EMERGING AND REEMERGING HAEMOPROTOZOAN DISEASES OF PET ANIMALS

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Haemoprotozoan diseases constitute an important group of illnesses affecting dogs around the world. Vector borne haemoprotozoan diseases are historically endemic in tropical and subtropical regions and have increasingly been recognized, not only in traditionally endemic areas, but also in temperate regions. Vector borne haemoprotozoan diseases have long been recognized in India. Pet animals especially dogs are affected by many haemoprotozoans (e.g., *Babesia canis, B. gibsoni, Hepatozoon canis, Trypanosoma evansi, Leishmania donovani donovani and Leishmania tropica. In addition,* rickettsial diseases caused by *Ehrlichia canis* and the recently emerged *E. ewingi* are important haemoprotistan diseases of pet animals especially dogs. Although, all the aforementioned diseases are encountered in dogs for the past one century in India, this article will focus on haemoprotozoan diseases and ricketssial diseases of pet animals that have emerged or remerged in the recent past.

Canine hepatozoonosis

The American form of canine hepatozoonosis, which was more virulent, and dissimilar from that seen in the Old world was classified as a new species called *Hepatozoon americanum* by Vincent-Johnson and his team in 1997. Recently, experimental transmission studies have also shown that the Gulf coast tick, *Amblyomma maculatum* is the natural vector for *H. americanum, which* confirmed the fact that the species seen in USA was not *H. canis* that is seen in the Old World.

In India, Hepatozoon canis in a domestic cat was first described in Madras in India, early in the 20th century by Patton in 1908 and later from dogs in India by Christoper in 1912. Subsequently, *H. canis* was reported from many species of domestic dogs, wild carnivores and in cats in Far East, Northern and Central Africa, Middle East and Italy. Hepatozoonosis caused by *H. canis* in the Old World is often found in apparently healthy dogs but can also cause serious disease and death.

The principal signs are irregular fever, severe anorexia, weight loss, lethargy, neutrophilia, thrombocytopaenia and profound anaemia, possibly caused by an immune mediate process. Severe neutrophilia with hepatozoonosis has been described and seems to be a common finding. The high percentage of parasitized neutrophils is probably a manifestation of widespread dispersion of *H. canis* schizonts in the tissues of the dog (Harikrishnan *et al.* 2008). This massive parasitaemia reflects the large number of tissue meronts, and takes its toll on the host by demanding nutrients and causing direct injury to affected tissues, leading to extreme weight loss and cachexia. In cats, lethargy, anorexia, stomatitis, gingivitis, glossitis, oculonasal discharge and pyrexia has been reported. Lumbar paralysis which is mentioned as one of the clinical sign in canine hepatozoonosis as described in some books is in fact caused by *Hepatozoon americanum* in the New World and not in canine hepatozoonosis that is prevalent in the Old world.

In canine hepatozoonosis caused by *H. americanum,* the asexual stages of the parasite are seen predominantly in skeletal and cardiac muscle rather than in spleen and liver as in the Old World strains. Inside the cells of these organs, the parasite reproduces by schizogony and eventually ruptures a cell. The damage caused by rupturing of these cells causes severe muscle pain. The disease produced in dogs by *H. americanum* has been well characterized as a chronic, progressive, often fatal condition accompanied by muscle weakness, wasting, leucocytosis and periosteal bone proliferation. Lumbar paralysis, cyst formation in muscles and bone and periosteal new bone formation has been reported in dogs in the New World. In diseases caused by *H. americanum* severe lameness, muscle pain, and an inability to rise are often observed. These signs may occur on and off for years.

The dissimilarity in the clinical manifestations of *H.americanum* and *H. canis* is attributed partly to the different target organs in which merogonic development takes place. *H. americanum* principally infects skeletal and cardiac muscle, and induces pyogranulomatous myositis. The host cell appears to be a phagocytic cell that is initially located between myocytes. Concentric layers of mucopolysaccharide material are deposited around the host cell and form a large cystic structure (250–500 µm diameter) giving it the appearance of an `onion skin' cyst, which is not found in *H. canis*. The rupture of mature meronts induces the formation of highly vascularized pyogranulomas, where the released merozoites invade leukocytes and develop to gamonts that circulate

in the blood, or possibly give rise to secondary meronts. In the absence of an easily detectable parasitaemia, muscle biopsy and recently developed serological testing are the principal means of *H. americanum* diagnosis.

Hepatozoon canis is mostly diagnosed by microscopic detection of intracellular H. canis gamonts within neutrophils in stained blood-smears. The gamonts are large (11µm×4 µm) and have an ellipsoidal shape. Mature *H. canis* meronts in tissues contain elongated merozoites arranged in a circle around a clear central core, forming a unique shape of a `wheel spoke' meront. H. canis is treated with the antiprotozoal imidocarb diproprionate and the tetracycline doxycyline. Elimination of gamonts from the peripheral blood is slow and could require periodic treatment with imidocarb diproprionate for eight weeks.

Canine babesiosis

Canine babesiosis has been recognized in India since the beginning of the 20th century. This disease is caused by *Babesia canis and Babesia gibsoni* (Piroplasmida: Babesiidae). In the recent past, cases of *Babesia gibsoni* infection in Indian dogs are reported commonly. The only proven vector of *B. canis* in India is *Rhipicephalus sanguineus*, which is also the suspected vector of *B. gibsoni*. In India both *B. canis* (Varshney et al (2004) and *B. gibsoni* (Harikrishnan *et al.,* 1999 & 2005; Varshney et al., 2003) are prevalent. Recent reports indicate that *B. gibsoni* is an emerging vector-borne disease among dogs in India.

Babesia gibsoni was first identified in dogs and jackals from India in 1910. This parasite is considered endemic among dogs in Northern Africa, the Middle East and Southern Asia. In B. *gibsoni,* the parasitaemia ranges from 1 to 8 percent (Varshney et al., 2003 and 2008)). Mostly trophozoites were annular, oval, coma or band shaped. As many as 4 to 5 trophozoites are seen in a single erythrocyte (Varshney et al. 2008).

A wide variety of non specific clinical signs has been reported. Non specific referable to general stage in 58.5%, gastrointestinal tract in 96.07%, hematological in 50.9%, nervous system in 68.5% , cardiothoracic in 58.5%, urological in 46.6% and ocular in 3.8% cases were reported. Pyrexia was not a consistent finding in babesiosis, caused by *B. gibsoni.* Tender abdomen and epigastric pain were more marked in dogs and could be ascribed to splenomegaly or hepatomegaly. Ascites was evident in 11.75%

and therefore important not to neglect babesiosis in differential diagnosis of ascites. The spleen will be diffusely hyperechoic and enlarged in cases of babesiosis, caused by *B. gibsoni.* Haemorrhages, an important reflection of thrombocytopenic crisis will be manifested as epistaxis. Dullness, ataxia, posterior paresis, circling and tremors either alone or in combination are common neurological manifestations associated with babesiosis, caused by *B. gibsoni*. Haemoglobinuria, pyrexia and icteri marked in *B. canis* infection are extremely rare in babesiosis caused by *B. gibsoni.*

Babesiosis is usually diagnosed by demonstrating intraerythrocytic trophozoites in blood smears stained with Giemsa or Wright. *B. canis* may occur as single, pairs or in multiples and are usually pyriform although oval or round forms are also seen. *B. gibsoni* is usually smaller (measuring 1.9 x 1.2 micrometers), singular, and signet ring shaped. Sampling of blood from a capillary bed (from the ear, for instance) helps to demonstrate the organisms better than sampling blood from a larger vein. Isolation of infected erythrocytes with a Percoll gradient can be used to enhance the recovery and identification of parasitized erythrocytes. The degree of parasitemia is very low with *B. canis*, but may range from 2% to 6% (or greater) of the erythrocyte population with *B. gibsoni.*

Feline babesiosis

In India, feline babesiosis is not reported frequently. Lingard and Jennings (1904) were the first to report *B. felis* in India and thereafter there were reports published sporadically (Mangrulkar, 1937; Mudaliar et al., 1950; Changkija and Varshney, 2006). Recently, there are reports on its prevalence in cats (Varshney et al., 2008) in India. Although, it cannot be called as an emerging parasite, the protozoan is now frequently diagnosed in cats. *B. felis* resembles *B. gibsoni* of dogs. Fever and icterus, predominant in canine babesiosis are conspicuously absent. Muscle tremors, tachycardia and tachypnoea are observed.

Canine leishmaniosis

Canine leishmaniasis caused by *L. topica* is endemic in the Thar Desert and in some parts of Punjab, Delhi, Haryana and Gujarat. This has assumed greater significance in recent years, owing to the fact that the infection seems to be spreading in the Indira Gandhi Canal region. Both dogs and rodents serve as the zoonotic reservoirs for

cutaneous leishmaniosis in Thar Desert. Dogs are a major reservoir for *L. tropica* and an incidence of 6.8% and 6.12% was recorded during 1985 (Nirban, 1985) and 1993 (Chhangani, 1993).

Canine trypanosomiosis

Canine trypanosomiasis (Surra), caused by *Trypanosoma evansi,* has been studied in India since the beginning of the 20th century. The vectors of *T. evansi* (a salivarian species) are hematophagous flies of the genera *Tabanus* (Diptera: Tabanidae) and *Stomoxys* (Diptera: Muscidae). The infection in dogs is also severe and potentially fatal. Surra is more severe in exotic dogs than in native dogs and serious in pups than in adult dog. The acute form is characterized by sudden death following a few attacks of fever. In the chronic form, which is more common, the clinical signs noticed are fever, oedema of head and throat, corneal opacity or even blindness. Oedema of the larynx changes the voice of the dog that can be confused with that or rabies. Muscular spasm of limbs, staggering gait and excitement like biting of kennel bars, simulating rabies is also seen. The parasitaemia fluctuates with the temperature of the host. Parasites are detectable during fever but are rare during regression of fever. Untreated dogs usually die. Chronic cases are reported in cats. Adult cats showed slight anaemia, progressive loss of weight and discharge from the eyes, nose and oedema of the perineum.

Canine Granulocytic Ehrlichiosis (CGE)

Canine Granulocytic Ehrlichiosis (CGE) caused by *Ehrlichia ewingii,* is a newly emerged disease of dogs. *E. ewingi* is seen in neutrophils and, rarely, eosinophils. CGE classically presents with mild signs including fever, lethargy, anorexia, weight loss, vomiting, diarrhea, severe but transient thrombocytopenia, and transient mild nonregenerative anemia with ineffective erythropoeisis. Commonly, the major presenting clinical signs associated with *E. ewingii* include lameness and joint swelling due to polyarthritis. This form of ehrlichiosis is generally seen in the southern and mideastern United States. Ticks including *Ixodes pacificus, Dermacentor variabilis, Rhipicephalus sanguineus, Amblyomma americanum,,* and *Ixodes scapularis* have been implicated as vectors The diagnosis of CGE differs from that of CME as *E. ewingii* has not yet been cultivated in an *in vitro* system, therefore antigens have not been available for comparative serological testing. Diagnosis of CGE requires visualization of morula within

neutrophils in peripheral blood), joint effusions, and PCR or Western immunoblot. Polyarthritis associated with *E. ewingii* may be self-limiting.

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