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SURGICAL MANAGEMENT OF PERINEAL HERNIA IN CANINES

Introduction

Perineal hernia in canines is usually seen in uncastrated dogs above 8 years and results from weakening of the the perineal muscles i.e. the Lateral coccygeal muscle and the external anal sphincter, resulting in rectal sacculation and herniation of prostrate, fat, bladder, or intestines in advanced cases. The exact causes is unknown but may be related to chronic tenesmus (from constipation / prostatomegaly) or hormonal imbalances.

Case history and clinical observation

A Pomeranian male dog aged about 8 years was referred to the Govt. Veterinary Hospital, Tonca with a complaint of swelling on the right lateral side of the anus since last I month with straining and inappetance. Clinical examination of the uncastrated dog revealed a reducible swelling on right lateral side of the anus. The palpation of the swelling revealed fluid thrills and tubular mass feeling. It was decided to operate after the blood parameters were found to be within normal range.

Surgical Proceedure

The dog was premedicated with Xylazine hydrocloride @ 1 mg/Kg I/V and general anesthesia was induced with 5 % Thiopentone inj I/V, to effect. The area around the anus was prepared for aseptic surgery, and anal purse string sutures were put to prevent soiling of the operative site. The dog was positioned in sternal recumbecy with raised hindquarters.

Fig.1

A crescentric incision of 5 cm length was done, just lateral (right) to the anal spincter on the skin pouch of the hernia. The hernial contents i.e. the bladder, omental fat were reduced into the pelvic cavity and the hernial ring (pelvic diapraghm) was sutured using Vicryl No 1 suture. The dorsal aspect of the hernia was sutured first, by using mattress suture between the coccygeus musle and external anal sphincter muscle as shown in the (Fig.2). The medial surface of the sacrotuberous and the obturator internus muscle was then united with the border of sphincter muscle thus closing the hernia ring completely. The skin was sutured with silk mattress sutures as shown in Fig. 1

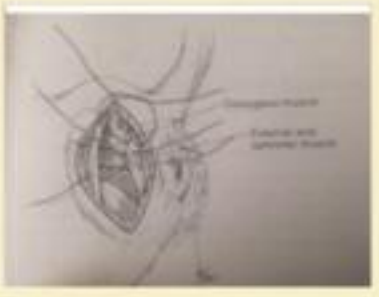

Fig.2

The dog was then castrated by pre-scrotal method and treated with Cefataxim inj 500 mg and tetanus toxoid inj. followed by oral post operative antibiotics of Amoxicillin Sulbactum combination for 6 days. The skin sutures were removed on the 8th Post operative day with no history of recurrence.

Summary

Perineal hernia was successfully treated in a Pomeranian male dog using the technique described above. Recurrence is directly correlated with the skill of the surgeon and the severity of the hernia. Castration helps to decrease recurrence in cases of prostatomegaly.

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PRODUCE PIG FOR POLLUTION CONTROL AND HUMAN NUTRITION

Very important role of pig in human life is control of environmental pollution by scavenging the biological waste. Pig converts biological waste material to quality proteins in form of pork. These are the sturdy animals requiring minimal basic needs like shelter in adverse environment, few vaccines and food. This is the species maintained by lowest strata of the society.

In Goa most farmers feed their pigs with freely available food materials like hotel/kitchen waste, bakery waste, garbage from vegetable market, broiler offal etc. It is suggested that these unconventional feeds should be fed as the partial replacement for the ingredients in standard ration to economize the pig production.

Meat has become an integral part of human food to meet the essential nutrients, as proteins. For a balanced diet a good biological availability meat is preferred in modern day era. The per capita consumption of meat in India is only 14g per day as against actual requirement of 125g. Non availability of quality meat and its exorbitant prices have restricted meat consumption. Increasing the meat production through intensive rearing of various meat animals will help to meet the ever growing demand of balanced human diet. In modern era and educational status social taboos are also getting relegated with need of quality proteins.

Pigs have a rapid growth rate, high reproductive efficacy, require comparatively less space, and can be reared on small scale in backyards or as commercial units.. A major advantage of pig farming is that they can be fed on fibrous low quality agro byproducts and material that are not suitable for human consumption leading to low production cost. Hence pig rearing can be a lucrative source of income for rural farmers in India.

In spite of above facts regarding pig rearing in India many youth from different states are coming forward to establish modern commercial pig units. These units are of tow type one is breeding unit which maintain parent stock ,produce the piglets and sale for fattening. Another group is majority where piglets are purchased and fattened for pork. To increase productivity and profitability there is a need of improved pig husbandry like suitable breed, feeding practices, advance technologies like breeding through Artificial Insemination sound diseases control and hygienic production can improve pig production of state as well as profit of individual farmer.

Among all the domestic animals pigs are the most prolific animals with shorter generation interval. A sow can be bred as early as 8-9 months of age and can farrow twice in a year. They produce 6-12 piglets in each farrowing. Pig products such as pork, bacon, ham, sausages, lard, etc. are increasingly in demand both for local consumption and for the export. Pig industry has a special significance as an enterprise in developing countries which raise pigs mainly by scavenging as traditional occupation in the rural areas.

Looking to the meager availability and tremendous demand of animal protein diet in India, it is felt that such demand could substantially be met by improving and multiplying pigs, mainly because of their prolificacy, faster growth, efficiency of feed conversion and shorter generation intervals. Efficiency and cost of production for a swine enterprise are directly associated with reproductive efficiency of the breeding herd.

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RABIES (HYDROPHOBIA, LYSSA)

Etiological agent: Rabies is caused by a bullet-shaped, Lyssa virus belonging to the family Rhabdoviridae. Rhabdo viruses are enveloped viruses and hence are relatively easily destroyed by common household soaps and detergents.

Epidemiology: Rabies can be found on all continents of the world except Australia and Antarctica. It is ordinarily a disease of bats and carnivores, including the domestic dog and cat and many wild species. Despite the availability of excellent human and animal rabies vaccines, rabies remains a special cause of concern among human populations, especially in the developing world.

Occurrence and Transmission

Rabies is maintained in nature by wild and domestic carnivores and by certain other wildlife species. In Latin America, vampire bats are particularly notorious for spreading rabies. Among domestic species, only dogs and cats are important carriers of the infection. In most developing nations today, dogs remain the primary reservoir of the disease and the principal source of human exposure. Rabid animals excrete vast numbers of rabies virus particles in their saliva-a fact that accounts for the primary means of rabies virus transmission, the bite of an infected animal.

Pathogenesis

The incubation period-the time between exposure to the rabies virus and the development of signs quite variable, ranging from one week to one year. Most of this variability appears to reflect the length of time the rabies virus spends within muscle cells at the site of the bite, prior to gaining access to the nervous system. Once the virus has entered nerve endings, however, it advances relentlessly up the nerve bodies until it reaches the spinal cord and eventually the

brain. From there, it can spread to other tissues important in transmission of the virus-the salivary glands, respiratory system, and digestive tract. To date, the actual mechanism by which the virus produces locomotion and cerebral derangement, with eventual death of the host, remains unclear.

Clinical Signs In Animals

Two principal forms of rabies are recognized:

1) **an excitatory or "furious" form**
2) **and a paralytic or "dumb" form**

In actuality, most rabid animals exhibit some manifestations of both forms. The paralytic form of rabies always represents the terminal or end stage; however, some animals may die during the convulsive seizures of the furious stage without exhibiting the final stage. Some will show few or no signs of excitement, the clinical picture reflecting instead the effects of paralysis.

During the furious stage, which lasts variably for one to seven days, affected animals become wild and aggressive. Rabid cats are extremely dangerous animals because of their viciousness and quickness of action. Rabid animals frequently snap at imaginary objects and may attempt to bite any animals or humans that approach them. If restrained, an animal may chew viciously on metal chains or the bars of its cage. It may break its teeth, lacerate its mouth and gums, and drool ropy saliva tinged with blood.

Within a short time, these signs give way to those of the final or paralytic stage, which lasts only for a day or two. The paralysis usually appears first in the muscles of the head and neck, the most characteristic sign being difficulty in swallowing. Signs of localized paralysis are quickly succeeded by more generalized paralysis, with death following usually within two to four days of onset.

For both animals and humans, rabies is an inevitably fatal disease once clinical signs have appeared (only three human survivors are documented in the medical literature). Therefore, utmost care must be taken if one suspects that a pet has been exposed to the rabies virus.

For both animals and humans, rabies is an inevitably fatal disease once clinical signs have appeared (only three human survivors are documented in the medical literature). Therefore, utmost care must be taken if one suspects that a pet has been exposed to the rabies virus.

Public Health Significance

The signs and course of rabies in humans are similar to those seen in animals. Both excitatory and paralytic symptoms may be manifested. The incubation period, as in animals, is quite variable from about two weeks to as long as one year-but on the whole it averages between three and six weeks. The course of the disease is short-only a few days-and the mortality rate is essentially 100 percent. Patient feels pain and irritation in region of wound. There is extreme sensitivity to light and sound and dilation of pupils. There is excessive salivation and spasms of deglutitory muscles leading to hydrophobia.

Diagnosis

A definitive diagnosis of rabies can be made only by laboratory examination of brain material from an affected animal. Any wild or domestic mammal that has bitten a human being and is showing signs suggestive of rabies should be humanely destroyed, and the head submitted to a qualified rabies laboratory for diagnostic testing. In addition, any bat or wild carnivore, regardless of signs manifested, that has bitten a human being should be destroyed immediately and the brain examined for the presence of the rabies virus (this latter action is necessary because of the variable period of salivary virus-shedding that can occur before clinical signs appear). Any healthy domestic animal that has bitten a human being should be confined for at least ten days and observed for the development of clinical signs of rabies.

Laboratory diagnosis of rabies:

1. Immunofluorescence microscopy, the most rapid and accurate method, in which slides of brain tissue are examined for the presence of the rabies virus using special antibodies and a fluorescent microscope;
2. Histopathology, in which sections or smears of brain tissue are examined for the presence of Negri bodies, intracellular inclusion bodies seen in many (but not all) cases of rabies;
3. Mouse inoculation, which is frequently used to confirm positive results or to investigate further suspected cases that have proven negative by other methods.

Treatment

Because of the potential risk of exposing susceptible humans to the rabies virus, treatment of animals suspected of having rabies is strongly discouraged. Treatment of humans exposed to a rabid animal, however, must be aggressively applied. Treatment of humans consists of thorough flushing and cleansing of the bite wound with soap and water (the importance of this simple step cannot be overemphasized); administration of rabies immune globulin (rabies virus antiserum) to exposed individuals who have never been vaccinated against rabies; and administration of the human diploid-cell rabies vaccine in five doses, given on days 0, 3, 7, 14, and 28 postexposure.

Prevention

Effective rabies vaccines are available for use in domestic animals. Mass immunization of dogs has been employed for many years to control the spread of rabies by creating an "immunological barrier" between wildlife reservoirs of rabies and human populations. It is recommended that all dogs and cats be vaccinated for rabies at three months of age and revaccinated as required by vaccine specifications. At present, there are no rabies vaccines licensed for use in wild animals; however, genetically engineered rabies vaccines are being tested in selected wildlife populations.

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AVIAN INFLUENZA (HIGHLY PATHOGENIC)

Disease from highly pathogenic avian influenza is also known as fowl plague, fowl pest, peste aviaries or fowl disease.

Influenza Virus

Influenza viruses belong to the family *Orthomyxoviridae*. They are classified into three main types. Influenza type A viruses affect multiple species. Influenza types B and C both infect humans, but type C is also known to infect swine.

a) Influenza A

1. Influenza type A infects multiple species. Several human influenza strains are type A while all avian strains are type A.
2. They are considered the most virulent group, although not all strains cause clinical disease.
3. Type A influenza viruses are classified into subtypes based on two surface antigens known as hemagglutinin (H) and neuraminidase (N), sometimes also referred to as HA and NA respectively.
4. Influenza A viruses infects a variety of species.
5. Human infections that occur yearly are most often the result of H1N1, H3N2, and H1N2 influenza A viruses.
6. Human infections by other subtypes are sporadic, have not sustained human to human transmission, and are rare to date.
7. Avian influenza viruses of most concern to the poultry industry are the H5 and H7 subtypes.
8. These two subtypes are associated with high pathogenicity in domestic poultry. These viruses are of concern to human public health also because they have infected and caused serious disease in humans. Although the infections have been rare they are concerning because the human population is immunologically naïve to those subtypes.

Avian Influenza

Avian influenza only includes type A viruses and described based on their pathogenicity. Genetic features and/or severity of disease in poultry determines whether the virus is classified as low pathogenic (LPAI) or high pathogenic (HPAI) avian influenza. Low pathogenic avian influenza (LPAI) includes viruses in all H1 to H16 subtypes. On the other hand, highly pathogenic avian influenza (HPAI) have traditionally been either H5 or H7 subtypes. H5 and H7 LPAI viruses do exist and are of concern because they can mutate into a HPAI.

HISTORY

Avian influenza was first identified in Italy in 1878.

1. The first US cases of highly pathogenic avian influenza (HP AI) were reported in the U.S. in 1924-25 and 1929.
2. Milder disease caused by AI viruses were recognized in the middle of the twentieth century. Today these AI viruses are termed non-highly pathogenic or mildly pathogenic, designated MP AI.
3. In the 1970's surveillance for Newcastle disease virus showed that migratory waterfowl were asymptomatic carriers of AI. Since then it has been shown that wild waterfowl (especially ducks and geese) and other aquatic birds are the original reservoir of all influenza viral genes. Avian influenza viruses have caused epizootics of respiratory disease in mink, seals and whales
4. In India first outbreak of avian influenza occurred in Maharashtra in 2006.

Epidemiology

Epidemiology: Birds

1. Migratory waterfowl are widely considered to be the reservoirs of avian influenza virus.
2. Feces and respiratory secretions contain large amounts of virus, which can infect a new host through the conjunctiva or respiratory tract.
3. Avian influenza virus can spread by aerosols when birds are in close proximity, and might also be transmitted through shared drinking water.
4. The virus appears to be present in eggs laid by infected hens, but they are unlikely to survive and hatch.
5. Fomites and infected birds can transmit the disease between flocks. Airborne dissemination may be possible as well as movement of infected poultry. In experimental studies AI viruses can be excreted in the feces and maintained in the environment and can re-emerge after a significantly stressful event.
6. Once a flock is infected, it should be considered a potential source of virus for life.

Epidemiology: Pigs

1. Pigs have also been infected with avian influenza viruses.
2. One of the concerns with this, is that pigs have receptors for both avian and human influenza viruses (as well as swine influenza viruses).
3. Pigs can serve as "mixing" vessels for these various subtypes and serve as a site for genetic reassortment (or "mixing") of these viruses.
4. The alterations may develop a novel virus more virulent or transmissible than the original subtypes.

Transmission

Animal Transmission

1. Migratory waterfowl are widely considered to be the reservoirs of avian influenza virus. Feces and respiratory secretions contain large amounts of virus, which can infect a new host through the conjunctiva or respiratory tract.
2. Avian influenza virus can spread by aerosols when birds are in close proximity, and might also be transmitted through shared drinking water.

3. The virus appears to be present in eggs laid by infected hens, but they are unlikely to survive and hatch.
4. Fomites and infected birds can transmit the disease between flocks. In one outbreak in Pennsylvania, the virus may have been spread by garbage flies.
5. Airborne dissemination may be possible as well as movement of infected poultry. In experimental studies AI viruses can be excreted in the feces and maintained in the environment and can re-emerge after a significantly stressful event
6. Once a flock is infected, it should be considered a potential source of virus for life.

Human Transmission

Human to human transmission do not take place until the virus in human get mutated and then transmit to the other person.

Pathogenesis

- The avian influenza virus adsorbs to glycoprotein receptors containing sialic acid on the cell surface.
- The virus then enters the cell by **receptor-mediated endocytosis.**
- The tissue tropism of a virus is involved in its pathogenicity.
- The basis for tissue tropism is receptor specificity.receptor recognition by the virus is an important factor in both tissue tropism and pathogenicity.
- Infectivity depends on post-translation clevageof the haemagglutinin molecule. That is, on a split or division (cleavage) of the haemagglutinin molecule after it has been formed/synthesized (post-translation). This cleavage is brought about by host proteases, and takes place at the cleavage site.
- Susceptibility of the haemagglutinin molecule to cleavage by host proteases depends on the number of basic amino acids at the cleavage site. Trypsin-like enzymes can cleave if only a single amino acidarginine is present at cleavage site, whereas other host proteases require multiple basic amino acid.
- The HAs of low to moderately virulent influenza viruses have only a single basic amino acid arginine at the cleavage site. These viruses are therefore cleaved in tissues where trypsin-like enzymes are found i.e. Respiratory and digestive tract, as a result their pathogenicity is limited to these areas.
- On the other hand, highly pathogenic viruses possess HAs with multiple basic amino acids at the cleavage site, therefore can be cleaved by proteases found throughout the body. Hence viruses replicate in all body tissues and result in generalized disease and death.

Avian Influenza

Incubation period is from 3-14 days and is dependent on the dose of virus, the route of exposure, the species exposed.

1. Some birds are found dead prior to observance of any clinical signs. There may be neurological signs and reduction in normal vocalizations.
2. Depression is common as is a precipitous drop in egg production. Respiratory signs are less prominent but can include rales, sneezing and coughing.
3. In mature chickens, the combs and wattles are often swollen and may be cyanotic. Conjunctivitis, edema of the head and neck, coughing, sneezing and nasal discharge may also be seen
4. Egg production in hens stops; the last eggs laid often have no shells
5. Death is common, but severely affected hens occasionally recover.

Sampling

Before collecting or sending any samples from animals with a suspected foreign animal disease, the proper authorities should be contacted. Samples should only be sent under secure conditions and to authorized laboratories to prevent the spread of the disease. Some isolates of the avian influenza virus may be zoonotic; samples should be collected and handled with all appropriate precautions.

Diagnosis

1.Highly Pathogenic Avian Influenza (HP AI) is clinically indistinguishable from virulent Newcastle Disease.
2.HP AI should be suspected when severe depression, inappetence, and a drastic drop in egg production are followed by sudden deaths in the flock.
3.Facial edema, swollen and cyanotic combs and wattles, and petechial hemorrhages on the internal organs support this diagnosis.
4.Because of the broad spectrum of signs and lesions a definitive diagnosis must be made by virology and serology.

Two National Reference Laboratories

- **for animals HSADL, Bhopal -**
- **for humans NIV, Pune. humans-**
- **Five regional diagnostic labs (RDDLs).**

Treatment

1. No practical, specific treatment exists for avian influenza virus infections in commercial poultry
2. Supportive care and antibiotic treatment have been used to reduce the effects of concurrent bacterial infections.
3. Amantadine has been shown experimentally to be effective in reducing mortality but the drug is not approved for food animals and quickly results in amantadine resistant viruses.
4. Amantadine hydrochloride, and other antivirals have been licensed for use in humans to treat influenza since 1966.
5. The medication is effective in reducing the severity of influenza Type A in humans.

Avian Influenza in Humans

Clinical signs(H5N1)

Fever, respiratory, vomiting, diarrhea, pain

In Fatal cases: severe bilateral pneumonia, liver dysfunction, renal failure, septic shock (H7N7)

Conjunctivitis

Mild influenza or respiratory symptoms

Fatal case: Acute respiratory distress syndrome

Prevention and Control

1.Due to the economically devastating nature of this disease, authorities should be notified immediately of any suspicious cases of highly pathogenic avian influenza.

2.While waiting for the authorities or a confirmed diagnosis, all suspect animals should be quarantined.

3.Should highly pathogenic avian influenza be confirmed by diagnosis, depopulation may need to occur.

4.Depopulation protocols include plans for the infected premises, contact-exposed premises, and contiguous premises.

5.Proper destruction of all exposed cadavers, litter and animal products are requiredTo control an outbreak of HP AI the premises must be thoroughly cleaned and disinfected. Insects and mice on the premises should be eliminated, then the flock depopulated and the carcasses destroyed by burying, composting, or rendering.

6.Once the virus have been killed, the manure and feed should be removed down to a bare concrete floor

7.If the floor is earthen, one inch or more of soil should also be removed.

8.The manure can be buried at least 5 feet deep. It may also be composted for 90 days or longer, depending on the environmental conditions.

9.The compost should be tightly covered with black polyethylene sheets to prevent entry of birds, insects, and rodents.

10. Feathers can be burned; alternatively, they may be removed and the area wet down with disinfectant.

11. High pressure spray equipment should be used to clean all equipment and building surfaces.

12. Once all surfaces are clean and free of all organic material, the entire premises should be sprayed with an approved residual disinfectant

13. Cresylic or phenol disinfectants are usually effective.

14. The practice of accepted sanitation and biosecurity procedures in poultry operations is of the greatest importance in the prevention, control and eradication of HP AI.

15. In areas where waterfowl, shorebirds or sea birds are prevalent, the rearing of poultry on open range is incompatible with a sound AI prevention program.

16. Appropriate biosecurity practices should be applied, including the control of human traffic and introduction of birds of unknown disease status into the flock.

17. One critical goal of prevention and control is the education of the poultry industry regarding how the virus is introduced, spread and how it can be prevented.

18.HP AI can emerge from MP AI outbreaks so prompt response to MP AI outbreaks is important.

19. The vaccine would only be used in an emergency situation especially to preserve breeding stock. This vaccine bank is being expanded.

Prevention: Humans

1.This vaccine will not be made commercially available to the general public.

2.Other H5N1 vaccines are being developed by other companies against different H5N1 strains. The vaccine contain one strain of type A and 2 strain of typeB.

3.It's safe to eat properly cooked chicken, turkey, and any other poultry . But do not eat raw (uncooked) or undercooked poultry or poultry products.

4.When cooking, separate raw meat from cooked or ready-to-eat foods. the same cutting boards, knives, or utensils should not be used on uncooked meats and other foods.

5. Heat can destroy flu viruses, so poultry meat should be cooked until the temperature of the meat reaches at least 158° Fahrenheit (70° Celsius)

Treatment(Antiviral drugs)

- Neuraminidase inhibitors Oseltamivir (Tamiflu), Zanamivir (Relenza) still drugs of choice
- New neuraminidase inhibitor – peramivir not available yet; can be given I/V and I/M
- Resistance of A/H5N1 to oseltamivir still not a worry.

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A RARE CASE OF FOETAL MUMMIFICATION IN CROSSBRED COW AND ITS SURGICAL MANAGEMENT

Introduction

Foetal mummification is associated with a series of morphological alteration that occurs to a foetus which dies and retained in uterus. However the occurence of disease in cattle is very low (0.43 to 1.8%) and is usually reported between 3 – 8 months of gestation (Roberts, 1986). Most mummified foetuses will remain in uterus untill treatment is given to expell them or until their removed by caesarean section (Wenkoff and Manns, 1977). In the present communication a rare case of failure to expell mummified foetus by treatment with PGF2α, to overcome this type of complication its surgical management has been placed on record.

Case history and observations

Six years old sahiwal cossbred cow was presented with a history of 300 days of gestation. At the relevant time the animal was bred by artificial insemination and the pregnancy was confirmed by rectal examination on 60 th day, clinically there was absence of visual signs of pregnancy. Per rectal examination revealed the closed cervix in addition to palpation of a hard bony mass adhering to uterine wall, while there was absence of cotyledons, fremitus and foetal fluid. The animal was apparantly healthy and taking food and water normally. According to case was diagnosed to be a foetal mummification and decided to treat medically.

Treatment

The choice of treatment for foetal mummification is Inj. ProstaglandinF 2α so double shot of Lutalyse (PGF 2α) @ 25 mg was injected intramusculary to the animal and kept under observation for lysis of CL and cervical dilatation. A long thick shred of brownish mucoid discharge from vulva was reported after 72 hours of therapy. On vaginal examination cervix was partially dilated and bony mass was palpated due to failure of complete dilatation of cervix after second shot of PGF 2α , casearean section would have been the choice of treatment. The Casarean operation was done by standing position at the farm. The animal was controlled by both physical as well as chemical method, Lignocain hydrochloride 2% was used for paravertebral regional anaesthesia. Ventral to the left paralumbar fossa was selected for operation (Fig. 1) and this area was prepared for operation by clipping, shaving and finally sterilizing with Tr. Iodine soaked cotton.

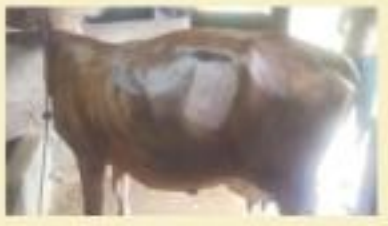

Fig.1 Site selected for operation

Fig. 2 Showing gravid uterus having mummified foetus.

During operation 1000ml of 5% Dextrose saline was infused intravenously to compansate dehydration from fluid and blood loss. Following aseptic preparation of the operative field a 14 inches long vertical incision lateral to the paralumbar fossa was made to open the abdomen. The distended gravid uterus was pulled out through the incised opening (Fig. 2)

Fig. 3 Showing bony pieces of mummified foetus.

Drapes and sterile gauge were used to prevent the leakage of uterine fluid to peritoneal cavity. A longitudinal incision along the greater curvature of uterine horn was made to remove the bony pieces of mummified foetus (Fig. 3).

The inner surface or endometrium and peritoneal cavity were thoroughly flushed out with normal saline and 200 ml Metronidazole i/v to compansate visceral moisture and to combate anaerobic infection. The incised uterus, peritoneum and muscles were sutured with chromic catgut No. 2 subsequently and finally the skin was closed with silk thread (Fig. 4, 5 & 6).

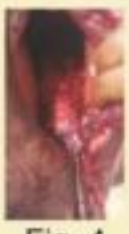

Fig. 4

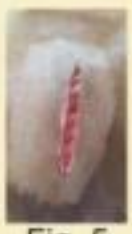

Fig. 5

Fig 6

Showing the suturing of incised uterus, peritoneum, muscles and skin

A Tr. Benzoin seal was applied over the suture line. Post operatively Dextrose saline 5% (1lit./day) was continued intravenosly for 3 days. Inj. Dicrystacine 2.5 gm was injected intramuscularly for 5 days to prevent bacterial infection. 15 ml of meloxicalm was injected intramuscularly daily for 5 days to reduce inflammatory pain and tablets Uterovet @ 10 bid, p/o were fed for 10 days. On the day 10 th the suture of skin was removed and animal was recovered uneventfully (Fig. 7).

Fig. 7 Showing recovered animal

Discussion

The main goal when treating an animal with abnormal pregnancy related to the foetus is to expel the abnormal foetus so cow can became pregnant again within shortest possible time. The choice of treatment in cases of foetal mummification is Inj. of Prostaglandin F 2α to induce leutolysis leading to expulsion of foetus within two to five days (Wenkoff and Manns, 1977). In the present case study we observe luteal regression and uterine contractions with the first PGF 2α Inj. but it was probably not enough to produce the continuous contractions required to expel mummified foetus therefore a second PGF 2α inj. was administered but expulsion of foetus was not achiEved. To overcome this type of complication we decided to deliver the mummified foetus with Casarean section as suggested by Arthur etal 1996. Treatment with PGF 2α in present case might have resulted in maceration of mummified foetus and thus it was stucked at external os of cervix. This complication has already been discuss by Arthur etal 1996 and reported that treatment of

mummified foetus with PGF 2α create some complexity in cattle like maceration of foetus and packed in birth canal instead of expell out.

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Zonal Veterinary Dispensary, Sakhali

Dr. P. R. Parab
Veterinary officer
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Dr.Thomas Edison D'sa	10
Dr.Maria Niceta Cunha Costa	11
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