg (adult male).
- **Height:** 63.5 cm (adult), 55.9 – 78.7 cm (adult male), 50.8–71.1 cm (adult female).
- **Temperment:** Gentle, calm, intelligent, and territorial, but loyal to their owners and very protective.



Diet-related Recurrent Hematuria in A Puppy: Diagnosis and Successful Treatment of the Rare Clinical Syndrome

Sabita Rakshit, Kabita Roy and I C Datta

Angel Animal Hospital, 24307 Halsted Road, Farmington Hills, MI, USA and College of Veterinary Science and Animal Husbandry, Jabalpur, Madhya Pradesh.

Abstract

Ralph Rothenberg, a male Doberman Pinscher puppy was presented to the Angel Animal Hospital, Farmington Hills, MI, USA with history of recurrent bouts of blood in the urine. The hemato-biochemical panel pointed to normal functional status of the vital organs: heart, liver and kidney. The electrolyte balance was not compromised. Referral ultrasound report did not reveal any serious patho-morphological anomalies in the urinary tract organs. However, slightly thickened bladder wall suggested chronic cystitis, possibly as a consequence of repeated exposure to newly formed batches of struvite crystals in the stored urine, leading to hematuria, and subclinical urinary tract infection (UTI). Protein-positive urine supported the tentative diagnosis. Switching over to the prescription diet, c/d® along with supportive medicines resolved the clinical problem, successfully.

Introduction

Abnormal urinary excretion of blood with mostly intact red cells, and discoloration ranging from amber to red, orange or brown often points to a serious underlying disease. The multiple causes include urinary tract infection (UTI), more common in the females, struvite (phosphates) accumulation in bladder, malignant growths, trauma, bleeding disorders, and benign prostate hypertrophy in the grown-up male dogs (1). However, beets, vitamin feed supplements, or certain medicaments may also impart reddish tinge to the urine. Diagnosis is based on in - depth analysis of the case history focused on trauma, exposure to toxic substance, or new diet / feed supplement (2).

On presentation in the clinic, an anamnesis is followed by thorough physical examination, urinalysis (pH, inorganic

Table 1. Patient's pre-treatment hemogram*

Parameter	Result	Ref. Interval	Status
TEC (x 106 /µL)	5.27	5.39-8.7	Low
Hematocrit (%)	38.9	38.3-56.5	Normal
Hemoglobin (g/dL)	12.2	13.4-20.7	Low
MCV (fl)	74	59-76	Normal
MCH (pg)	23.1	21.9-26.1	Normal
MCHC (g/L)	31.4	32.6-39.2	Low
Reticuloctyte (%)	1.8		
Reticulocyte (x106/µL)	95	10-110	Normal
TLC (x 103/µL)	12.5	4.9-17.6	Normal
Neutrophil (%)	39.6		
Lymphocyte (%)	38.6		
Monocyte (%)	5.7		
Eosinophil (%)	15.7		
Basophil (%)	0.4		
Neutrophil (x103/µL)	5.0	2.9-12.7	Normal
Lymphocyte(x103/µL)	4.8	1.1-5.0	Normal
Monocyte (x103/µL)	0.7	0.1-1.2	Normal
Eosinophil (x103/µL)	2.0	0.1-1.5	High
Reticulocyte (103/µL)	295	143-448	Normal

*Automated CBC

Table 2. Patient's blood biochemical profile

Parameter	Result	Ref. Interval	Status
Glucose (mg/dL)	120	63-114	High
SDMA (µg/dL)	10	0-14	Normal
Creatinine (mg/dL)	0.4	0.5-1.5	Low
BUN (mg/dL)	14	9-31	Normal
BUN/Creatinine ratio	35	-	
Phosphates (mg/dL)	9.2	2.5-6.1	High
Calcium (mg/dL)	11	8.4-11.8	Normal
Sodium (m mol/L)	145	142-152	Normal
Potassium(m mol/L)	4.4	4.0-5.4	Normal
Na+/ K+ ratio	33	28-37	Normal
Chloride (m mol/L)	110	108-119	Normal
HCO3- (m mol/L)	22	13-27	Normal
Anion gap (m mol/L)	22	11-26	Normal
Total protein (g/dL)	5.1	5.5-7.5	Low
Albumin (g/dL)	2.9	2.7-3.9	Normal
Globulin (g/dL)	2.2	2.4-4.	Low
A/G ratio	1.3	0.7-1.5	Normal
ALT (U/L)	20	18-121	Normal
AST (U/L)	35	16-55	Normal
ALP (U/L)	220	5-160	High
GGT (U/L)	2	0-13	Normal
Bilirubin-total (mg/dL)	0.1	0-0.3	Normal
Bilirubin-unconjugated (mg/dL)	0	0-0.3	Normal
Bilirubin-conjugated (mg/dL)	0.1	0-0.1	Normal
Cholesterol (mg/dL)	214	131-345	Normal
Amylase (U/L)	342	337-1469	Normal
Lipase (U/L)	88	138-755	Low
Creatine kinase (U/L)	619	10-200	High

Blood chemistry auto analysis*

crystals/ casts, red blood cells, hemoglobin, protein, ketone bodies and glucose) on a freshly collected sample, complete blood count, and blood biochemical panel. The size and shape of urinary crystals, if present, are determined conventionally under the microscope (3). Survey/ contrast radiography of the thorax and abdomen, ultrasonography, and biopsy may also be needed for

Table 3. Patient's circulatory thyroxin (T4) titer

Titer (µg/ dL)	3.1	1.0-4.0	Normal
<1.0	Low (Hypothyroid)		
1.0-4.0	Normal (Euthyroid)		
>4.0	High (Hyperthyroid)		

definitive diagnosis of any regional growth (4). Diet therapy plays an important role in the amelioration of the patient's patho - clinical condition

Case History

Rothenberg, a 16 weeks old male Doberman Pinscher puppy was presented to the Angel Animal Hospital on March 11, 2018 with a history of blood in the urine for the last few days. Physical examination revealed empty bladder with a few red drops of urine, dribbling from the penile orifice. Urinalysis, blood work, vWF test, blood coagulation panel (being Doberman breed), and ultrasonography were advised to the client.

The ethical and legal norms of the American Veterinary Medical Association on the humane care and treatment of sick animals were observed in full. In the instant case, the young owner was briefed and motivated regarding the desirability of suitable dietary modifications, besides the medicaments package, to eliminate the suspected root cause of blood in the voided urine, the pet's ongoing malady (5).

Diagnostic Panel

The dog patient's hemogram (Table 1), blood biochemical profile (Table 2), circulatory total thyroxin (T4) titer (Table 3), urinalysis (Table 4), and some special parameters (Table 5) were determined at the IDEXX Laboratories, Regional Center, MI on 13.03.2018.

Ultrasound Probe

Following in-house detection of protein +ve urine, ultrasound (single system renal) analysis was carried out at the referral Advanced Veterinary Care Center, Canton, MI on 28.03.2018. The results indicated that both kidneys were similar in size, shape and echotexture. Further, no masses or calculi were noted in any of the scanned urinary tract organs. The urinary bladder was distended with urine containing no detectable sediment. Both kidneys did not reveal any cysts, and there was no evidence of uroliths in either organ. Located within the pelvic canal, the prostate was not accessible.

As per the Board Certified Pathologist's observations, the urinary bladder wall appeared slightly thickened, presumably as a consequence of idiopathic cystitis. Further, the abnormal presence of protein in the patient's urine sample could not be attributed to any discernible abnormalities.

Remedial Strategy

The pet remained under treatment at the Angel Animal Hospital from March 11 to April 28, 2018. Cystitis leading to recurrent hematuria - the only diagnostic lead - was presumably the outcome of daily exposure to food origin minute, sharp-edged mineral crystals / casts. In perspective, depending on the patient's closely monitored patho - clinical response the line of treatment

Table 4. Patient's Urinalysis Panel

Parameter	Interval I (17-3-2018)	Interval II (25-4-2018)	Normal range
Collection	Free-catch	Free-catch	
Color	Brown	Red	
Clarity	Turbid	Turbid	
Sp. Gr.	1.060	1.046	
pH	6.5	5.0	
Protein	4+>0.5g/dL	4+(>0.5 g/dL)	Negative
Glucose		Negative	
Ketones	Negative	Negative	
Blood	3+	3+	
Bilirubin	Negative	Negative	
Urobilinogen	Normal	Normal	
Leukocytes	2-5	10-15	0-5/ HPF
Erythrocytes	>100	>100	
Bacteria	None seen	None seen	
Epithelial Cells	Rare (0-1)	1+ (1-2/ HPF)	
Mucus	None seen	None seen	
Casts	None seen	None seen	
Crystals	3+ MAP*	2+ MAP	

* Magnesium-ammonium phosphate /struvite

Table 5. Special Tests

Blood clotting parameters: PT, PTT within normal range vWF values within the normal range.

needed judicious periodic modifications.

1. March, 11, 2018. The dog patient was treated with Polyflex® @ 30 mg / kg, s/c and sent home with Clavamox® (62.5 mg tablet) PO 1 tab bid for 10 consecutive days.

2. March 19, 2018. Since the red urine persisted, it was evident that the antibiotic was ineffective in the instant case. The patient was given a dose of Dexamethasone [4 mg/ ml] @ 1.5 ml, s/c and Prednisone (5 mg tablet)PO 1 tab bid for 7days, 1 tab sid for 7 days, and 1 tab every other day (eod) for 7 days. The owner was also strongly advised to switch over to Hill's Prescription Diet c/d® dry Multicare Canine (12-13% protein). According to the owner's feedback, his pet's urine had cleared up completely.

3. March 28, 2018. Recheck in the clinic revealed red urine again. The steroid dose was doubled: Prednisone (5 mg tablet)PO 2 tabs under the same weekly therapeutic regimen. Glycoflex® (glucosamine prep) was prescribed, concurrently in view of efficacy in subsiding cystitis in pet dogs.

4. April 7, 2018. A dose of Polyflex® and Dexamethasone was given. The red urine stopped.

5. April 12, 2018. The red urine reappeared, which was highly intriguing. On persistent questioning the young owner admitted having arbitrarily changed the diet in the fallacious belief that his puppy(25.5 lb) will grow faster and stronger on the high protein diet. The pet was finally put on Hill's Prescription Canine Diet c/d® dry (12-13% protein) and Glycoflex®.

6. April 28, 2018. The puppy (28.5 lb) was given Polyflex® 1.5 ml, s/c in the clinic, Meloxicam® was also

administered, s/c. Take home Cephalexin® (250 mg tablet) PO 1-tab bid was prescribed for 10 days. The patient was continued on the Canine Diet c/d® dry, and the owner was convinced on staying steady with this food till the puppy turned into an adult dog.

7. April, 30, 2018. The highly satisfied owner informed that the puppy was doing very well with no blood in urine, continuously over the last several days.

Discussion

Demonstration of aggregates of sharp-edged struvite crystals (**Fig. 1**) along with blood (mostly intact red cells) with extremely low pH (interval II) in the patient's urinalysis report (**Table 4**) is highly relevant. Relatively low TEC, hemoglobin and MCHC in the blood circulation (**Table 1**) point to the onset of anemia, presumably because of reduced food intake and continuing blood loss in urine(hematuria).

Eosinophilia, evidenced by markedly enhanced absolute count, appears to reflect a hypersensitivity state (6). The low to normal serum creatinine, BUN and SDMA values (**Table 2**) point to the structural and functional integrity of the kidneys. Normal circulatory titers of ALT, serum bilirubin (total and fractions) albumin, and amylase activity remaining within the normal limits indicate unimpaired hepatic function. Normal AST titer attests to the structural patency of the myocardium with uncompromised pumping action of the heart. Hypoproteinemia appears to be related to the low circulatory concentration of globulin which, in turn, points to absence of any superimposed acute bacterial infection. The low lipase titer attests to normal pancreatic function. The high creatine kinase (CK) titer may be the consequence of extra strain imposed on the abdominal muscles in voiding urine (7). High circulatory phosphates concentration appears to be significant in relation to continuing precipitation of the struvite crystals (**Fig. 1**) inside the stored urine in the bladder (8).

Selection of the proper diet proved to be the turning point. The composition, especially the protein content and quality, sources: animal or vegetable of the Proprietary Prescription Diets c/d®Multicare Canine (canned/dry) SKU # 7001 and 10153, and k/d® Canine (canned/dry)

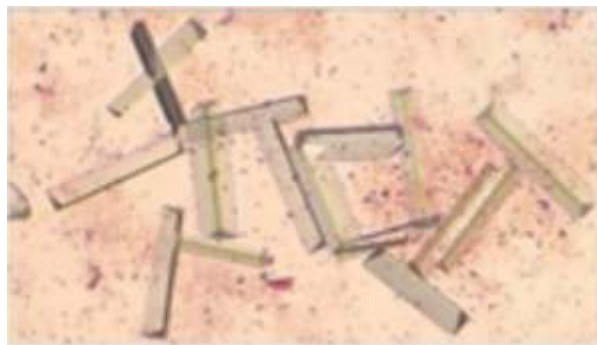


Fig.1 Ammonium Magnesium Phosphate Crystals or Struvite Crystals

SKU # 7010 and 8597 was carefully monitored by us. All four products were found well-balanced in nutrients, essential amino acids, omega-3 and omega-6 fatty acids, plus glucosamine and chondroitin sulphate. Finally, for the cystitis dog patient the c/d® product (12-13% protein) was preferred over the k/d® product (18- 20% protein).

Conclusion

The instant case of hematuria - without any covert pathomorphological anomalies in the urinary tract organs, or evidence of trauma, or bleeding disorders, or uroliths - appeared a perplexing clinical puzzle to us. However, there is always a cause-and-effect relationship and this malady could not be named, simply for clinical convenience, 'idiopathic cystitis'.

It was soon established that the basic cause was the very high protein food offered to the puppy by the overzealous young owner in the fallacious belief to own a sturdy, vigorous companion pet without bringing the fact to the attending clinician's knowledge. Food-related hematuria with latent UTI was successfully resolved; pH modulation (6.6-6.8 in the moderate acidic range) with the formula food might be crucial in pre-emptying the recurrent episodes of crystals-induced blood in dog urine. It is sincerely hoped that our contention will be substantiated with controlled clinical trials in future.

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Masticatory Muscle Myositis in Dogs - A Review

C Jayanthi, S Kavitha, A Prakash and P Srinesh
Madras Veterinary College, TANUVAS, Chennai, Tamil Nadu.

Introduction

Masticatory muscle myositis is an autoimmune, focal inflammatory myopathy with clinical signs restricted to muscles of mastication including temporalis, masseter, pterygoid and rostral digastricus, all of which are innervated by trigeminal nerve. Historically, it has been called as eosinophilic myositis or atrophic myositis (1). It is more common in young adult to middle aged dogs (2) and over represented in large breeds such as German Shepherd, Labrador Retrievers, Doberman, Pinschers and Golden Retrievers. No gender predilection has been found (1). It has also been reported in a grey wolf (3).

Pathophysiology

Biochemical and histological differences between the myofibres of masticatory and limb muscles provide the basis for selective targeting of masticatory muscles. Masticatory muscles are composed of muscle fibres that possess a unique myosin isoform and unique heavy and light chains, distinguishing the Type 2M fibres from the common Type 2C fibres of limb muscles. Inflammation, necrosis and phagocytosis are limited to these Type 2M fibres and there is circulating IgG directed against the unique myosin component (2). It remains unknown what initiates the formation of auto antibodies or why they are directed specifically against Type 2M fibres. Some theories suggest that molecular mimicry may play a role, with antibodies or T cells generated in response to an infectious agent that subsequently cross-reacts with self antigens. In this scenario, bacterial antigens would have a similar peptide sequence or conformational structure to some component of the 2M myofibres. Antibodies directed against these bacterial antigens could potentially cross-react with these myofibres (1).

Clinical Signs

Masticatory muscle myositis generally consists of two phases - acute phase and chronic phase. Dogs with acute masticatory muscle myositis often have a history of decreased activity, lethargy, fever, reluctance to eat, weight loss, drooling of saliva and change in bark (more high-pitched) (2). The acute phase is characterised by edema, swelling and pain in the masticatory muscles with restricted jaw movement (trismus) (4). Temporalis and pterygoid muscle swelling may cause exophthalmos resulting

in an inability to blink properly, ocular discharge, conjunctivitis and keratitis (2). If severe enough, exophthalmos can result in stretching of the optic nerve and subsequent blindness. Ocular signs have been noted in 44% of all the patients with masticatory muscle myositis. Mandibular and prescapular lymphadenopathy have also been reported during initial 1 to 3 weeks (1).

Conversely, the chronic phase is characterised by marked muscle atrophy due to the decreased size of the muscle fibres that lead to fibrosis. It may evolve to severe muscle atrophy and alteration of normal mouth movements (4). Enophthalmos may be present in the chronic phase because of atrophied pterygoid muscles.

Clinical signs are usually bilateral, but may appear to be unilateral in some cases if one side is more severely affected than the other (1).

Diagnosis

Complete physical and neurological examinations are important to confirm that clinical signs are restricted to the muscle of mastication (1). Masticatory muscle myositis must be differentiated from a variety of disorders of the head and neck that can make a dog unwilling or unable to open its mouth. These disorders include maxillofacial trauma, temporo mandibular joint disease, bone and soft tissue neoplasia, ocular and space occupying retro-orbital lesions, ocular disease and other muscular diseases such as polymyositis, extra ocular myositis, dermatomyositis and laryngeal myositis (2).

Detection of auto antibodies against Type 2M muscle fibres in serum is confirmatory test for diagnosis of masticatory muscle myositis (1). Haematological and serum biochemical abnormalities with masticatory muscle myositis may include eosinophilia and increased serum total protein concentration, creatine kinase activity and hepatic enzyme activities. Proteinuria has occasionally been identified in affected dogs (2).

Electromyography examination demonstrates severe and spontaneous pathologic activities (fibrillation and positive sharp waves) in the temporal and masseter muscles with no pathologic activities in either tongue or skeletal muscles of limbs (4).

Magnetic resonance imaging (MRI) deflected

widespread, symmetrical and in homogeneously hyperintense areas in the masticatory muscles using T2 weighted and GE STIR sequences. The T1 weighted sequences showed hypo intense areas between the masticatory muscles. T1 weighted sequences obtained after administration of the contrast agent detected intense and in homogeneous enhancement of the masticatory muscles, but the other muscle bellies were normal (4).

Hematoxylin and eosin staining of muscle biopsies from masticatory muscles (temporalis or masseter) revealed variable number of mononuclear cells with an endomysial and perimysial distribution. Varying stages of necrosis, phagocytosis and invasion of non-necrotic muscle fibres by inflammatory cells were seen. Abnormal variation in fibre size, increased number of fibres with centrally located nuclei and fibres with basophilic cytoplasm were also observed suggesting muscle regeneration. Indirect immunofluorescence can be done to analyze the extent of regeneration (5).

Computed tomography of the skull revealed the change in size (atrophy, swelling or both) of muscles of mastication. Enlargement of muscle was interpreted to be consistent with edema and active inflammation, whereas a decrease in muscle size was attributed to atrophy, necrosis or fibrosis. Hypoattenuating musculature was considered to be indicative of increased fluid content, consistent with inflammatory muscle edema. Swelling in the mandibular and medial retropharyngeal lymph nodes and bilateral new bone formation in the masseteric fossa were also observed (2).

Treatment

The treatment includes immunosuppressive therapy using corticosteroids (Prednisolone 2mg/kg, q12h) with antibiotics (Clindamycin 12.5mg/kg q12h). Clindamycin is chosen because of its activity against the most frequent cause of infectious myositis (*Toxoplasma*, *Neospora*) (4). The dose of Prednisolone is tapered to the lowest every other day dose that abates clinical signs. Tapering should generally occur slowly over 4 to 6 months with no more than 50% decrease in the dose every month. Minimum maintenance dose that abates clinical signs can be established. Many patients require this maintenance dose for lifetime. Others can ultimately discontinue all therapy (1).

Long term corticosteroid therapy may result in iatrogenic hyperadrenocorticism and susceptibility to infections. Owners must be prepared for resultant polyuria, polydipsia and polyphagia associated with prednisolone administration as well as the potential for steroid induced gastric ulcers (1).

Alternate immunosuppressive agents like azathioprine, cyclosporine, and colchicines may be used. Azathioprine (2mg / kg PO, q24-48h) have side effects like bone marrow suppression and hepatotoxicity. In addition to immunosuppression, colchicine is reported to have antifibrotic properties (1).

For chronic phase of masticatory muscle myositis, corticosteroids must be administered at lower doses. Gruel diets can be fed to dogs to maintain adequate nutritional intake. Patients can be encouraged to chew toys or bones to promote use of their masticatory muscles (1).

Prognosis

Prognosis is determined by the degree of fibrosis present and the clinical response to immunosuppression. Aggressive therapy in acute phase will have a good prognosis. Treatment failure and relapses usually result from inadequate immunosuppression and hasty discontinuation of corticosteroids. Dogs with chronic form of masticatory muscle myositis have uncertain prognosis but can do well if extensive fibrosis does not result in persistent jaw dysfunction (1).

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Surgical Management of Urolithiasis in A Female Dog

P Mukherjee, A Sanyal and D K Das
Veterinary officers, B.A.H.C., Taldangra, Bankura, West Bengal.

Introduction

Urolithiasis is a general term referring to the cause and effects of stones anywhere in the urinary tract (1). The term urolith is derived from the Greek word uro meaning urine and lith meaning stone. Cystolithiasis is the development of calculi in the urinary bladder and surgical removal of calculi from the urinary bladder is known as cystolithectomy (2). The large number of canine uroliths are found in the bladder or urethra.

Struvite (i.e. magnesium ammonium phosphate) and oxalate calculi are the most common canine uroliths followed by urates, silicates, cysteine and mixed type (3). Dachshund, Mastiff, Chihuahua, Basset Hound and Newfoundland breeds are predisposed to forming cysteine uroliths. Male dogs are affected to much greater degree than females, and most cysteine uroliths are found in lower urinary tract i.e. bladder and urethra(4).

History and Clinical Examinations

A 6 years old weighing 10kg, female Dachshund was presented with the history of haematuria and stranguria (difficulty in urination) for the last 6 months. After the clinical examination, it was found that the bitch had abdominal pain and rectal palpation revealed mild distended bladder. The owner was advised to do the plain oblique lateral radiograph of the abdomen and subsequently complete blood count(CBC), blood urea nitrogen (BUN), serum creatinine for confirmatory diagnosis and further course of action.

Primary treatment was done by Tab Marbofloxacin @ 5 mg / kg body weight once daily for 10 days and Iron-Folic acid syrup @ 5ml twice daily orally.

Diagnosis

The radiograph of abdomen revealed numerous radio-opaque spots in the urinary bladder. All haematological and serum biochemical parameters were higher than the normal values. It was presumed to diagnose as a case of cystolithiasis. So exploratory laparotomy followed by cystotomy were planned.

Treatment and Management

The bitch was premedicated with Atropine sulphate @0.04mg/ kg body weight SC and Xylazine hydrochloride 1.5mg/kg body weight IM. The general anaesthesia was administered and

maintained by Propofol @ 5mg/kg body weight IV to achieve the effect.

The bitch was placed on the table with dorsal recumbency after preparation of the surgical site with antiseptic lotion. A skin incision was made on ventral midline from umbilicus caudal to the pubis and abdominal cavity was approached through linea alba. Intra operative palpation of bladder revealed the presence of calculi. Then bladder was isolated from the abdominal cavity by placing normal saline soaked gauze beneath it. After that stray sutures were placed at the cranial end of the bladder to facilitated manipulation.

A 22 gauge needle and syringe was used for the aspiration of urine from the bladder. A stab incision was then made into the dorsal aspect of the bladder and incision is extended with the help of scissors. The calculi were removed with the forceps. The urethral catheter was passing from the urethral opening to the bladder to check for patency. Then lukewarm normal saline was flushed through the urethra and urinary bladder with the help of catheter to dislodge the remaining calculi.

Cystotomy wound was sutured in two layers using Vicryl (No:2-0) with simple continuous pattern engaging the sero-muscular layers and submucosa, while avoiding the mucosa. Then the abdomen was lavaged with luke warm normal saline and metronidazole solution. Laparotomy wound was closed with Vicryl (No. 1) using Ford interlocking pattern. The skin was apposed with nylon (No.1-0) in horizontal mattress pattern.

Post operatively the bitch was prescribed Ceftriaxone- tazobactam injection @ 25 mg / kg b.wt., IV for 5 days and inj Tramadol @ 2 mg / kg b.wt. SC for 5 days. The animal was further kept on Ringer lactate saline solution for 5 days and then subsequently shifted to semi solid diet.

The skin suture was removed on 10th post-operative day. The bitch made uneventful recovery without any complication.

Discussion

Urolithiasis is the most common cause of lower urinary tract infection in dogs and cats. Uroliths form as a result of multi congenital and or acquired pathological processes leading to increased concentration of less soluble crystalloids in urine. It is a common challenge faced by canine

practitioners as at most of the times the treatment is frustrating due to limited therapeutic options (5).

In dogs and cats, incidence of struvite uroliths is decreasing, whereas the incidence of calcium oxalate uroliths is increasing (6). Calculi are particularly common in the Corgi and Dashchund. Clinical symptoms include haematuria, pollakiuria, stranguria, abdominal discomfort, vomiting, anorexia, lethargies. In female dogs, repeated micturition, dribbling of urine and haematuria are the common clinical signs.

Diagnosis is based on history, physical examination, laboratory evaluation includes complete blood count, serum bio-chemical analysis and an oblique lateral radiograph of the abdomen. Most uroliths are larger than 3 mm, and have varying degrees of radio-densities, and therefore, can be detected by survey abdominal radiography or ultrasonography (7). Medical dissolution, surgical or combination therapy are the main treatments of urolithiasis. Surgical intervention along with medical management is the choice of therapy of urolithiasis. Urethral hydropropulsion followed by cystotomy can be adopted to remove large cystic calculi and small urethral calculi (8).

The incidence and composition of uroliths may be influenced by a variety of factors including species, breed, sex, age, diet, anatomical abnormalities, UTI, medication and urine pH (9).

The high protein diet increases hepatic production of urea, thereby increasing urea concentration in urine and renal medulla, and this concentrated urine is more prone to develop urethral obstructions (10).

Cystotomy and urethrotomy for rapid removal of uroliths from lower urinary tracts of canines was recommended (11). This surgical procedure remains same after hundred years also.

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Fig.1: Lateral view of abdominal radiograph shows numerous radio-opaque spots(arrow)



Fig. 2:Removal of Cystolith from Urinary bladder



Fig. 3:Collected Cystoliths after Cystotomy

Cardiomyopathy with High Grade Murmurs in A Female Chihuahua Dog: Diagnosis and Treatment

Rakshit Sabita and Roy Kabita

Angel Animal Hospital, Halsted Road, Farmington Hills, MI, USA and
TVCSA, CVSAH, Nanaji Deshmukh Veterinary Science University, Jabalpur, Madhya Pradesh.

Abstract

In this communication, the therapeutic management of severe cardiomyopathy with high grade (5/6) heart murmurs in a spayed geriatric female dog, along with the emergency care and long-term therapeutic management contributing to improved quality of the companion dog's life, the biophysical and biomedical implications are discussed in-depth. This information is expected to provide useful clues to the practicing veterinary clinicians.

Introduction

Dilated cardiomyopathy (DCM), an adult onset syndrome in dogs involves progressive degenerative thinning of the cardiac muscles, culminating in abnormal dilatation of the chambers. Obstructed flow of blood resulting from impaired myocardial function, is responsible for heart murmurs. These abnormal sounds originate from turbulence in the blood flow because of stenosed / leaky valves, or the deformed shunts with ventricular septal defect (VSD): hole between the left and right ventricles, and patent duct arteriosus (PDA). In the dog patient, heart murmurs with or without clinical symptoms are auscultated, often as a chance observation by the physician (1). Reduced viscosity of the peripheral blood may produce heart murmurs (2). Young puppies may exhibit 'innocent' heart murmurs, which usually pass off uneventfully by six months of age (3). The etiology of DCM remains uncertain. The suspected genetic predilection in some breeds, e.g. New Foundland and Dashchund is currently under investigation.

In DCM, the clinical symptoms depend on the grade, configuration and location of the heart murmurs (4). The initial warning signs are pale or bluish gums, often detected during the pet's weekly healthcare schedule at the client's home. The highly distended abdomen may be the consequence of body fluid retention, secondary to the primary cardiac disorder. Panting / fainting episodes mandate urgent veterinary medical care (5).

The diagnostic tests for DCM include chest x-rays to determine accumulation of fluids or air in the lung parenchyma, enlargement of the heart, engorgement of air in the thoracic cavity, tumors, fractures; colour flow doppler (ultrasound) to monitor the direction and speed of circulating blood in the cardiovascular system; echo cardiogram (sonogram) of the patient, without sedation, to

evaluate with high precision the heart's pathophysiological status, and blood work to corroborate anemic murmurs (6). The morphological site where the heart murmur is loudest is named the point of maximal intensity (PMI). The clearly audible S1 (AV valve closure) and S2 (semi-lunar valves closure) sounds represent the Lub-dub, Lub-dub, Lub-dub... synchronized acoustic credentials of the normal functioning heart. Turbulence in the blood flow, on the contrary, generates the extra 'whooshing' sounds at any point in the cardiac cycle. In our long-term shared clinical experience these are often heard as lub - whoosh - dub, or whoosh-lub-dub, or lub - dub - whoosh (extrapolated S3, S4) sounds.

Survey radiographs pinpoint the patient's pathoclinical state like pulmonary edema or venous congestion, and enlargement of the myocardium. Evaluation of the blood biochemical profile (changes in the values of BUN, creatinine, symmetric di-methyl arginine / SDMA, and creatine kinase - a sensitive biomarker of cardiac myopathy) is useful in monitoring the state of renal perfusion. The electrocardiogram (ECG) pattern is aimed to elucidate the nature and magnitude of deviation from the classical wave pattern of 'PQRST', and the time intervals between the successive waves. Imaging protocol records evidence-based chamber enlargement, ventricular premature contractions (VPC), tachycardia, bradycardia, heart block, and atrial fibrillations.

The diagnostic back-up is highly useful in deciding on the optimized holistic remedial therapeutic option, often incorporating the initial use of a diuretic e.g. Frusemide, Prilactone to reduce the fluid overload and resolve the concurrent pulmonary edema, angiotensin I→II converting enzyme (ACE) inhibitor drug e.g. Benazepril as an effective synergistic vasodilator, and anti-hypertensive agent acting through suppression of ACE-mediated biosynthesis of the antidiuretic hormone (ADH). Pimobendan,

with positive inotropic-cum-vasodilator effects, may also be prescribed. The anti-arrhythmic agent e.g. Fortekor / Enacard is aimed to relieve the overstretched heart muscles in a variety of cardiac arrhythmias, e.g. atrial fibrillation with accelerated heart rates.

Fig 1. Radiograph of patient's thorax and abdomen



The standardized ascending linear scale (1 to 6) is now universally accepted in grading heart murmurs.

Grade 1: A very soft focal murmur, audible after several minutes of attentive listening.

Grade 2: A soft murmur, readily auscultated and well located.

Grade 3: A moderate intensity murmur, auscultated simultaneously in more than one location.

Grade 4: A moderate to loud intensity murmur, radiating all over the chest wall without accompanying palpable thrill.

Grade 5: A loud murmur, radiating all over the chest wall with palpable thrill.

Grade 6: The loud grade 5 murmur, audible even after the withdrawal of stethoscope.

Case Presentation

Mona Johnson, an aging 12 years and 6 months old, 3.2 kg, spayed female Chihuahua dog was presented to the Angel Animal Hospital, Farmington Hills, Michigan, USA on January, 23, 2018 with the history of labored breathing and continuous cough overnight, leading to rapidly deteriorating body condition.

Physical examination revealed subnormal (97.7° F) rectal temperature. The visible mucous membranes were cyanotic. Heart murmur (grade 5) was clearly auscultated. Respiration was gasping. With an abnormal bluish tinge, the tongue was conspicuously protruding.

The ailing animal was apparently dehydrated with

the abdominal wall tensed-up and rigid. The dog patient needed 24 hr intensive care in veterinary emergency center, but because of financial constraints the owner declined. The critically ill patient was attended to by a team of veterinarians and technicians on a challenging life-saving mission in the Angel Animal Hospital.

Diagnosis

Mona Johnson, the seriously endangered dog patient needed to be adequately stabilized, prior to the routine in-house diagnostics protocol: regional survey radiographs and blood work (1, 7). The patient was placed in the 482001 Oxygen chamber (4 hr). Survey radiograph of the thoracic cavity and abdomen (**Fig. 1**) was taken promptly. Enlarged cardiac silhouette with accumulation of fluid in the chest and abdomen explained gasping and cyanotic mucus membrane was clearly discernible. The lung parenchyma, trachea, rib cage and blood vessels remained within the normal morphological limits.

Medical Management

The prognosis varies depending on the severity of cardiac myopathy, remedial strategy, and the important, but often underestimated criterion: daily diet and light exercise schedule. In the instant case, the initial treatment in the clinic comprised Frusemide, Lasix® [10 mg/ml] 1.1 ml i/v along with a dose of Polyflex® [250mg/ml] 0.35 ml s/c. The body condition improved markedly. The patient kept under close clinical surveillance for 8 hr was released in the same evening to the owner's care. Recheck in the clinic on the following day revealed that the patient was feeling much better, and breathing normally. Follow - up treatment schedule at the client's home: Lasix® (20 mg) @ ½ tablet b.i.d. for 14 days, Benazepril (10 mg) @ ½ tablet b.i.d, and Pimobendan (Vetmedine®) 2.5 mg½ tablet b.i.d. for 14 days. The patient was rechecked in the clinic after two weeks, and life-time treatment (with the dose schedule reassessed periodically) was advised to the well-informed, satisfied owner.

Discussion

In both humans and dogs, dilated cardiomyopathy (DCM) presents strikingly similar features: enlargement of the heart, grossly impaired ventricle, dilated left atrium, and arrhythmias that may progressively lead to heart failure.

The heart's ability to maintain the steady state is overwhelmed, and the ever busy pump is unable to function properly. This results in fluid retention in the blood vessels, tissue edema, dyspnoea and exercise intolerance. Cough represents the body's reflexes in an attempt to clear off the exudates from air passages. In this case, the patient responded

well to the holistic therapeutic regimen combined with the well-planned home healthcare package, including balanced nutrition.

Oxidative stress-mediated cardiac cell apoptosis (programmed cell death) precedes progressive ventricular dysfunction in DCM in dog (8). Presumably because of the accelerated production and release of highly deleterious free radicals in the myocardium cells (9). A cascade phenomenon, triggered by superoxide anion O_2^- and involving toxic oxygen derivatives, reactive hydroxyl radical OH^- and hydrogen peroxide H_2O_2 (10) may be involved. Dietary essential omega 3 and omega 6 fatty acids significantly reduce the cell sensitivity to toxic free radicals. Coenzyme Q 10, vitamin E (tocopherols), polyphenol antioxidant molecules protect the cell by neutralizing the toxic free radicals. The high-risk middle aged companion dogs need close monitoring with judicious nutritional support (11).

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Pericardial Lipoma in A Senescent Maltese Dog: A Case Report

Sabita Rakshit, Dale Clark and Kabita Roy

Milford Veterinary Clinic, Milford, USA and College of Veterinary Science and Animal Husbandry (NDVSU), Jabalpur, Madhya Pradesh.

Abstract

A ten-year-old Maltese neutered male dog was presented to the Milford Veterinary Clinic (MVC), Milford MI, USA on 29 November, 2018 for treatment of suspected otitis, and was prescribed topical antibiotic application, after cleaning each ear, b.i.d. for 7 days. On repeat visit to the clinic on 16 January, 2019 the client informed that the pet was limping with pain, holding the tail down. Heart murmurs of moderate intensity (4/6) could be auscultated easily. Enhanced pain perception on digital palpation of the spine in the mid-thoracic and lumbar regions was evident. In-house survey radiograph images revealed compression in the mid-thoracic and lumbar regions. Besides cardiac enlargement, radiopacity in the pericardial area suggested the existence of extraneous tissue growth. The patient was, therefore, referred to the veterinary cardiologist for ultrasound evaluation and specialized treatment.

In dogs, tumor in the heart and associated structures is often a chance observation (1, 2). *Hemangiosarcoma*, *myxoma* and *rhabdomyo sarcoma* are the most common primary lesions (3). Metastatic tumors include thyroid carcinoma, mastocytoma, and lymphoma (4). Lipoma usually appearing as a soft, well-circumscribed, smooth subcutaneous mass on the trunk or proximal extremities - a benign growth originating from the adipose tissue - is commonly observed in the middle aged and senescent dogs (5). The path morphological entity appears as a homogenous mass in computed tomography images (6). Primary cardiac lipomas, usually encapsulated, are extremely rare benign tumors. The commonly affected sites are the right atrium, left ventricle, and inter-atrial septum (7, 8). This communication reports on the chance detection of intrathoracic lipoma in an aging Maltese dog with clinical signs of neuromuscular incoordination.

Case History

A 10-year-old Maltese neutered male dog, Romeo Giannandrea was brought to the home clinic, MVC for periodic health check/ vaccination schedule. On November 29, 2018 the pet was treated for ongoing otitis: Otomax® topical application b.i.d. after cleaning with TrizUltra® solution with cotton swabs, daily for 7 days. During the next visit on January, 15, 2019, the lady owner complained about her companion animal limping with pain for the past three days. The pet was eating well and exhibiting near normal bowel movements, but holding the tail downwards in an abnormal posture.

Diagnosis and Treatment

On physical examination, rectal temperature 102.6° F, heart rate 96/ minute, pink visible mucous membranes, CRT < 2 seconds were recorded. Further, heart murmur of moderate intensity (4/6) left systolic, dental tartar 2/4 (periodontal disease), and turgid anal glands were observed. Both eyes exhibited perceptible cloudiness. Accentuated pain perception on systematic digital palpation of the spine in the mid-thoracic and lumbar regions was noteworthy. In-house survey radiograph images revealed mild compression in the mid-thoracic and lumbar regions. With marked cardiac enlargement, radiopacity in the pericardial area suggested the existence of abnormal tissue growth. The patient was, therefore, promptly referred to the cardiologist for ultrasound evaluation and specialized treatment. In the interim period, injectable Meloxicam® @ 0.2mg/ kg was administered S/Q, followed with oral Meloxicam® OD with food for 5 days with need-based adjustments. In the home clinic, the anal glands were expressed carefully. Relatively high values of Hb and erythrocyte indices: MCV and MCHC and circulatory total protein (Table 1) pointed to hemoconcentration, presumably because of tissue dehydration. Increased monocyte count indicated pathogenic bacterial infection, corroborated by the elevated circulatory globulin concentration. Elevated SDMA value (creatinine remaining within the reference interval) a highly sensitive biomarker not impacted by the muscle mass-indicated the onset of renal dysfunction. Increased values of alkaline phosphatase, bilirubin and cholesterol pointed to impaired hepatic function.

Table 1. Patient's hemato-biochemical profile on January 15, 2019 (IDEXX Diagnostics Lab).

Parameter (units)	Value	Reference interval	Status	Parameter (units)	Value	Reference interval	Status
TEC (1x10 ⁶ /μL)	155.0	70-143		Glucose (mg/dL)	155.0	70-143	High
Hematocrit (%)	45.3	37.3-61.7		SDMA (μg/dL)	17.0	0.0-14.0	High
Hemoglobin (g/dL)	21.5	13.1-20.5	High	Creatinine (mg/dL)	0.7	0.5-1.8	
MCV (fL)	72.2	61.6-73.5		BUN (mg/dL)	13.0	7-27	
MCH (pg)	34.3	21.2-25.9	High	BUN/ Creatinine ratio	19	7.0-27.0	
MCHC (%)	47.5	32.0-37.9		Inorganic P (mg/dL)	2.8	2.5-6.8	
RDW (%)	17.7	13.8-21.7		Total calcium (mg/dL)	9.3	7.9-12.0	
Reticulocyte (%)	1.7			Total proteins (g/dL)	8.4	5.2-8.2	High
Reticulocyte-Hb (pg)	27.0	22.3-29.6		Albumin (g/dL)	3.3	2.2-3.9	
TEC (1x 10 ³ /μL)	12.6	5.1-16.6		Globulin (g/dL)	5.1	2.5-3.9	High
Neutrophil (%)	76.8			A/G ratio	0.6		
Lymphocyte (%)	9.1			Alanine amino- transferase (U/L)	87	10-125	
Monocyte (%)	9.1			Alkaline phosphatase (U/L)	702	23-212	High
Eosinophil (%)	2.5			Gama glutamyl transferase (U/L)	0.0	0-11.0	
Basophil (%)	2.5			Total bilirubin (mg/dL)	1.7	0.0-0.9	High
Thrombocyte (%)				Cholesterol (mg/dL)	409.0	110-320	High
Neutrophil (1x10 ³ /μL)	9.66	2.95-11.64		Amylase (U/L)	540.0	500-1500	
Lymphocyte (1x10 ³ /μL)	1.15	1.05-5.10		Lipase (U/L)	559.0	200-1800	
Monocyte (1x10 ³ /μL)	1.15	0.16-1.12	High	Na ⁺ (m mol/L)	4.6	3.5-5.8	
Eosinophil (1x10 ³ /μL)	0.32	0.06-1.23		K ⁺ (m mol/L)			
Basophil (1x10 ³ /μL)	0.31	0.00-0.10	High	Na ⁺ /K ⁺ ratio	33.0		
Thrombocyte (1x10 ³ /μL)	653	148-484	High	Cl ⁻ (m mol/L)	112.0	109-122	
MPV (fL)	11.4	8.7-13.2		Osm. Calcium (m mol/kg)	301.0		
PDW (fL)	15.8	9.1-19.4		Total T ₄ (μg/dL)	1.4	1.0-4.0	Normal
PCT (%)	0.74	0.14-0.46	High	Diagnostic interpretation for total T ₄ values (μg/ dL) <1.0 Hypothyroid; 1.0-2.0 Low normal; 1.0-4. Normal (Euthyroid) Hyperthyroid; 2.1-5.4 Therapeutic			

Radiography

The patient showed accentuated pain perception on digital palpation of the spine in the mid-thoracic and lumbar regions. In-house spinal radiographs (Fig.1a, b; Fig.2a, b) revealed narrowing of the intervertebral disc spaces and compression at T8-T11, and L6-L7 spinal vertebrae. The pericardial region exhibited a conspicuous area of radiopacity with the cloudy adjoining pulmonary domain,

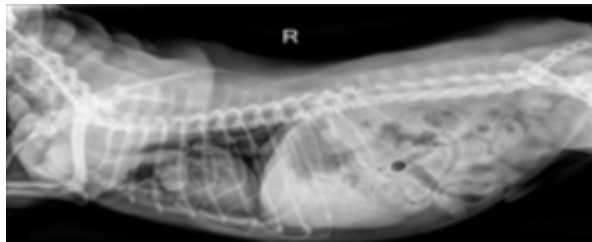


Fig. 1a. Right Lateral Survey Radiograph: Spherical mass near heart and T 8-11 compression.

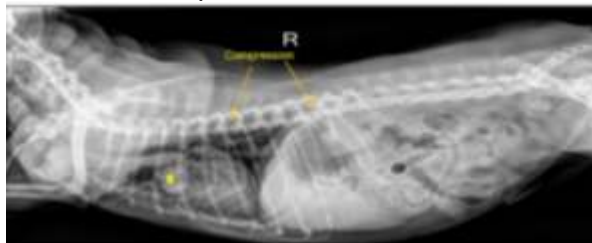


Fig. 1b. Right Lateral Radiograph: Note Spherical mass (*) near heart and T 8-11 compression.

suggestive of abnormal spherical tissue mass. Myocardial enlargement was clearly discernible. Enlarged appearance of the liver lobes indicated hepatomegaly. For further evaluation, an ultrasound (echocardiogram) probe was suggested. The case was referred to the veterinary cardiologist.

Computed Tomography Scan

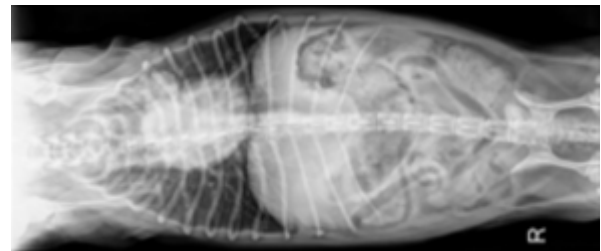


Fig. 1b. Right Lateral Radiograph: Note Spherical mass (*) near heart and T 8-11 compression.

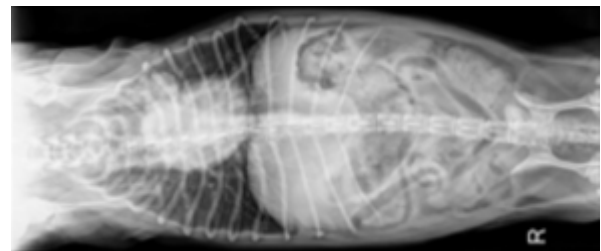


Fig. 2b. VD: Narrowing in T 8-11 and L6 – L7 and pericardial mass (*) at 3 - 6th intercostal space.

CT scan is the best option to confirm the tumor's precise location in the thoracic cavity, histo-architecture, peripheral extensions, and possible local lymph node involvement. CT scan needs a bolus of iodinated contrast dye, injected I/V. In the instant case, lipoma of pericardial origin with localized pericarditis was diagnosed through ultrasound-guided histopathology.

Surgery

Exploratory thoracotomy of the pericardial mass removal depends on the size, location and the pathophysiological implications. In the instant case, the patient was at high - risk to undergo the surgical procedure for a benign, unobstructive, small growth. Therefore, in compliance with the cardiologist's expert opinion surgical intervention was ruled out. However, in the standard protocol, the sedated patient is put under general anesthesia with isoflurane gas, and secured in right recumbence. A single 8-10 cm long incision is made in the intercostal space between two adjacent ribs, from the 3rd to the 6th rib. The patient's respiratory system is supported with the IPPV (Intermittent Positive Pressure Ventilator), or Manual Resuscitator, Ambu Bag (9). The mass, located with high precision in the thoracic cavity, is dissected out with the minimum one cm safe margin all-round. The surgical incision is carefully closed with adequate number of absorbable sutures. Very importantly, before tying up the last suture, negative pressure is generated with the specially designed vacuum pump, or judicious syringe suction through the sterile tubing fixed securely in the thoracic cavity to enable the lungs to inflate without mechanical aid.

Histopathology

In the case under report, ultrasound-guided histopathology confirmed the mass as lipoma, localized in the pericardial area. Microscopic evaluation of H&E stained section revealed a nodular homogenous lesion comprising mostly mature adipocytes. The mass appeared to be encompassed in a fibrovascular capsule with a few hemorrhagic foci and patchy infiltration of evaginated neutrophils, lymphocytes, plasma cells and a few macrophages. Irregular bands of inflamed fibrovascular tissue from the pericardium were also seen.

Discussion

Scanning of the published literature revealed that in a senescent dog with imaging evidence, the marked pericardial effusion leading to congestive heart failure was attributed to a heart base growth (7). In another report, extensive locally infiltrative mass, involving the myocardium in the right ventricular area promoted congestive heart failure concurrent

with thrombosis in the right atrium, and pleural/pericardial effusions (8). In the instant case, an early decision was taken for referring the patient to the cardiologist for proper diagnosis and specialized treatment. The original complaint of pain with limping was effectively managed in the home clinic with judicious Meloxicam® supportive therapy Vitamin E and joint supplements. There is a striking similarity in the gross and microscopic appearance of pericardial lipoma in the instant case and an earlier report (6). The patient is receiving the cardiologist's long-term medical care. The follow-up feedback from the client is positive.

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Canine Pyometra: An Overview

Jisna K S and Sivaprasad M S

Division of Animal Reproduction, Division of Veterinary Public Health, ICAR - Indian Veterinary Research Institute, Izatnagar, Bareilly, Uttar Pradesh.

Abstract

Pyometra is one of the most important and common reproductive disorders in canines. The condition results from the bacterial invasion of uterus and leads to severe septicaemia and toxæmia if left untreated. The breed, age, parity and administration of exogenous hormones are some of the predisposing factors of pyometra. An exaggerated and abnormal response to chronic and repeated progesterone stimulation leads to Cystic Endometrial Hyperplasia, which can later progress in to accumulation of purulent contents inside the uterine lumen. The Cystic Endometrial Hyperplasia does not always precede to pyometra and pyometra can occur without Cystic Endometrial Hyperplasia. Pyometra can be of open and closed type. Closed cervix pyometra is more dangerous and death of the patient can happen due to septicaemia and toxæmia. The condition can be best treated by ovariectomy however hormonal treatments are preferred in cases where the owner desires to retain the breeding potential of the dog.

Introduction

One of the most common reproductive emergencies of canine seen in the veterinary field is pyometra. It is the secondary infection of uterus that is resulting in the accumulation of pus inside the uterus. Occurs as a result of hormonal imbalance in the female's reproductive tract and making the bitch very ill. The condition occurs mainly during diestrus period in adult nulliparous bitches (1,2). The condition is classified in to open or closed cervix pyometra. Closed cervix pyometra is more severe and death of the patient can happen due to septicaemia and toxæmia if left untreated. The condition is not only limited to genital system and may establish systemic illness resulting in impaired homeostasis, cystic hyperplasia of the endometrium, inflammatory cell infiltration of and accumulation of exudates in the uterine lumen. The condition succeeds following cystic endometrial hyperplasia resulting in infertility either due to fertilization failure or early embryonic death (3). Although the disease has been recognized for several decades, the true disease process has still not been completely identified.

Breeds and age of the dog susceptible to pyometra

Pyometra can occur in any breed of dog. Although the condition is more common in Spitz (39.56 percent), followed by Labrador (12.94 percent), Alsatian (11.87 percent), non-descript dogs (11.51 percent), Doberman Pinscher (7.19 percent), Boxer (3.95 percent), Dachshund and Pomeranian (2.87 percent each), Rottweiler (2.51 percent) (4). It has been suggested that pyometra is a condition of

middle aged and older dogs. However, pyometra has been reported in dogs as young as 4 months old and as old as 16 years. The average age of diagnosis is about 9 years.

Pathogenesis

The pyometra is a progesterone mediated uterine disorder occurring during the diestrus period. The pathogenesis involves stimulation of the uterus by the estrogen which enhances the susceptibility of uterus. Excess level of estrogen leads to over proliferation of endometrium and lengthens the period in which the cervix remains open. It also enhances the stimulatory effects of progesterone on the uterus. For approximately 9 to 12 weeks following ovulation in each estrous cycle, there is a markable rise in the concentration of progesterone often exceeding 40 ng per ml. During progesterone stimulation the canine endometrium proliferates, branching and coiling of endometrial gland results in production of uterine glandular secretion called "uterine milk". During this period progesterone maintains the functional closure of cervix and inhibit the myometrial contractility. Progesterone-induced endometrial hyperplasia generally leads to the development of pyometra (5). Further hyperplasia becomes cystic, with the end result being a pathologic process referred to as cystic endometrial hyperplasia. The endometrium becomes more thick due the increase in the size and number of endometrial glands, which may show secretory activity. Occasionally, the cystic hyperplasia results in an accumulation of thin or viscid sterile fluid within the lumen of the uterus. This condition is commonly termed as hydrometra or mucometra. The hormonally compromised uterus becomes

susceptible to infection by opportunistic bacteria that are derived from normal vaginal microflora or urinary tract. Bacteria proliferate in the lumen containing excessive secretory fluid. Cystic endometrial hyperplasia does not always precede to pyometra. The pyometra can occur without cystic endometrial hyperplasia. These bacteria invade the

uterus through the relatively dilated cervix, during proestrus and estrus(6).The Lipopolysaccharides (LPS) released from Gram-negative bacteria cause dysfunction of leucocytes and uncontrolled production of inflammatory cytokines finally leads to permanent damage to internal organs, sepsis and death of the animal.

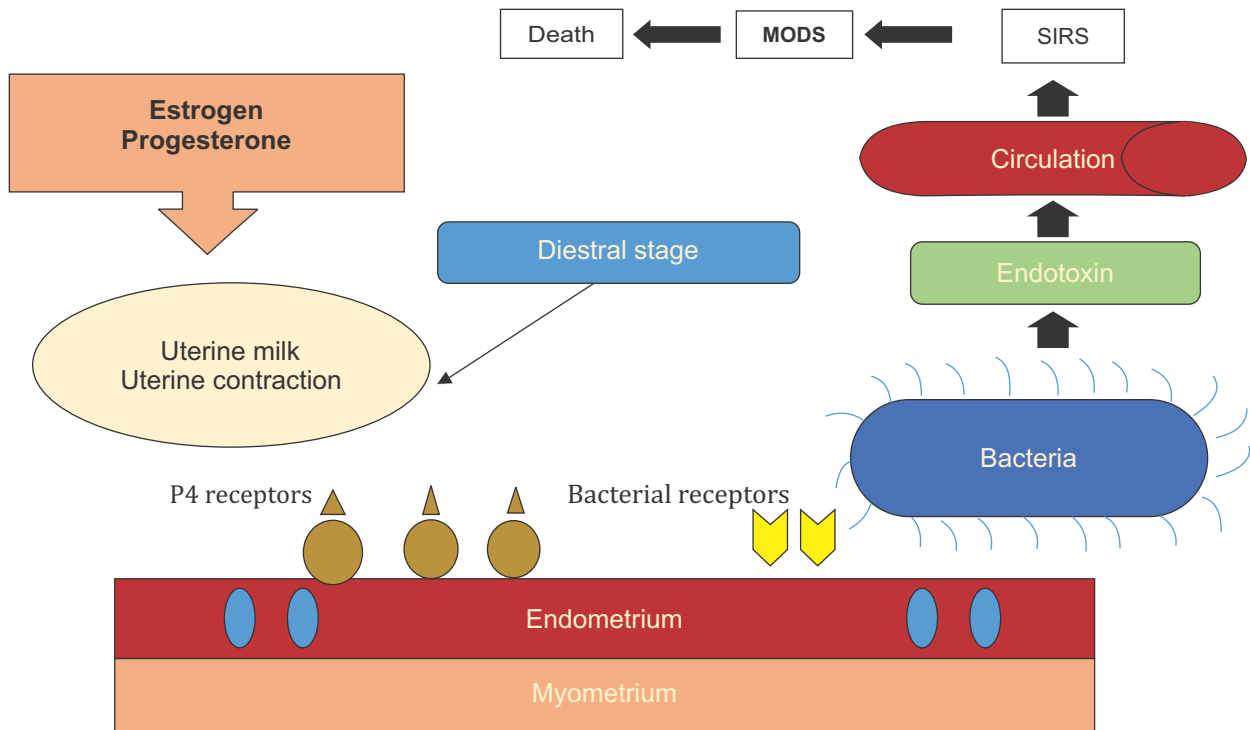


Fig.1 An illustration of the hormonal and bacteriologic components in the pathogenesis of pyometra.

Clinical signs

Because the infection can be so severe, the signs the pet may show are not limited to the genital tract. Dog can become so overwhelmed by the

inflammation associated with the infection and the uncontrolled inflammatory process may be life-threatening. The clinical signs depend on patency of the cervix.

Table 1. Clinical signs associated with pyometra

Open Cervix Pyometra	Closed Cervix Pyometra
▶ Discharge of pus through vagina	▶ Become severely ill rapidly
▶ Soiling of tail and bedding material with discharge	▶ Anorectic, very listless
▶ Fever	▶ Vomiting or diarrhoea
▶ Anorexia	▶ Polydipsia
▶ Lethargy	▶ Polyuria
▶ Depression	▶ Dullness and depression

A closed pyometra often goes unnoticed by pet owners until the patient is in critical condition. While any version of pyometra is serious, closed pyometra cases often have the poorest prognosis.



Fig.2 Distended uterus filled with purulent material



Fig.3 Endometrium with CEH

Diagnosis

Pyometra is diagnosed through a combination of examination, a history of any or all of the symptoms mentioned above. The definitive and important history includes history of estrus 4 weeks to 4 months back and in most of the cases there is a history that bitches have never been mated. Dogs that are examined early in the course of the disease may have a slight vaginal discharge and show no other signs of illness. However, most dogs with pyometra are seen later in the illness. A very ill female dog with a history of recent heat that is drinking an increased amount of water should be suspected of having pyometra. The history includes onset of most important clinical signs such as polydipsia, polyuria, together with vomiting, lethargy, depression and inappetence. This is especially true if there is a vaginal discharge or a painful, enlarged abdomen.

Clinical examination

The findings during clinical examination depend

upon the patency of cervix. In open-cervix pyometra, the most important finding is presence of foul-smelling, sanguineous, mucopurulent discharge(7). Systemic signs such as vomiting, depression, lethargy, polydipsia, polyuria are less in bitches with open-cervix pyometra (8). In contrast, closed-cervix pyometra is regarded as a medical emergency. Bitches are very ill at presentation and show marked systemic signs and distended abdomen. Affected bitch are dehydrated, hypothermic due to onset of toxæmia (9).

CBC and Serum biochemistry

Dogs with pyometra usually have a severe elevation of the white blood cell count and often have an elevation of globulins (a type of protein often associated with the immune system) in the blood. Absolute neutrophilia > 25000 cells / mm³ with shift to left is usually seen in 50-75% of bitches. Elevation of blood urea nitrogen is seen in cases where pre renal uraemia is present. Haematology is an integral part of pyometra diagnosis.

Urinalysis

The specific gravity (concentration) of the urine will be generally low due to the toxic effects of the bacteria on the kidneys. However, these changes are non-specific and may be seen in any dog with a major bacterial infection.

Radiographs

If the cervix is closed, radiographs (X-rays) of the abdomen will often identify the enlarged uterus as a dense tubular structure larger in diameter than small intestinal loops. The differentiation of condition from early pregnancy is a difficult task until mineralization take place.

Ultrasound

An ultrasound examination may be helpful in identifying an enlarged uterus, thickness of uterine wall and the accumulation of contents inside the lumen. The uterine contents are usually homogenous but usually appear as echo dense with slow swirling patterns (10).

Systemic inflammatory response syndrome (SIRS)

SIRS is the clinical manifestation of the response to inciting stimulus, that is capable of releasing inflammatory mediators. Canine pyometra is called the naturally occurring model for SIRS. The condition occurs when circulatory shock is superimposed on sepsis and is often associated with canine pyometra. Even the less severely affected patient with SIRS is at the risk of multiple organ dysfunction syndrome (MODS). There are four proposed criteria to identify a patient with SIRS. If the suspected animal exhibits any of the two

criteria it can be considered as SIRS positive. These are not so accurate to be considered definitive but can be used as guidelines along with clinical judgement until specific markers for SIRS are identified.

Table 2. SIRS criteria

Criteria	Value
Temperature (F)	<100.6o or >102.6o
Heart rate (beats/min)	<120
Respiratory rate (breaths/min)	>20
White blood cells (×103)	<6 or >16

Treatment

The most appropriate treatment is to surgically remove the infected uterus and ovary by performing an ovariohysterectomy. Dogs diagnosed in the early stage of the disease are very good candidates for surgery. The surgery is somewhat more complicated than a routine spay at this stage. Intravenous fluids are required to stabilize the dog before and after surgery. Antibiotics are usually given for two weeks after surgery. Surgery is always advised for “closed” pyometra, where the purulent contents will retain in the uterine lumen.

Surgical drainage is another treatment strategy where purulent material is aspirated out and each of the uterine horn is aspirated with antiseptic solution with an indwelling catheter. The technique requires plenty of skill and luck and not normally recommended.

Although surgery is the treatment of choice, based on certain criteria veterinarian may allow valuable breeding animals to be treated medically. However, in addition to the previously mentioned multiple organ problems, the dogs will generally exhibit excessive vomiting, abdominal pain, defecation, salivation, high heart rates, panting, breathing difficulties and fever with medical management. Some pets may take several days to show a positive response to injectable prostaglandins and antibiotics. Along with injectable broad spectrum antibiotics, IV fluids with appropriate electrolyte and bicarbonate supplementation is also needed.

Prolactin is luteotropic in bitches hence use of antiprolactin drugs will be effective to reduce the concentration of progesterone. Dopamine agonists such as Bromocriptine and Cabergoline are substantially used for the treatment of pyometra.

The use of PGF2α is common in cases where the cervix is open. It causes the contraction of myometrium, reduction in the circulating progesterone concentration and relaxation of cervix. Should not be used in older bitches and in

bitch with known cardiac and respiratory disorders.

If treatment is not performed quickly, the toxemia from the bacteria will be fatal. If the cervix is closed, there is chance for the uterus to rupture, spilling the infected contents into the abdominal cavity. Pyometra is an emergency medical condition that requires prompt treatment. If animal is not responding to medical treatment after five days, spaying should be considered. If treatment does prove to be successful, the bitch should then be bred on her next estrous cycle. However, in intact dogs that have had pyometra before, there is a high risk of recurrence.

Conclusion

About one in four older female dogs suffer from pyometra. The best way to prevent pyometra is to spay the dog at younger age or at the end of their breeding age. The pyometra is a medical emergency which need immediate treatment, leaving a pyometra without treatment is likely to cause severe illness, suffering and death. The prognosis for survival with ovariohysterectomy can be as high as 80–100% if abdominal contamination is avoided and the systemic infection is treated and responds appropriately. However, if severe sepsis and organ failure develops, the prognosis can be less favourable.

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Clinical Management of Necrotic Right Eye in A Domestic Short Hair Cat

Dale Clark, Sabita Rakshit and Kabita Roy

Milford Veterinary Clinic (MVC), 110 Canal Street, Milford MI-48381, USA
Dept. of Veterinary Medicine, College of Veterinary Science (NDVSU), Jabalpur, Madhya Pradesh.

Abstract

A 16 year old Domestic Short Hair spayed female cat was brought to the home clinic, MVC for periodic health check and vaccination schedule. On 22 August, 2018, the companion pet was presented with the complaint of anorexia, weight loss, painful mouth with severe stomatitis and left eye dryness with perceptible conjunctivitis. The blood work revealed hyperthyroidism and increased circulatory globulin concentration. The steroid preparation Depomedrol® and antibiotic Convenia® were given. Methimazole® for hyperthyroidism and Tobramycin® eye drops for conjunctivitis were also prescribed. On November 12, 2018 the owner informed about the marked swelling in the pet's right eye. After critical appraisal of the in-house diagnostic radiography and blood panel reports, eye enucleation surgery was scheduled. Referral histopathological evaluation of the necrobiotic tissue did not reveal any neoplastic growth.

In domestic pets, traumatic insult to the eye resulting from infective scratch, ulceration, impact of any sharp object, tumors, or glaucoma may cause excruciating pain, often associated with the loss of vision. For the eyes that are rendered irreversibly blind, or in which intraocular neoplasm (1, 2) is suspected, or cannot be excluded on the basis of clinical findings, enucleation may become inevitable (3). In the surgical procedure, the feline patient's affected eye is carefully excised, and the eye lids are stitched together at the margins. These stitches are removed on day 10-14 post-surgery, or may get embedded inside the eye socket. Minor tissue aberrations are often noticed during the wound healing process, post-surgery. Thus, the eye lids may appear edematous, and exhibit some bruising. A little red-tinged fluid discharge may seep from the site of incision. These generally subside within one week. Effective pain management is crucial. An Elizabethan neck collar is often used continuously - barring the major meals time - to prevent hazardous self rubbing or scratching of the eye area. Because of the loss of one side vision, the pet needs time and compassionate attention to get adjusted to the strange new situation. Post-surgery homecare with proper hygiene and sanitation minimizes the chances of microbial infection. If the eye area remains swollen, and pus is seen in the incised area veterinary medical care is recommended for proper drainage and antibiotic therapy.

If the other eye remains healthy, the companion cat will soon overcome the handicap. Once the facilitated healing is complete, life shall certainly return to normal. Therefore, the functional eye needs extra care, both from the owner and the attending veterinary practitioner.

Case History

Ivy Le Pard, a 16-year-old DSH spayed, 3.03 kg, female cat was presented to the home clinic, MCV for periodic health check and vaccination protocol. Perusal of the office records showed that on 4 February, 2018, the pet was presented with the complaint of loss of appetite, weight reduction and slight dehydration. CBC and total T4 hormone assay were done in the in-house diagnostic facility. Lactate-Ringers I/V infusion, admixed with Polyflex® 0.5 ml and vitamin B12, was prescribed. On the next visit to the clinic on 22 August, 2018 the lady owner complained about the patient not eating well with painful mouth and left eye issues. Physical examination revealed severe stomatitis and conjunctivitis in the left eye. Some pre-molar and molar teeth were missing. Blood analysis revealed hyperthyroidism (total T4 titer 7.3 µg/dL, reference interval 0.8-4.7 µg/dL). The cat was kept on Methimazole® regimen for hyperthyroidism. Further, Depomedrol® and Convenia® were given S/Q. For stomatitis, antibiotic Clindamycin was given. For the left eye, topical Tobramycin was advised, bid for 10 days. Special food (Hill's A/D) was prescribed for the nutritional support. On November 12, 2018 the owner informed that Ivy was having a prominent swelling in the right eye with increased pain perception. Further, the eye was edematous with the eyeball bulging out (glaucoma) with perceptible corneal ulceration. Radiographs of the whole body including the skull were taken. The tentative clinical diagnosis was suspected neoplastic eye. The in-house blood analysis report revealed anemic condition (low TEC and Hb concentration). Notably, the patient's thyroid status was normalized with Methimazole. Enucleation of the blind right eye of the feline patient

was scheduled with the formal consent of the well-informed client.

Diagnostic panel

I. Blood work

Table 1. Patient's pre-surgery hematological profile* (10 AM on 13.11.2018.)

Parameter (units)	Value	Reference Interval*	Status
TEC (1x10 ⁶ /μL)	3.03	6.54-12.2	Low
Hematocrit (%)	11.5	30.3-52.3	Low
Hemoglobin (g/dL)	3.6	9.8-16.2	Low
MCV (fL)	38.0	35.9-53.1	Normal
MCH (pg)	11.9	11.8-17.3	Normal
MCHC (%)	31.3	28.1-35.8	Normal
RDW (%)	30.2	15.0	High
Reticulocyte (%)	20.3	3.0-50.0	Normal
Reticulocyte-Hb (pg)	13.8	13.2-20.8	Normal
TLC (1x 10 ³ /μL)	13.98	2.87-17.0	Normal
Neutrophil (%)	84.7		
Lymphocyte (%)	10.4		
Monocyte (%)	2.8		
Eosinophil (%)	1.9		
Basophil (%)	0.2		
Neutrophil (1x10 ³ /μL)	11.84	2.30-10.29	High
Lymphocyte(1x10 ³ / μL)	1.45	0.92-6.88	Normal
Monocyte (1x10 ³ /μL)	0.39	0.05-0.67	Normal
Eosinophil (1x10 ³ /μL)	0.27	0.17-1.57	Normal
Basophil (1x10 ³ /μL)	0.03	0.01-0.26	Normal
Thrombocyte (1x10 ³ / μL)	280	151-600	Normal
Auto CBC Analyzer		*In-house Diagnostics	

Treatment

The feline patient (**Fig.1**), suitably prepared overnight in the client's home, was hospitalized on 13 November, 2018. After premedication (acepromazine + butorphanol 0.05 ml S/Q), anesthesia was induced with ketamine-midazolam @ 0.15 ml+0.15 ml I/V. Endotracheal tube (size3) was used. The vital functions (HR 130, RR 16) were automonitored under Oxygen flow rate of 1L with isoflurane gas 2%. Buprenex® 0.09 ml was injected S/Q, and fentanyl patch was placed on the left rear leg. A catheter (22 g I/V) was inserted in the right cephalic vein for lactate ringers infusion. With the patient secured in lateral recumbence, the operative area shaved and sanitized with chlorhexidine scrub was washed with sterile saline solution. The necrotic right eye was carefully enucleated (**Fig. 2**).

The blood vessel in the underlying tissue was carefully ligated. The subcutaneous tissues were

Table 2. Patient's pre-surgery blood biochemical profile* (10.30 AM on 13.11. 2018)

Parameter (units)	Value	Reference Interval*	Status
Glucose (mg/dL)	125	71-159	Normal
SDMA (μg/dL)	15	0-14	High
Creatinine (mg/dL)	1.1	0.8-2.4	Normal
BUN (mg/dL)	17	16-36	Normal
BUN/ Creatinine ratio	15		
Phosphate (mg/dL)	5.8	3.1-7.5	Normal
Total calcium (mg/dL)	8.1	7.8-11.3	Normal
Total protein (g/dL)	8.2	5.7-8.9	Normal
Albumin (g/dL)	2.4	2.3-3.9	Normal
Globulin (g/dL)	5.8	2.8-5.1	High
A/G ratio	0.4		
ALT (U/L)	<10	12-130	Low
ALP (U/L)	32	14-111	Normal
GGT (U/L)	8	0-4	High
Amylase (U/L)	1637	500-1500	High
Lipase (U/L)	593	100-1400	Normal
Total Bilirubin (mg/dL)	0.2	0.0-0.9	Normal
Cholesterol (mg/dL)	172	65-225	Normal
Na+ (mmol/L)	158	150-165	Normal
K+ (mmol/L)	3.4	3.5-5.8	Low
Cl- (mmol/L)	117	112-129	Normal
Total T4 (μg/dL)	3.1	0.8-4.7	Normal
Blood Chemistry Auto Analyzer		*In-house Diagnostics	

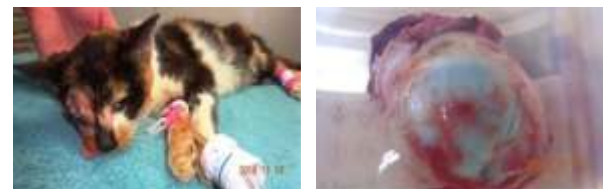


Fig.1 Pre-surgery facial profile Fig. 2. Enucleated right eye

stitched with absorbable sutures in the simple interrupted pattern. The skin margins were sutured with 3-0 non-absorbable nylon in the same pattern. Post-surgical hemorrhage was prevented with bandage, and hypothermia (94° F) was controlled with a warm blanket. Antibiotic Polyflex® (0.5 ml) and Vitamin B₁₂ (0.4 ml) solutions were injected S/Q, separately. Capstar® was given for the control of fleas and ticks. Within one hour the body temperature was restored to near normal (99.4° F). The enucleated eye was submitted for referral histopathology.

II. Pathological Evaluation

(I) Gross: The tissue mass in the enucleated right eye and eyelids was preserved in 70% alcohol for further fixation prior to macroscopic examination and sampling for the pathologist's evaluation in the

referral Marsh field Labs, Cleveland, Ohio, USA. In the report, the ocular tissue (2.0 x 2.0 x 2.5 cm) feels moderately firm. The optic nerve (0.1 cm dia meter) is cut flush with the globe. The diffuse tan white opaque cornea is nearly 1.5 cm in diameter. An irregular raised area (0.5 cm x 0.4 cm) located in the centre of the cornea exhibits a small axial crater. The incised anterior chamber is filled with tan white apparently soft material, adjacent to the cornea. No intact portion of lens / retina is discernible.

(ii) Microscopic: In the ulcerated cornea, notably transmural necrobiosis has involved the entire epithelial layer, stroma with Descemet's membrane, and the endothelium. The degraded tissue reveals the presence of numerous degenerated and some intact neutrophils. The adjoining viable corneal stroma contains granulation tissue with many neutrophils. The residual intact corneal epithelium is hyperplastic. The interior region of eye is packed with fibrillar material: necrotic debris, and neutrophils, both degenerated and intact. Some pigment-laden macrophages, lymphocyte and plasma cells are intermingled with the neutrophils. Scanty fragments of lens fibers are intermixed with the tissue debris. Occasional pale staining bacterial rods and cocci are perceptible. Descemet's membrane is partially detached from the necrotic corneal stroma. No intact lens, retina, ciliary body or iris tissue is visible. The drainage angle contains a large population of inflammatory cells, intermixed with fibrin. The choroid, heavily infiltrated with neutrophils, contains blood vessels which are clearly necrotic and contain intraluminal fibrinous thrombi in a pool of extravagated blood. The inflammatory process extends into the periocular tissues, evidenced by the wide spectrum inflammatory cells, intermixed with reactive fibroplasia.

Diagnosis

The necrotic, suppurative, fibrinous panophthalmitis is apparently the culmination of

severe corneal ulceration and biodegradation foci in the right eye. The feline patient's enucleated right eye is highly inflamed, and the inflammatory process is also involving the periocular tissues. Interior eye chamber tissues are poorly preserved because of inflammation and necrosis. Since no clinical history is forthcoming, it is uncertain whether the panophthalmitis is secondary to corneal ulceration / necrosis with superimposed bacterial infection, or the consequence of an unnoticed traumatic insult to the eye. However, notably there is no evidence of any underlying neoplastic/ malignant lesion.

Discussion

Ocular tumors in the animals and humans are on record (2,4,5). Removal of one eye of the companion animal is understandably of much emotional concern to the pet lover. However, the majority of suffering patients cope up with the situation admirably. Pain perception is highly subjective in the feline fraternity, as in the humans. In the instant case, presumably because of the high tolerance threshold, the clinical syndrome of on-going necrobiotic degradation of the patient's right eye tissues remained undetected continuously for several weeks. However, as soon as the potentially hazardous clinical condition came to light no time was lost to take the pragmatic clinical decision: enucleation of the blind right eye, irrespective of cosmetic considerations, or the apparently anemic status (low TEC, Hb concentration) of the patient with no gross impairment of the hepatic or renal function (**Table 1**). The main concern was to minimize the chances of spread of undetected malignancy to other body parts/ organs, though the pre-surgery whole body survey radiographs (**Fig. 3**) R/L view did not reveal any evidence of metastatic neoplasia.

Ventro-Dorsal view showed some radio lucency in the eyeball area suggestive of pathological eye and needed drastic remedy. It may be noted that

Fig. 3 In-house survey radiographs



enucleation was preferred over evisceration within trascleral prosthetics implantation, since the reported success rate of the latter procedure in cats is lower than in dogs (6, 7). However, post-surgery complications, such as accumulation of tears in the orbital cavity, attributable to persisting residual lacrimal gland tissue, need to be addressed adequately (8). Mild edema, inflammation and bruising around the sutured tissue margins are quite possible. Occasionally, blood-stained tissue fluid may trickle from the nose. Epistaxis subsides in 2-4 days. In the instant case, recovery was uneventful, and the skin sutures were removed on day 14 post-surgery.

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Introducing

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Congenital Deafness in Cat

Anshu Ahlawat, Ankita Verma, Odedra M D, Gamit V D and Savalia KB

Dept of Animal Genetics & Breeding, Dept of Livestock Production Management, C V S & A.H., and Polytechnic in Animal Husbandry, J A U, Junagadh, Gujarat.

Introduction

Cats like humans have five senses, however, these senses are far better developed and more acute as compared to humans. They have high degree of visibility in the dark, can hear higher pitched sounds, and have well developed sense of touch and smell. However, the sense of taste is better developed in humans as compared to cats.

Deafness is the inability to hear sounds. Cats can experience total or partial deafness in one or both ears. It can be acquired, meaning it is developed later in life due to factors like disease, trauma, or toxicity. The inability to hear can be congenital, meaning it occurs at birth. It may be caused by degeneration of inner ear. The first report describing the relationship between white pigmentation and deafness in cats was presented in the first half of the nineteenth century (1). Since then, the interest in this relationship has been increasing steadily, resulting in numerous studies (2, 3, 4, 5, 6), especially because the congenitally deaf, mixed-breed white cat has been used as an animal model of human deafness.

Breeds at risk

Any mammal can fail to develop functional hearing. In many species, such as domestic cats and dogs, there is a higher incidence of deafness in animals with a white coat. Deafness can occur in white cats with yellow, green or blue irises, although it is mostly likely in white cats with blue irises. In white cats with mixed-coloured eyes, it has been found that deafness is more likely to affect the ear on the blue-eyed side. There are some breeds of cats viz; Persians, Angoras, and Ragdolls which have shown to have higher incidences of deafness.

Genetics of White Cat Deafness

In cats, inherited congenital deafness is seen almost exclusively in white coated individuals. The deafness is caused by degeneration of the auditory apparatus of the inner ear and may affect one ear (unilateral) or both ears (bilateral).

The gene responsible is an autosomal dominant gene termed W (for White). This gene has pleiotropic effect, i.e. it has more than one effect, responsible for white coat color, blue eyes and deafness. However, while the gene has complete penetrance for white coat colour (all cats that carry



the gene will have a white coat), it has incomplete penetrance for blue eye colour and for deafness (but these two are strongly linked). Thus deafness is strongly linked to the white coat colour and blue eye colour.

White cats with blue eyes are at higher risk because of a genetic mutation that not only affects coat and eye color but also the development of structures within the ears. The inheritance of this abnormal gene (called "W") is complex, so not every cat which carries it is deaf. Also, white hair and blue eyes can be caused by other genes that do not increase the chances of deafness.

Symptoms

When a cat is deaf from birth or begins to go deaf later in life, it compensates for this by using its other, better developed senses more, and it is thus sometimes hard to tell whether it really is deaf. While living with a pet white deaf cat, you can make out to some degree by making a key sound or clap your hands when she is not looking at you. Deafness is irreversible and progressive however some early signs of deafness may include

- A very deep and sound sleeper
- No longer afraid of loud appliances
- A lack of response to everyday sounds that

would ordinarily elicit a response

- Not hearing your footsteps when you come close
- Meowing may be more often and more loud as they cannot regulate their own volume
- Failure to respond when called or spoken off

Now a days, white cats are being checked for deafness e.g. using BAER (Brainstem Auditory Evoked Response) testing involving a clicking sound being directed into the ear while computers measure the brain’s electrical activity in response to the sound.

Treatment

Treatment of deafness in your cat will depend on the type of deafness they are suffering from and its cause. Congenital deafness and many causes of nerve deafness are untreatable, and the inability to hear will be permanent. There is not treatment available for deafness caused by the “W” gene

Management of White Deaf Cats

It is more common to find white cats as pets due to selective breeding (human preference and intervention). Many cat breeds are known to have the white coat gene and can, therefore, produce deaf white individuals. These breeds do not allow deaf white cats to be bred from keeping them safe at home which would avoid situations that they need to use auditory cues. White cat may be prone to

accidents if he or she will not be able to hear threats, such as from roaming animals. Also, these cats are to be taken out with leash, as once let loose they shall not be able to hear owners call to come back. Affected cats can live long and happy lives if they are kept indoors.

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Maternity Care in Bitches - Guidelines for Pet Parents

R Rajalakshmi, U S Kalyaan, S Rangasamy, P Selvanathan and K Krishnakumar
Dept. of Veterinary Gynaecology and Obstetrics, MVC, TANUVAS, Chennai, Tamil Nadu.

Whelping is the process of a dog giving birth to puppies. Most dogs are capable of whelping on their own without difficulties. But a close eye on your dog throughout her late pregnancy and labour is always recommended. Having a good idea of what's normal for a dog in labour will allow us to spot signs of trouble early.

How Long Are Dogs Pregnant?

Dogs are pregnant for approximately 63 days, or about two months and this may be a bit inaccurate. This being said, if your dog is a couple of days over her due date you should get her checked out by the veterinarian. By the end of the second month and the start of the third, the puppies are ready to be born, which means you need to be prepared for their delivery.

Checklist for Whelping

- Newspaper to line the whelping box during delivery for easy clean up
- Non-skid bath mats for bedding after whelping is done
- Dry, clean towels to clean the puppies
- Paper towels to help with clean up
- Thermometer to check your dog's temperature before whelping
- Unwaxed dental floss to tie off the umbilical cords
- Clean scissors to cut the umbilical cords
- A heating pad or hot water bottle to keep the puppies warm (be careful of it not being too hot)
- Iodine to clean the puppies' abdomens after the cord is cut and dab on the end of the cut umbilical cord
- A baby scale in ounces
- Your veterinarian's phone number and the number of a nearby pet clinic

Keep these supplies in a clean, easy-to-access location.

Dog Whelping Box

A whelping box is merely a place for your dog to have her puppies comfortably. Since this will be where the pups spend the first weeks of their lives, it should have 12 to 18 - inch sides to keep the puppies safely inside.

- The box should be easy to clean. After all, your dog will be giving birth in it and then the puppies will be living there. Not only is cleanliness important to



Whelping Bitch

keep the current litter of puppies healthy, but you may also want to use it again for another litter.

- There should also be some kind of pad on the floor to make it comfortable for your dog since she will be spending a lot of time in the box.
- The whelping box should be large enough for your dog to lie down and stretch out. There should also be enough space that she has room to turn around without stepping on a puppy and lie down to nurse the puppies easily. If the box is too big, a puppy can get pushed away from the mother and become chilled.
- Lastly, the box should be convenient so you can easily care for your dog and her puppies.

Signs of a dog going into labour

Common behavioural signs prior to whelping includes:

- Restlessness
- Lack of appetite



Preparing for whelping

- Nesting
- Panting
- Excessive urination
- Clinginess

Warning signs of labor in dogs

- Many pregnant dogs start to pant heavily, and her temperature will drop from a normal temperature (100 to 102.5°F) to 99°F or even lower shortly before going into labor.
- Approximately 24 hours after this temperature drop, she will whelp, and you will be the proud owner of a new litter of puppies.

What are the stages of dog labour?

There are three stages of labour in dogs:

- The start of contractions
- Delivery of puppies
- Delivery of the placenta

Stage I



Greenish black discharge in whelping bitch

- Averages 6-12 hours but may last for 24 hours.
- Bitch appears restless, nervous and may see to shiver, pant, vomit, chew, scratch at the floor.
- Most dogs seek seclusion and / or digs / tears the material to create nest / bedding for puppies.
- At this stage privacy and whelping area / box must be provided to the dog.

Stage II and II

- Stage II ends with expulsion of the foetus and Stage III ends with expulsion of the placenta.
- Bitches may deliver pups over a period as short as few hours to as long as 24-36 hours.
- Contractions are usually visible, and the bitch is either on her side or in a squatting position.
- With passage of each pup, either the bitch frees the pup by biting and / or licking the membrane away or must be removed by the owner.
- Active straining for > 30mins from Stage II initiation

and birth of first puppy is worrisome and needs veterinary assistance.

- A lag of 30 mins to 1 hour with straining and a lag of >4-6 hours between births of subsequent pups is worrisome and needs veterinary assistance.
- A disturbed, frightened, or nervous bitch may actually interrupt whelping.
- The placenta is usually passed within 5 to 15 minutes of the birth of each puppy.

Post-whelping routine

- Eating of placentas by the bitch should not be encouraged.
- Vomiting of the placental material is common.
- The bitch should lick each new-born vigorously to remove all membranes from the face and to promote respiration. If this does not occur within 1 to 3 minutes, the owner can intervene.



Foetus and placenta with whelping bitch

- A new dry soft towel must be used to hold pups and all membranes should be removed by rubbing the puppy with the other end of the towel.
- Fluids can be removed from the mouth by suction, usually using a soft, blunt-ended rubber air bulb.
- The puppy can be cupped in the hands of owner (with its head at the fingertips and its tail at the wrist) and the arms are swung in an up and down motion (as if chopping wood) to promote respiration and clean the respiratory tract.
- The bitch severs the umbilical cord with her teeth. If she does not do this, the owner can use thread, tying two knots in the cord. The first knot should be at least 1 inch from the puppy, and the second is an additional ¼-inch away.
- Clean new scissors are used to cut between the knots, and the severed end is dipped into a mild antiseptic such as tincture of Iodine or Betadine.
- The pups should definitely be left with the bitch, except in unusual circumstances, and handled as little as possible.
- Some bitches nurse new-born puppies while delivering subsequent puppies; others do not.

Clinical Factsheet on Rabies

H Prabhavathy

Madras Veterinary College, TANUVAS, Chennai, Tamil Nadu.

- Rabies is a viral disease that affects the central nervous system of mammals, including humans. Rabies is a disease of warm-blooded animals.
- It causes inflammation of the brain, leading to death. Once the first clinical symptoms appear, rabies is almost always fatal.
- The main vector of rabies in India is dog in over 95% of human cases and transmitted most commonly through the bite of a rabid animal
- Rabies is 100% fatal but it is preventable through systematic vaccination.
- The incubation period (from initial exposure to clinical symptoms) may range from two weeks to many months. It depends on a number of factors, including the strain of rabies and the location of the bite.

Symptoms in Humans

- Incubation period - Usually 20-90 days, but may be as short as nine days. Very rarely as long as several years.
- Fever, anorexia (poor appetite), nausea, vomiting headache malaise, lethargy pain or paresthesia (numbness or tingling) at site of the bite.
- Disorientation, hallucinations, seizures, neck stiffness, hydrophobia or aerophobia (intense fear of water or air caused by pain from tightening of muscles in the throat), paralysis and weakness
- Coma, death.

Hydrophobia in Human



Two Forms of Rabies:

Dumb Form of Rabies

- Domestic animals may become restless, depressed and try to hide in isolated places.
- Abnormal behaviour, salivation, weakness of the

back legs and paralysis. The dog is hyper-excitable, aggressive and bites even imaginary objects.

- Wild animals may lose their fear of humans and appear unusually friendly.
- Wild animals that usually only come out at night might be out during the day.
- Animals may have paralysis. Areas most commonly affected are the face or neck (which causes abnormal facial expressions or drooling) or the hind legs.

Furious Form of Rabies

- Animals may become very excited and aggressive.
- Periods of excitement usually alternate with periods of depression.
- Animals may attack objects or other animals. They may even bite or chew their own limbs.
- Unable to drink water due to painful spasms and paralysis of the muscles which assist swallowing. This condition causes salivation, since the dog cannot swallow saliva. In the last stage, paralysis causes respiratory failure, leading to coma and death.

Furious Form of Rabies



Hyper Salivation in Rabid Dog



After Exposure to Rabies

- Wash the wound for at least 10 minutes with soap and running water. It removes 94.4% of the viral particles.
- Disinfect the wound with Dettol, Betadine or spirit.
- Human treatment consists of a dose of rabies-immune globulin administered as soon as possible after exposure.
- Start post exposure vaccination course. If any unknown dog makes an unprovoked attack, take the full course. If a known dog makes a provoked attack, take 3 vaccinations. In either case the dog should be observed for at least 14 days. This is because in dogs, death generally occurs within 10

to 12 days after the virus reaches the salivary glands.

Types of Contact

- Category I – touching or feeding animals, licks on the skin
- Category II - nibbling of uncovered skin, minor scratches or abrasions without bleeding, licks on broken skin
- Category III – single or multiple transdermal bites or scratches, contamination of mucous membrane with saliva from licks; exposure to bat bites or scratches.
- For category I: no treatment is required
- For category II: immediate vaccination
- For category III: immediate vaccination and administration of rabies immune globulin are recommended in addition to immediate washing and flushing of all bite wounds and scratches.

Don't Do in Dog Bitten Wound

- Religious practices like turmeric powder application in the wound do not help.
- Do not bandage or suture the wound unless recommended by a medical practitioner.

Pre-exposure Vaccination Need

Pre-exposure vaccination schedule of 0, 7 and 21 or 28 day in deltoid area of the arm for adults and Anterolateral area of the thigh is recommended for children aged less than 2 years.

Treatment for Rabies

- There is no treatment for rabies once clinical signs appear.
- All domestic animals should be vaccinated in

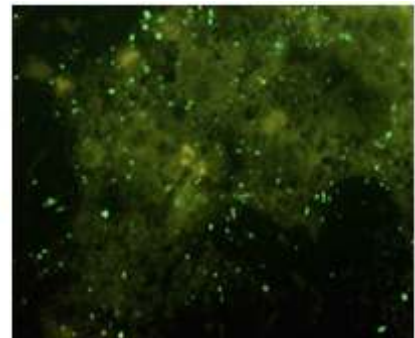
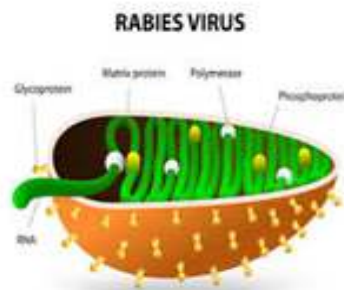
areas where rabies is endemic.

- The wound is washed thoroughly with soap and water.
- Dog bitten wound should not be sutured.
- Post-Exposure vaccination

Prevention

- Dogs and cats are up-to-date on their rabies vaccinations.
- Vaccination for all cats, dogs and beginning at three months of age. Vaccinated pets serve as a buffer between rabid wildlife and humans. If vaccination is necessary before 3 months of age, then a booster dose shall be administered.
- Advise the children to tell about bitten or scratching by an animal.
- Do not approach, handle, or feed wild or stray animals.
- Do not leave pets outside unattended or allow them to roam free.
- Cover garbage cans tightly and do not leave pet food outside that will attract wild and stray animals.
- Teach children to stay away from wild animals or animals that they do not know.
- Wear gloves when handling an animal if it has been in a fight with another animal.
- Animal Birth control Policy.

Educate the students about rabies at school level and College campus.



COVID - 19: The New Corona Virus and Dogs

A R Ahlawat

College of Veterinary Science & A.H., Junagadh Agricultural University, Junagadh, Gujarat.

Introduction

Several coronaviruses have caused serious problems in humans and animals in the past two decades. The best known examples are severe acute respiratory syndrome coronavirus (SARS-CoV), Middle East respiratory syndrome coronavirus (MERS-CoV) and porcine epidemic diarrhea virus (PEDV) (1). In early March 2020, the World Health Organization declared that the Corona Virus disease 2019 is a global pandemic (2). COVID-19 is a new strain of coronavirus, and scientists are still collecting information and conducting research on the virus. In current scenario, people are not only worried about their own health but also worried about health of their pets

Types, Host and Distribution

Coronaviruses are enveloped non-segmented positive-sense RNA viruses belonging to the family *Coronaviridae* and the order *Nidovirales* (3). The coronavirus gets its name from the crown-like spikes on its surface ("corona" in Latin translates to "crown"). The genus coronavirus is composed of at least three groups that cause mild to severe enteric, respiratory, or systemic disease. The host range is characteristic for each group of the virus. Coronaviruses belong to the family *Coronaviridae*. Alpha- and beta-coronaviruses usually infect mammals, while gamma and delta coronaviruses usually infect birds and fish. *Canine coronavirus*, which can cause mild diarrhea and feline coronavirus, which can cause feline infectious peritonitis (FIP), are both alpha-coronaviruses.

Coronavirus is a family of viruses that are common in people and animals. Coronaviruses are common in several species of domestic and wild animals, including cattle, horses, dogs, cats, ferrets, camels, bats, and others (4). Although not common, coronaviruses can be transmitted from animals to humans. Bats can be reservoir hosts for viruses which can cross species barriers to infect humans and other domestic and wild mammals.

Transmission

Currently there is limited evidence that companion animals can be infected with SARS-Cov-2 and no evidence that pet dogs or cats can be a source of infection to other animals or to humans resulting in

COVID-19. This is a rapidly evolving situation and information will be updated as it becomes available (5).

Coronaviruses spread from person to person through droplets released when people who are infected cough or sneeze. It is also possible that a person can get COVID-19 by touching a surface or object that has the virus on it and then touching their own mouth, nose, or possibly their eyes., but appears to be a secondary route. Smooth (non-porous) surfaces (counter tops, door knobs) transmit viruses better than porous materials (paper money, pet fur) because porous and fibrous materials absorb and trap the virus, making it harder to contract through simple touch.

First known case of human-to-animal transmission of the novel coronavirus

In case of human to animal transmission, a Hong Kong coronavirus patient's pet dog (17 year old Pomeranian) tested "weak positive" for a "low level" of the virus after oral and nasal tests(6). The dog, which did not exhibit any symptoms, was put into a two-week quarantine, after which it tested negative, however, the dog died after a few days(7). It was also reported that the dog may not have actually been carrying the virus but tested positive due to "environmental contamination". The death of the dog may also have been due to other health ailments as the dog was quite old. In an animal with no clinical signs of disease, it's hard to say what this means. It was a single case, and we learned that we need to do a lot more research into the potential of the human SARS-CoV-19 virus to infect animals (8). However, it is so important to note that viruses can sometimes infect a species but not cause illness in that species, nor become transmissible to others.

Vaccination

The canine coronavirus vaccines available in some global markets are intended to protect against enteric coronavirus infection and are not licensed for protection against respiratory infections. Many dogs, for example, are vaccinated for another species of coronavirus (*Canine Coronavirus*) as puppies. However, this vaccine does not cross protect against COVID-19(6). Currently, there are no COVID-19 vaccines available for humans or

animals.

Dos and don'ts

It is important to have plans for your pets as well as yourself. The following are some measures to be adopted to keep our pets safe. Actions to prevent the spread of viruses include:

- Always wash your hands many times with soap and water for at least 20 seconds.
- When you don't have access to soap and water, use a hand sanitizer containing at least 60% alcohol.
- Avoid touching your eyes, nose and mouth.
- Avoid contact with sick people and stay home, or, practice quarantine if you're sick.
- Cover coughs and sneeze.
- When in some public place, stay at least 6-7 feet away from other people.
- Follow all public health or government requirements to avoid gatherings of multiple people. Follow stay at home/shelter in place orders.
- Ensure that your pets are wearing a collar and ID tag.
- Pet owners should avoid contact with animals they are unfamiliar with and always wash their hands before and after they interact with animals.
- You should restrict contact with pets and other animals while you are sick with COVID-19. If you are sick, have another member of your household to take care of handling of your pet.
- Keep an emergency pet medical kit ready.
- If you are sick with COVID-19, and you believe your pet or service animal is ill, you should seek assistance from your veterinarian.
- Stock of food and medicine for your pet in case

movement is necessary.

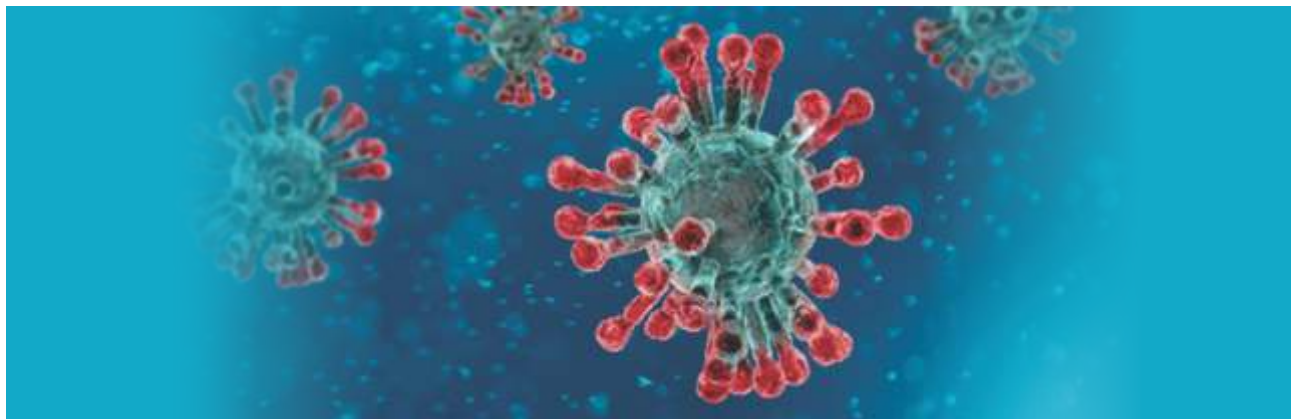
- If you know older/elderly people or others who cannot go out due to health issues, we encourage you to check in with them to make sure they have necessary food, medications and supplies, including for their pets.

Summary

This being a new strain of coronavirus, scientists are still collecting information and conducting research on the virus. The author also recognizes that not all recommendations will apply to all areas or all regions at all times. This is a rapidly evolving situation and information will be updated as it becomes available.

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Traumatic Hyphema in Kathiawari Foal: A Case Report

Vijay L Parmar, Amit Prasad, P G Dodiya, K K Murabiya and J S Patel

Department of Veterinary Medicine, College of Veterinary Science and Animal Husbandry, Junagadh, Gujarat.

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 showed 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

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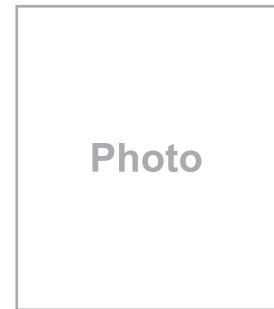
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