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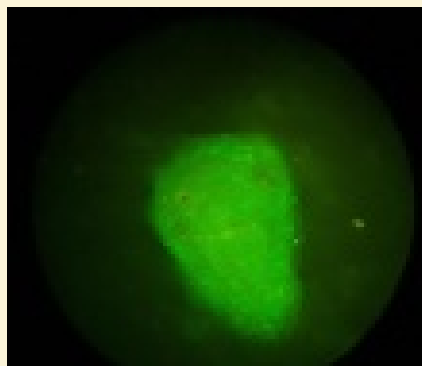
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POSTMORTEM INVESTIGATION OF RABIES IN A STRAY DOG

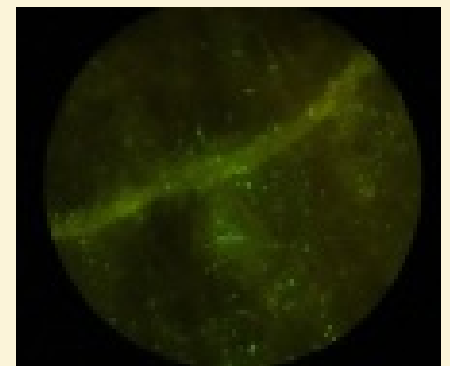
A 4 months old dog with multiplicity of clinical signs including seizures, lack of muscular coordination, paralysis in the mandible and larynx, hypersalivation and dropped jaw condition was found wandering in the college campus. Dog was suspected for Rabies and was euthanised immediately. A detailed post mortem examination was carried out which revealed no systemic findings.

The impression smears of cerebrum, cerebellum and salivary gland were taken for Fluorescent antibody test examination (an immunohistochemistry procedure) on clean glass slides and fixed with cold acetone for half an hour. Fluorescent antibody test examination was carried out by using Rabies DFA reagents from light diagnostics. 100 ml, of nucleocapsid antibodies conjugated with FTCC was added and then incubated at 37degree Celsius in a moist chamber. After washing with PBS and drying, the slides were observed by using a fluorescence microscope. In this way, the labelled antibody was incubated with rabies suspect brain tissue, it showed binding to rabies antigen. Unbound antibody was washed away and areas where antigen were present could be visualized as fluorescent- apple -green areas showing Negri bodies confirming the existence of rabies in the tissue.

Existing green fluorescent particles in microscopic examination indicated positive result for Negri bodies and rabies infection in a stray dog. So, this suggest us that the protocol used for rabies diagnosis is an effective and applied method, which is recommended by the World Health Organisation. However, molecular techniques are useful in antemortem diagnosis.



NEGRI BODIES IN DOG BRAIN TISSUE
INFECTED WITH RABIES VIRUS
(APPLE GREEN FLUOROSCENT WITH
A DARK GREEN BACKGROUND)



NEGRI BODIES IN SALIVARY GLAND
INFECTED WITH RABIES VIRUS
(APPLE GREEN FLUOROSCENT WITH
A DARK GREEN BACKGROUND)

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BOVINE ABORTION AND INFERTILITY RELATED TO INFECTION BY SPECIFIC PATHOGENS

1. Bovine Viral Diarrhoea (BVD)

Bovine Viral Diarrhea (BVD) is a disease of cattle caused by the Bovine Viral Diarrhea Virus (BVDV). BVD diminishes production and in the individual impacts multiple body systems including the reproductive, respiratory, digestive and immune systems. Clinical signs can vary from pneumonia, abortions, stunted calves, stillbirths, PI calves, weak calves, unthriftiness, increase disease susceptibility and full blown disease characterized by a watery diarrhea that frequently leads to death.

BVDV has two biotypes, cytopathic (CP) and non cytopathic (NCP), a distinction based on the characteristics of the virus when grown in a laboratory in tissue culture. Antigenic variations of the viral surface proteins result in two BVDV types, type 1 and type 2. Both type 1 and type 2 BVDV can be CP or NCP.

Transmission and Sources of BVD

- Transmitted by, ingestion, inhalation, insects, carried on boots and vehicles
- Sources
 - transient infected animals including wildlife
 - Persistently infected (PI) animals are the main source of infection. PI's shed viruses in high numbers and infect others, even if they are vaccinated.

Either through a congenital infection of the foetus or after birth. Congenital infections may cause resorption, abortion, stillbirth, or live-birth. Congenitally infected foetuses that survive in utero infection (i.e., the live-births) may be born as BVDV-infected calves. The BVDV infection in these calves will persist during the entire life of the calf, and they will shed virus continuously in the farm environment.

BVD can cause a whole range of disease syndromes in cows. When the virus circulates in the cow, it is able to reach the growing foetus through the placenta. If a cow who is not immune is exposed to the BVD virus in the first trimester, an early embryonic death or abortion may occur, and if the calf is not expelled from the uterus, it may become mummified.

However, if the calf is exposed to the BVD virus between 42 and 125 days of gestation, and if it does not die, it may be born as a "persistently infected" calf. During the second 3 months of gestation, an infection may result in an abortion, or a calf which will be born with birth defects. Generally, if a calf is exposed in the uterus during the last trimester, the virus will have no effect on the calf, except that it will be born with antibodies to BVD in its blood. Occasionally a late-gestation abortion may result from a BVD virus infection. While immunity in the cow (by exposure or vaccination) should help to protect the developing foetus, the protection offered is not 100% since there are different strains of BVD virus and only a few virus particles need to get to the foetus to cause an infection.

Clinical Signs

Respiratory infection with BVDV is characterized by signs typical of viral respiratory disease, including fever, depression, inappetance, and ocular and nasal discharge, followed by diarrhea several days after onset. Sores or ulceration in the mouth and gums may be present, along with reduced milk production in cows.

Bovine viral diarrhea includes several distinct disease syndromes, all caused by BVDV:

1. Respiratory infections, with signs similar to IBR, plus oral and tracheal ulceration
2. Thrombocytopenic (bleeder) syndrome, where type 2 BVDV infects blood cells and bone marrow, causing destruction of red blood cells, reduced clotting function, bleeding from wounds, lesions, and internal organs.
3. Reproductive disease resulting in embryonic loss and abortions.
4. Persistent infection (PI), which results when a calf is infected in utero with NCP BVDV during the first trimester of gestation and survives, and results in a continuing reservoir of BVDV infection. Animals with PI are often outwardly normal.
5. Mucosal disease occurs when an animal with PI is exposed to a CP strain of BVDV, resulting in explosive diarrhea and ulceration throughout the digestive tract and in most cases, death.

In calves

- o Death of fetus if infected in the first 120 days of pregnancy
- o Abortion
- o Congenital malformations of nervous system and eyes; blindness and underdeveloped brains
- o Skeletal defects
- o The most commonly recognized birth defect is cerebellar hypoplasia. The signs of this are:
 - Ataxia/ lack of voluntary coordination of muscle movements;
 - Tremors
 - Wide stance
 - Stumbling

Diagnosis

- Based on clinical signs
- Virus isolation from blood, milk, and tissues is useful in diagnosing BVDV.
- Detection of antibodies

Individual cows can be blood tested (or tissue tested) to see if they have been exposed to the virus and have mounted an immune response (antibody test), or to see whether the virus is present (antigen test). Transiently infected animals may be antigen positive due to temporarily circulating virus, and should be retested in 4 weeks, by which time they should be antigen negative but antibody positive, thus distinguishing them from the PIs.

Treatment and Control

There is no specific anti-BVDV therapy. Treatment is limited to antibacterial therapy for secondary bacterial infections and supportive treatment.

Control measures should include:

- o Diagnosis and removal of BVD-PI cattle
- o Vaccination to reduce BVD infection rate (Vaccination will not eliminate BVD virus from cattle herds)
- o Biosecurity to prevent introduction of BVD-PI animals

2. Infectious Bovine Rhinotracheitis (IBR)

IBR is an acute, contagious respiratory disease of cattle caused by bovine herpesvirus type 1 (BHV-1), IBR is characterised by acute inflammation of the upper respiratory tract.

BHV-1 cause a variety of different disease syndromes, the most common of which is respiratory disease (pneumonia, "red nose") leading to acute inflammation of the upper respiratory tract. It remains the most commonly diagnosed viral cause of abortions in cattle. Abortions most commonly occur from 4 months to term, and may occur weeks after the disease has gone through the herd.

Transmission of IBR

Transmission of infectious bovine rhinotracheitis can occur both directly and indirectly. Direct contact between infected cows and unaffected cows can generally result in the transfer of the bovine herpes virus type 1. As a latent virus, previously infected cows may shed the disease without showing any signs and therefore contribute to spreading it.

The ability of Virus to "periodically" reactivate from latency is crucial for viral transmission. Periodic reactivation episode occurs due to stress, decrease in immune response. Infected bulls are able to transmit the virus via semen and so can infect many members of the herd in a short period of time. Additionally, humans are also able to act as a source of infection via contaminated clothes and footwear if they come into close proximity with an infected cow and then another cow. Embryonic transfer and sometimes aerosol droplets can also cause transmission.

Signs and symptoms of infectious bovine rhinotracheitis

IBR is an acute infection usually characterized by visible signs, including sudden onset of fever, salivation, rhinitis (red nose), conjunctivitis (red, watery eyes), inappetance, and dyspnea (difficult breathing).

The nasal mucosa and muzzle are distinctly inflamed, with abundant nasal discharge. Nasal lesions become large, consolidated hemorrhagic (red) areas or white plaques.

In advanced cases, respiratory distress increases and open-mouth breathing is evident. If primary BHV-1 infection does not resolve in five to ten days, secondary bacterial infection can occur, leading to bronchopneumonia and death in complicated cases.

The reproductive system may also become involved manifested by infertility problems, abortion, and birth defects.

Diagnosis

- Based on signs and symptoms
- Virus isolation from nasal or ocular swabs
- Detection of antibodies

A presumptive diagnosis can be made on the basis of history, clinical signs, and the presence of intense nasal hyperaemia or the presence of necrotic debris or plaques in the nasal passages.

From the live animal, nasal or ocular swabs may be taken for virus isolation studies. From the dead animal, bronchial lymph nodes or tracheal tissue may be submitted. The aborted foetus is usually autolyzed and shows no typical gross lesions. Fixed samples of fetal liver and kidney should be submitted for histopathological study since the IBR virus produces lesions of necrotic foci in these organs. Frozen sections of the placenta, liver and kidney, and fluid from the foetal thoracic cavity can also be used for viral isolation study.

Serum can be submitted for virus neutralisation test and ELISA. ELISA test is also available to detect antibodies in milk.

Treatment and Control

There is no specific anti-BHV-1 therapy. The most appropriate treatment is antibiotic therapy designed to control secondary bacterial infection. Management practices designed to reduce stress, isolate infected animals, and provide adequate food and water will limit disease transmission and severity.

Control of the disease is based on the use of vaccines

- Since BHV-1 is a ubiquitous, highly contagious virus, vaccination is recommended as soon as passive immunity in calves has disappeared, usually around four to six months of age. Currently available vaccines for IBR include modified-live-virus (MLV) vaccines and inactivated or killed-virus (KV) vaccines.
- The use of marker vaccines is preferred since the antibody they stimulate can be distinguished from the BHV-1 antibody that follows a natural infection and so secondary vaccination is required.

Appropriate biosecurity will also reduce risk on farm

COMMON DISEASES OF BACK YARD POULTRY AND THEIR CONTROL

Back yard poultry rearing is an age old practice in India in which a small number of birds are maintained without much initial investment. The birds are economically maintained by utilization of kitchen left over and agricultural by-products. Commonly local indigenous birds are maintained all over the country in addition to improved breeds with better egg production and weight gain developed by Research Institutions. The keeping of backyard poultry provides the small scale farmers with additional income and source of nutrition to the family. In general indigenous birds are resistant to most of the diseases but their growth and productivity are affected by diseases. Better healthcare in terms of vaccination against important diseases, deworming, application of anti-coccidial agents etc can protect the birds from diseases and can help to increase the productivity. The major category of diseases includes bacterial, viral, parasitic diseases and nutritional deficiencies.

Viral diseases:

The common viral diseases are New Castle Disease (Raniket Disease/RD) characterized by sudden high mortality, severe respiratory signs including gasping, coughing, sneezing, and rales. Often this disease is associated with nervous signs of tremors, paralyzed wings and legs, twisted necks, circling and digestive disorders like greenish diarrhoea disease results in complete cessation of egg production. Infectious bronchitis is a rapidly spreading respiratory disease in young chicks. In adult laying hens disease cause reduced production, egg shell abnormalities, and decreased internal egg quality. Marek's disease (MD) is another viral disease characterized by paralysis, mortality, enlargement and presence tumors in liver, kidney and spleen. Another important lesion is severe thickening of sciatic nerve. Avian leucosis is also a viral disease with lesions of tumours and the disease is usually seen in birds of more than 6 months old. Fowl Pox is another viral disease characterized by nodular proliferative Skin lesions on the featherless parts of the body like Combs, wattles and eyelids. Infectious bursal disease affects young birds and lesions are swelling of bursa of fabricius, haemorrhages in thigh, breast muscles. The Vaccines are available against the common viral diseases.

Vaccination schedule in poultry

Age	Disease	Vaccine	Route
1 day	Marek's	HVT vaccine	I/M
5-7 days	RD	Lasota/F	Occulonasal
10-14 days	IBD	IBD Live	Drinking water
24-28 days	IBD	IBD Live	Drinking water
8th week	RD	R2B/RDVK	S/C
16-18 week	RD	Killed/Live	S/C

Bacterial diseases:

Another cause of disease in backyard poultry is infection with different types of pathogenic bacteria which can be controlled by appropriate use of antibiotics. One of the common bacterial diseases is Infectious coryza caused by *Avibacterium paragallinarum*. It is a highly contagious acute disease of upper respiratory tract of chickens, characterized by nasal discharge, sneezing, and swelling of the face under the eyes.

Pullorum Disease caused by *Salmonella pullorum* is a fatal septicemia of young chicks with weakness, loss of appetite and white diarrhoea. Fowl cholera caused by *Pasteurella multocida* manifests either in acute septicaemic form with a high morbidity and death rates or as chronic local form. In acute fowl cholera death is the first sign with lesions in visceral organs due to vascular injuries. In less acute disease, the common signs are anorexia, ruffled feathers, oral and nasal mucus discharge, cyanosis and white or greenish watery mucoid diarrhoea. Chronic respiratory disease is a condition caused by *Mycoplasma gallisepticum* characterised by respiratory signs like gasping nasal discharge and lesions like thickening of air sac, tracheal congestion, and deposition of mucous or caseous material over the tracheal mucosa.

Bumble foot is clinical condition with swollen foot pad occurring due to bacterial infections of the foot mainly after injuries caused by rough perches, Splinters, Wire floors, Poor litter or bedding. Most common associated bacteria is *Staphylococcus aureus*. The affected bird shows signs like lameness, swollen foot pad which could be either hard or pus-filled abscess.

Parasitic diseases:

Due to their scavenging nature, the backyard poultry also carry a wide range of internal parasites from microscopic protozoa to large helminth worms. They inhabit the gastrointestinal tract, feed blood and drain the nutrients from the birds which affect the growth and productivity. The most important protozoan parasitic infection is coccidiosis caused by different species of *Eimeria*. The disease is commonly seen in chicks and grower birds. It is characterized by haemorrhagic enteritis and mortality. Oocysts are passed in feces of affected birds which can act as source of infection. The condition can be diagnosed by detection of oocysts microscopically. The disease can be controlled by use of anticoccidial drugs like Sulfa antibiotics and Amprolium. Control also requires change of litter and application of calcium oxide (lime) in the litter.

Helminth parasites include different species of Nematodes, Cestodes and trematodes. The Nematodes or round worms range from large *Ascarids*, to minute *Capillaria sp.* and *Heterakis gallinarum*. *Syngamus trachea* or gape worm inhabits the trachea and bronchi of birds and it is a serious problem in birds kept outdoors in endemic regions. The common cestodes or tape worms in chicken are *Railletina spp.*, *Davainea spp* and *Choanotaenia*. Tapeworms require an intermediate host to complete their life cycle. These intermediate hosts include ants, beetles, houseflies, slugs, snails, earthworms, and termites. For birds kept in cages, the most likely host is the housefly. For those raised on litter, intermediate hosts include termites and beetles. For free-range birds, snails and earthworms can serve as intermediate hosts. As it is difficult to cure the infection with anthelmintics control of the intermediate hosts of tapeworms is vital in preventing initial infections and reducing the risk of re-infection. A severe infestation with parasites can cause reduction in the nutrient absorption, intestinal blockage, and death. *Prosthogonimus* or oviduct fluke a trematode parasite is a serious threat to outdoor chicken. The worms are easily transmitted by contamination of premises with oocysts in the feces. Regular deworming is essential to reduce the parasite infection to ensure better growth and productivity of the flock. As the continuous use of same anthelmintic may result in development of resistance in parasite change in the anthelmintic drug is also required to effectively control the parasite infection.

Poultry can carry a large variety of parasites on their bodies. These range in size from large, blood sucking ticks to the microscopic mites. The common mite is the red poultry mite (*Dermanyssus gallinae*). Other commonly encountered external parasites are the stickfast flea on the comb, chicken body louse (*Menacanthus stramineus*) and shaft louse (*Menapon gallinae*). External parasites generally cause mild clinical signs such as feather damage, anaemia and irritation, but they may also carry severe, life threatening diseases such as tick fever. Infection with these parasites can be controlled by regular examination and treatment using insecticides in the form of dusts, wettable powders, liquid sprays etc.

Nutritional deficiency:

As the back yard poultry is mainly fed on kitchen wastes and agricultural by products the feed is not sufficient to meet the nutritional demands of growing birds or layers. Common deficiencies include calcium (resulting in poor bone growth and rickets), energy (poor growth, weight loss, poor egg production), and vitamin A (poor skin and feathering). In case of deficiency disorders the diet may be supplemented with vitamin and mineral source supplements to form a balanced feed.

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SUCCESSFUL MEDICAL MANAGEMENT OF CLOSED PYOMETRA IN A BITCH

Introduction:

Canine pyometra is a common reproductive disorder of intact, nulliparous old bitches with accumulation of pus within the uterine lumen, typically occurring during (or) immediately following a period of progesterone dominance. It is caused by a bacterial infection within the uterus and results in mild to severe and life threatening bacteremia and toxemia. It is characterised by an inflammatory exudate and uterine bacterial colonisation associated with Cystic Endometrial Hyperplasia (Johnston et al., 2001).

Though several literatures are available regarding treatment of closed pyometra in bitches (Jena et al., 2013), the use of antiprogestins in combination with methergine is not reported. The present case report describes the successful medical treatment of closed pyometra in a bitch with the combination of mifepristone and methergine.

Case history and Observation:

A three year old nulliparous Great Dane bitch was presented to the Gynaecology and Obstetrics ward of the Teaching Veterinary Clinical Campus with the history of anorexia since five days. The bitch was said to be taking more water and frequently vomiting without any vaginal discharge. Owner reported that bitch had proestrus bleeding one month back. On general examination the bitch was dull and depressed and had rectal temperature of 101.6 °F. Abdominal palpation revealed enlarged uterine horns. The animal was subjected to trans-abdominal B-mode ultrasound scanning by using 3.5 to 5 MHz sector probe. Abdominal ultrasonography revealed enlarged uterus with convoluted tubular horns filled with hypoechoic fluid. The average diameter of the uterine horn was 33.0 mm. Haemato-biochemical analysis revealed neutrophilia, leucocytosis, increased serum Blood Urea Nitrogen (BUN) and creatinine levels.

Diagnosis and treatment:

Based on the history, general examination, ultrasonographic and haemato-biochemical investigations, the case was diagnosed as closed pyometra. The dog was treated with Mifepristone at the dose rate of 5 mg/Kg body weight orally and intravenous fluids for two days. Follow up of the case revealed that the dog evacuated around 1.5 litres of purulent vaginal discharge after the treatment. Ultrasonography of the abdomen on day 3 showed reduction in diameter of uterine horn (8mm). The dog was further treated with Cephalexin at the dose rate of 10 mg/Kg body weight and Methergin (0.124 mg) BID orally for a week. Ultrasonographical examination on day 10 revealed normal uterus with uterine horns free of hypoechoic fluids.

Discussion:

Pyometra is a disease that affects a large proportion of intact bitches, and typically is seen during the latter half of diestrus in nulliparous bitches (Younis et al., 2014). Several factors contribute to the development of pyometra, including genetic factors, an infectious component, and hormonal factors. Hormones may act directly on the endometrium, and also affect the immune system. In dogs, the phagocytic ability has been shown to decrease with age, and ovarian hormones have also been shown to affect immune resistance (Holst et al., 2013). The haematological and biochemical alterations commonly found during the course of the disease are leukocytosis with neutrophilia, hyperglobulinaemia and azotemia. The azotemia is secondary to glomerulonephritis by immune complex deposition in the glomerular basement membrane and is aggravated by pre-renal azotemia (Fieni, 2006; Johnson, 2006; Pretzer, 2008).

The bitch with closed cervix pyometra is often quite ill at the time of diagnosis compared with dogs that have open cervix pyometra. This is due to lack of an easily recognized, early sign of a serious problem. Indigenous signs include lethargy, inappetance, polydipsia, polyuria, and weight loss (Younis et al., 2014). Mifepristone is a progesterone receptor antagonist. It acts at the level of uterus independent of any additional effects on luteal function. Oxytocin and ergot derivatives induce very strong, short-lasting contraction of the uterine wall which may be dangerous if the uterus is fully dilated with pus and/or the uterine wall is thin and atrophied or the cervix is only partially dilated (Wheaton and Barbee, 1993). Methergine is an ergot alkaloid uterine stimulant and works by increasing uterine contractions (Votava and Podvalova, 1957). In the present case medical treatment was opted instead of surgical removal of uterus and ovaries since the owner was interested to breed the bitch in future.

Conclusion:

From present study it was concluded that ultrasonography is an accurate diagnostic tool for diagnosing closed pyometra in bitches and combination of Mifepristone and Methergine was found to be very effective in treatment of closed pyometra instead of going for ovariohysterectomy. However, further trial on more number of animals is required to evaluate the efficacy of Mifepristone and Methergin combination for treatment of closed pyometra.

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MASSETER MUSCLE ATROPHY (MMA) in a dog -A Case Study

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ABSTRACT:

Atrophic myositis is the result of prominent atrophy of masticatory muscles. The muscles selectively affected are the masseter, temporal, and pterygoid . A dog was presented to the Government Veterinary Hospital Sonsodo with the clinical signs of weight loss, dyspnoea and cough. The dog was reported to be unable to completely open his mouth (trismus) and with unusual prominences of zygomatic arches. After treatment the dog was able to open its mouth (90%) and lead a good quality life.

Keywords: trismus, masticatory, masseter, myositis

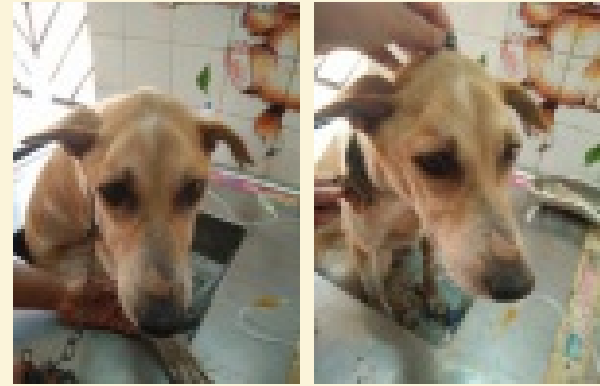


Image 1.a.

Image 1.b.

INTRODUCTION:

Canine masticatory muscle myositis (CMMM) is an immunomediated canine myopathy, which selectively causes focal myositis and progressive destruction of type IIM myofibres (Pompei BOLFÄ et al ., 2011).The masticatory muscles (masseter, temporal, and pterygoid) are selectively affected, leading to trismus in dogs (Brogdon, 1991; Nelson et al., 2000). This disease must be included in the differential diagnosis list of problems of the temporomandibular joint which is the main aspect of infection and thus the differential diagnosis is of utmost importance like trigeminal neuritis or the early form of tetanus in dogs (Pompei BOLFÄ et al ., 2011; Blot, 1995; McGavin, 1995; Shelton, 1991). The condition was previously designated as two separate disorders: eosinophilic myositis and atrophic myositis which are now recognized to be two ends of the spectrum of a single disease known as masticatory myositis (Pompei BOLFÄ et al ., 2011). Atrophic myositis is the result of prominent atrophy of temporal and masseter muscles (McGavin et al., 2007, Jubb et al., 2007). This report presents a 4 year old dog who progressively improved its quality of life by eating by himself but the muscle atrophy was progressive.

MATERIALS AND METHODS

A case of a 4 year old intact male dog was presented to the Government Veterinary Hospital Sonsodo with the clinical signs of weight loss, dyspnoea and cough. The dog was reported to be unable to completely open his mouth (trismus) and with unusual prominences of zygomatic arches. The owner did not want to euthanize the dog and was feeding him solely a liquid diet through a syringe through the space between the lip commissure including the space between molars of upper and lower jaw. Pain and trismus prevented a thorough oral examination and it was decided to proceed with treatment based on clinical signs and blood evaluation. Excessive force in opening the jaw manually was avoided as it may have resulted in fractures of the teeth or jaw.

RESULTS AND DISCUSSION

Muscle atrophy gradually became very obvious and the head appeared to have a fine fox-like contour with unusual prominence of the zygomatic arches (see image 1.a. and 1.b.). The primary inflammatory cells causing destruction of myofibers in masticatory muscle myositis of dogs are lymphocytes, presumably recruited following binding of antibody. In cases of dogs suspected with masticatory myositis, histologic examination is difficult. The masticatory muscles of dogs, especially the temporal muscles, appear to be particularly prone to a variety of generalized myopathic and systemic disorders. Dogs with X linked muscular dystrophy often have prominent atrophy of the temporal muscle and are unable to fully open their mouths, although this condition is not accompanied by pain. Dogs with any generalized, illness often develop rapid atrophy of the temporal muscles that resolves with treatment for the primary problem (Jubb et al., 2007). In this case there was no underlying illness as shown by blood evaluation and only mild pain on palpation of jaw region.

Blood report evaluation revealed eosinophilia and lymphocytosis along with neutrophilia. Treatment protocol included intravenous Normal saline, Ringers Lactate , Ceftriaxone ,tramadol, corticosteroid Methyl Prednisolone at tapering doses for a period of two weeks. During this time, after about a week and a half of treatment there was absolutely no improvement in the case. But the owner of the patient did not give up and continued feeding the patient soups and mashed food through a syringe along with oral prednisolone supplements and multi vitamins.

On 21st day post treatment , an opening distance of 2 inches was observed and the patient was able to lap some milk with the mobility of jaw just enough to take in the milk. On consequent weekly follow ups at the hospital there was a drastic improvement in the entire outlook of the patient. He looked more bright and alert (see image 2 a.), was eating



Image 2.a.

Image 2.b.

the pedigree pellets offered to him although with slight difficulty.

At the 4th week follow up at the Hospital, the patient came in with good mobility of jaw , tongue out and panting due to the heat of the external environment.

CONCLUSION

It was sheer dedication by the client who refused to give up on her pet along with the specific treatment at the right time that helped save this patient. At this point, a blood evaluation was repeated which showed only eosinophilia and lymphocytosis.

CMMM is a very rare disease that affects 20% of dogs in India. With a complete and correct history and timely treatment cases can recover for a better quality of life. The patient is on a three month course of steroid treatment. However as noticed in the image 2.b. the atrophy of the muscle further worsened. But the patient is as of now eating well and seems like a happy dog!

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Dr. Rameela Menezes

who retired under VRS .



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