An occurrence of clinical eperythrozoonosis in a German Shepherd dog and its therapeutic management

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Abstract *Eperythrozoon spp.*—a rickettsial (Mycoplasma) pathogen of the mammalian erythrocytic cell membrane, has been recognised to cause non specific, sporadic, febrile and haemolytic clinical disease in a variety of livestock, especially food animals. However, clinical eperythrozoonosis in companion animals seems not documented so far. An adult male German shepherd dog, aged 10.5 years and weighing 45 kg with clinical history of persistent mild fever in morning hours, depression and anorexia for the past 5 days and nasal bleeding for the past few months, was presented to clinicians for therapeutic management. Microscopic examination of Giemsa stained blood smear evidenced characteristic light pinkish to blue stained cocci and/or short rod shaped pathogens identified as *Eperythrozoon canis*, nesting in the depressions on the periphery of

erythrocyte cell membrane as well as extra cellular free bodies in the plasma. Rhinoscopic detailed investigation failed to demonstrate any lesion in the nasal passage that could be attributed to the frequent nasal bleeding for the past few months. The dog was subsequently therapeutically managed with success using specific therapy and followed by supportive therapy. Finally, the predisposing factors of the disease, reasons for frequent nasal bleeding, clinical course of the disease in canines and its public health significance vis-à-vis future projections have been discussed.

Keywords Eperythrozoonosis · Rickettsial · Nasal bleeding · Dog

Introduction

Eperythrozoonosis has been incriminated to rickettsial (Mycoplasma) parasite of the mammalian erythrocytic cell membrane, worldwide causing a sporadic, febrile and haemolytic clinical disease in a variety of livestock, especially food animals (Pigs, Sheep and Cattle). In enzootic areas, the parasite is mechanically transmitted by blood feeding dipteran and/or acarine parasites (Urquhart et al. 2003; Taylor et al. 2007). The acute course of the disease is characterised by ictero anaemia, progressively depressed haematological indices, milder fever and death is ascribed to haemolytic anaemia and/or hypoglycaemia (Gwaltney et al. 1993; Lefevre et al. 2010). However, to our knowledge, the occurrence of the disease in companion animals (especially dogs), seems not been documented in the literature so far. However, *E. felis* in cats had been described by Clark as early as in 1942 (Soulsby 2005). This article describes a clinical disease in an aged German shepherd dog and its therapeutic management.

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Materials and methods

An adult male German shepherd dog, aged 10.5 years and weighing 45 kg was presented on 28th April, 2011 before the clinicians at the Teaching Veterinary Clinical Complex, Apollo College of Veterinary Medicine, Jaipur. The animal was brought with clinical history of persistent mild fever during morning hours, depression and anorexia for the past 5 days. In December 2010, the animal was reported to have suffered from nasal bleeding in absence of any sort of physical trauma to the nose. The animal was intravenously administered anti carcinogenic drug (Vincristine “Cytocristin”, Cipla) indiscriminately by the field veterinarian. The owner was defence personnel in the Government of India and belongs to a well to do family. System wise clinical examination of the animal was under taken on spot and the findings were recorded. Laboratory investigations were undertaken of peripheral blood, rectal faecal samples and surface parasite, if any, in order to identify the pathogen (s) using standard techniques (Bowman et al. 2003; Soulsby 2005; Hendrix and Robinson 2006; Taylor et al. 2007). In order to correctly assess the presence of pathological lesions and their impact on nasal vessels suspected for nasal bleeding, rhinoscopy of nasal passage and/or septum was undertaken under general anaesthesia comprising of Propofol (“Propofol”, Neon labs) administered intravenously. Atropine sulphate (“Tropine”, Neon labs) was administered as preanaesthetic sedative. Subsequently, the dog was therapeutically managed with synchronous integrated specific therapy based on the identified pathogen (s) and clinical findings, including intramuscular injection of Ceftriaxone and Salbactam combination (“Vetaceph Plus”, Vetcare). Half gram of this combination was dusted in the nasal passage. 2 ml of Maloxicam (“Melonex”, Intas pharmaceuticals) was given intramuscularly. The animal was given tablet Doxycycline (“Doxy 100”), twice a day for 7 days.

Results and discussions

Clinical examination of the case revealed that the animal was anorectic, lethargic and depressed with dry and lustreless hair coat infested with a few developmental instars of *Rhipicephalus sanguineus* over soft skin areas. The palpable lymph nodes under the skin were neither enlarged in size nor were inflamed and/or oedematous. The conjunctival mucous membranes were pale suggestive of icterus and anaemia. The venous blood was relatively thinner and got coagulated spontaneously. The rhinoscopy examination revealed nasal passage and the septum free from any sort of lesions (granuloma or polyps). The air flow to the lungs was unobstructed. However, nasal

capillaries were delicately engorged with blood and were prone to bleeding. The pulse (115/min) and respiration (34/min) rates were accelerated. The rectal temperature was 101.6°F. Auscultation pulmonary and cardiac sounds were normal.

Microscopic examination of Giemsa stained blood smear evidenced characteristic light pinkish to blue stained cocci (0.5–1.0 µm dia) and/or short rod (1–3 µm long) shaped rickettsial pathogens nesting in the depressions on the periphery of erythrocyte cell membrane as well as extra cellular free rickettsial bodies in the plasma (Fig. 1). At places, the rickettsial bodies were seen attached to the blood platelets and/or thrombocytes. The haematological indices were depressed and lower than normal values (Hb - 6.0 g/dl, PCV - 20%, TLC - $1.1 \times 10^3/\mu\text{l}$, TEC- $4.03 \times 10^6/\mu\text{l}$). The altered differential cell counts were Neutrophils 13%, Lymphocytes 64%, Monocytes 1%, Eosinophils 22% and Basophils 0%. An analysis of the above haematological findings confirmed that the animal has been suffering from anaemia, leucocytopenia, neutropenia, lymphocytosis and eosinophilia. Copro samples were devoid of parasitic ova and/or cyst (s) that could be incriminated as cause of the disease. The dog favourably responded the specific therapy, completely recovered from the clinical course of acute eperythrozoonosis within a week. The altered haematological indices restore to normal levels by day 15 post therapy. No recurrence of the disease has been reported by the owner so far.

Ever since characterisation of the eperythrozoonosis in food animals (Pig, Sheep and Cattle) and man during 1930s, the incriminated pathogen *Eperythrozoon spp.* is considered to be associated with in apparent and/or non specific clinical syndrome. It parasitise the erythrocyte cell membrane, progressively causing icteroanaemia in animals either under stress or indiscriminately exposed to immunosuppressive medication (Henry 1979; Aiello and Mays

Fig. 1 *Eperythrozoon canis* in Giemsa stained smears

1998; Taylor et al. 2007; Lefevre et al. 2010). In this instant case, the subclinical eperythrozoonosis seems pre-disposed to clinical status while indiscriminately using anticarcinogenic drug (Vincristine), incriminated to cause immunosuppression, in absence of definite diagnosis. The prompt and positive response of the animal to specific therapy confirms this fact. Taxonomic placement of the pathogen has been debatable until recent times. It has now been ultimately reclassified under *Mycoplasma* based on 16 s rRNA gene sequences and phylogenetic analysis (Messick et al. 1999; Taylor et al. 2007; Lefevre et al. 2010). The parasite has been associated with two distinct forms of clinical manifestations (a) the acute icteroanaemic clinical syndrome and (b) an inapparent but progressively developing subclinical syndrome, adversely affecting the work efficiency, reproductive cycle and growth of the susceptible host (Henry 1979; Gwaltney et al. 1993).

The clinical picture of the disease in canines as described herein has been suggestive of the acute classical syndrome as described in young pigs elsewhere (Henry 1979; Taylor et al. 2007; Lefevre et al. 2010). The disease is characterised as sequential manifestations of anorexia followed by high fever and ultimately resulting in icteroanemia. The affected animal becomes weak and terminates into death in the event of no specific therapy (Henry 1979; Lefevre et al. 2010). However, clinical history of bleeding from the nose and its spontaneous coagulation is been now documented as special characteristic of canine eperythrozoonosis. This could be attributed to selective concentration of the parasitised erythrocytes in the nasal capillaries obstructing free flow of blood therein, sequential dilatation followed by increased fine nasal blood vascular tendency to rupture analogous to canine babesiosis in the enzootic regions. Speedy development of “haemolytic crises” and spontaneous coagulation of the blood has been documented as a characteristic manifestation of the eperythrozoonosis as well (Lefevre et al. 2010). The altered haematological picture (leucocytopenia, neutropenia, lymphocytosis and eosinophilia) seems to be a fall out of host parasite interaction coupled with response to anti carcinogenic medication and the resultant in situ immunological response of the dog to the pathogen.

In conclusion, above cited description on the clinical haemolytic eperythrozoonosis in canines, that often goes

unnoticed and/or indiscriminately addressed by field veterinarians, has been an eye opener for the academicians, field veterinarians as well as researchers to further investigate host parasite interactions with emphasis on differences between experimental and naturally acquired infections, magnitude of the disease, its pathogenesis and pathophysiological impact on health of the companion pets vis-à-vis public health concern for their owner, improved diagnostic for specific diagnosis of the disease exhibiting non specific signs/symptomatology through critically planned in vivo as well as in vitro studies. The advent of molecular biological techniques may improve the efficiency of detecting the hidden pathogen, causing slow progressing disease in canines. The disease, so far neglected, needs adequate attention.

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