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Rofique Ahmed
Department of Veterinary Epidemiology and Preventive Medicine, College of Veterinary Science, Assam Agricultural University, Assam, India

Mrinal Kumar Nath
Department of Veterinary Epidemiology and Preventive Medicine, College of Veterinary Science, Assam Agricultural University, Assam, India

Utpal Barman
Department of Veterinary Epidemiology and Preventive Medicine, College of Veterinary Science, Assam Agricultural University, Assam, India

Pubaleem Deka
Department of Veterinary Epidemiology and Preventive Medicine, College of Veterinary Science, Assam Agricultural University, Assam, India

Syed Abdul Arif
Department of Veterinary Clinical Medicine, Ethics and Jurisprudence, College of Veterinary Science, Assam Agricultural University, Assam, India

Jonmoni Barua
Department of Veterinary Microbiology, College of Veterinary Science, Assam Agricultural University, Assam, India

Lewamangphika Rapthap
Department of Veterinary Microbiology, College of Veterinary Science, Assam Agricultural University, Assam, India

Ritam Hazarika
Department of Animal Biotechnology, College of Veterinary Science, Assam Agricultural University, Assam, India

Corresponding Author
Rofique Ahmed
Department of Veterinary Epidemiology and Preventive Medicine, College of Veterinary Science, Assam Agricultural University, Assam, India

A case report on therapeutic management of *Anaplasma platys* infection in a labrador

Rofique Ahmed, Mrinal Kumar Nath, Utpal Barman, Pubaleem Deka, Syed Abdul Arif, Jonmoni Barua, Lewamangphika Rapthap and Ritam Hazarika

Abstract
Canine anaplasmosis is a tick-transmitted rickettsial disease caused by *Anaplasma platys*, usually detected as basophilic inclusions in the platelets of thrombocytopenic dogs. Here we describe a clinical case of *A. platys* infection in a dog evident from blood smear and haematological examination. The case was successfully treated with imidocarb and doxycycline, along with other supportive therapy.

Keywords: *Anaplasma platys*, dog, doxycycline, imidocarb, thrombocytopenia

Introduction
Anaplasma platys, previously known as *Ehrlichia platys*, is a rickettsial parasite that specifically infects blood platelets and causes infectious cyclic thrombocytopenia in dogs. *Anaplasma platys*, a Gram-negative, intracellular bacterium in the family *Anaplasmataceae* (Cardoso *et al*., 2010) [11]. *Rhipicephalus sanguineus*, the brown dog tick prevalent in the world's tropical regions, including India (Dantas-Torres, 2010) [16], is considered the primary vector for transmitting the disease. Two pathogenic species of *Anaplasma* cause canine anaplasmosis; *Anaplasma platys* and *A. phagocytophilum*. The disease was first identified in 1978 in dogs from Florida (Harvey *et al*., 1978) [18], and now it has been reported worldwide. Though reports of tick-borne infectious disease among the pet population compared to the livestock species are very few (Rani *et al*., 2011) [1], the hot and humid climatic conditions of the Indian sub-continent, including Assam, is conducive for the pathogen (Borthakur *et al*., 2014) [9].
Clinical and biological signs include fever, depression, anorexia, lymphadenomegaly, splenomegaly, hemorrhagic signs (including dermal petechiae and ecchymoses, epistaxis), weight loss, ophthalmological lesions (including anterior uveitis, chorioretinitis, papilledema, retinal hemorrhage, retinal perivascular infiltrates, and bullous retinal detachment) and neurological disorders. Co-infections with other vector-borne pathogens (VBPs) or intrinsic factors specific to the host (age, breed, physical condition, immune status, or stress) may contribute to a more severe form of the disease (Sparagano *et al*., 2003; Aguirre *et al*., 2006; De la Fuente *et al*., 2006; Yabsley *et al*., 2008; Santos *et al*., 2009; Cardoso *et al*., 2010; Andersson *et al*., 2013; Antognoni *et al*., 2014; Dahmani *et al*., 2015) [22, 2, 17, 23, 21, 11, 5, 6, 15]. The present case describes parasitological evidence of *A. platys* infection in a female Labrador retriever dog from Assam, India.

Case History and Clinical Observations
A five-month-old female Labrador Retriever dog was presented to Veterinary Clinical Complex (VCC), College of Veterinary Science, A.A.U., Khanapara with a history of fever, inappetence, depression, dark yellow coloured urine, epistaxis, and malena. The owner reported that vaccination and deworming were not performed. The abnormal signs during the clinical examination were the rise in the body temperature (104.5ºF), congested mucous membrane, hepatomegaly, splenomegaly, and generalized lymph node enlargement. Ticks were found attached to the body coat during the examination. The blood sample was collected from the cephalic vein in a 3.0 ml K2 EDTA 5.4 mg, BD Vacutainer for Giemsa-stained blood smear examination, and a clot activator for serum biochemical analysis was performed using a Celltac MEK-6450 instrument.

The Giemsa-stained blood smear examination showed *Anaplasma platys* as multiple basophilic inclusions that are morulae within thrombocytes under microscopy. Organisms ranged in shape and appeared to be bound by inner and outer plasma membranes (Figure 1). The details of haematological findings before treatment, i.e., on 18th Oct 2021, and post-treatment, i.e., on 2nd Nov 2021, are presented in Table 1.

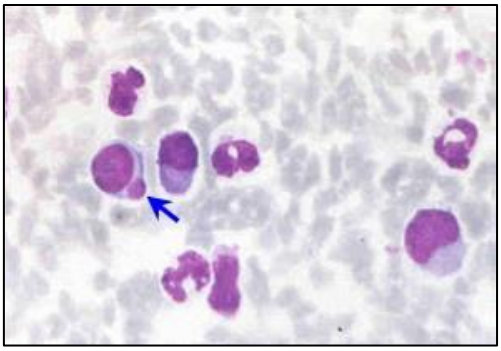

Fig 1: Blood smear showing *Anaplasma platys* infection (inclusions) in thrombocyte.

Table 1: Haematological findings of the affected case before and after the treatment.

Parameters	Pre-Treatment	Post-Treatment	Normal Range
WBC	16.64 m/mm3	15.36 m/mm3	6.0-17.0
Lymphocyte	16.6%	17.1%	10.0-30.0
Monocyte	2.0%	2.2%	2.0-10.0
Granulocyte	81.4%	80.7%	50.0-80.0
Lym#	2.76 m/mm3	2.62 m/mm3	0.6-5.1
Mon#	0.33 m/mm3	0.33 m/mm3	0.1-1.7
Gra#	13.55 m/mm3	12.41 m/mm3	3.0-13.6
RBC	R 6.08 M/mm3	6.80 M/mm3	5.5-8.5
MCV	R 60.9fl	58.5 f1	58.0-73.0
Hct	R 37.0%	39.7%	35.0-55.0
MCH	34.8 pg	20.0 pg	19.5-24.5
MCHC	57.2 g/dl	34.2 g/dl	28.0-40.0
Haemoglobin	R 21.2 g/dl	13.6 g/dl	10.0-18.0
Thrombocyte	91 m/mm3	125 m/mm3	120-600

Treatment And Discussion

Based on the microscopic examination, the case was diagnosed as an *Anaplasma platys* infection, and treatment was initiated with Imidocarbdipropionate (Babimido®) @ 6.6 mg/kg BW and Atropine sulphate (inj. Tropin®) @0.04mg/kg BW subcutaneously and repeated after 14 days. Doxycycline (Doxicip®) @ 10 mg/kg B.W. orally daily for 14 days along with Ranitidine (Aciloc®) @ 0.5mg/kg B.W. BID for 14 days. Inj. Vetalgin @ 0.5mg/kg BW was administered as antipyretic. Apart from the ongoing treatment, supportive drugs like Venkys Thromb Beat Syrup, Elfate-O Suspension, Gutwell powder were prescribed. After 48 hours of initiation of therapy, the animal showed improvement in appetite and activity. On the 14th post-treatment day, the animal showed complete uneventful recovery evident from the marked improvement in the clinical condition and the haemato-biochemical parameters (Table 1).

Fig 2: Dull and depressing pre-treatment

Fig 3: Active and healthy post-treatment

The clinical findings recorded in the present case were similar to reports of *Anaplasma platys* infection in the dog (Sainz *et al*., 2015; Carvalho *et al*., 2017; Bouzouraa *et al*., 2016; Alhassan *et al*., 2021; Bhoopathy *et al*., 2017) [20, 12, 10, 4, 7]. Detection of inclusion bodies in blood platelets is possible in an earlier stage of infection only and is non-specific later as there are non-parasitic inclusions within the figured elements (Ferreira *et al*., 2007). The present case was diagnosed based on microscopic examination of blood smears that showed *A. platys* morulae within the thrombocytes and blood profile.

The use of imidocarb dipropionate is approved by the Food and Drug Administration (FDA) in the United States of America (USA) for the treatment of *B. canis* infections in dogs, but it is also effective in the treatment of other haemo-protozoal diseases, including *A. platys* (Salah *et al*., 3013; Conrad *et al*., 1991; Birkenheuer *et al*., 2004a; Checa *et al*., 2017) [14, 8, 13]. The FDA-approved labelled dose of imidocarb propionates is 6.6 mg/kg intramuscularly (IM) or subcutaneously (SC) with a repeated dose in 14 days. The mechanism of action of Imidocarb dipropionate is uncertain though two mechanisms have been proposed: interference with the production and/or utilization of polyamines or prevention of entry of inositol into the erythrocyte containing the parasite. The adverse effects of imidocarb dipropionate in dogs include pain during injection and cholinergic effects such as salivation, drooling, nasal drip, or vomiting. These cholinergic effects can be mitigated by atropine at 0.05 mg/kg premedication. Doxycycline (10mg/kg BW for 14 days) is used to eliminate the organism (Leah *et al*., 2009) [19]. Therefore, a similar therapeutic approach was initiated along

with supportive therapy, after which the case showed considerable improvement in clinical signs and the haemato-biochemical parameters.

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