Transplacental transmission of *Babesia equi* (*Theileria equi*) from carrier mares to foals

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Abstract This article communicates the first per-acute and fatal clinical report of transplacental transmission of *Theileria equi* from carrier mothers to foals from Punjab, India. Two cases of equine piroplasmosis due to *T. equi* were diagnosed in neonatal foals borne to Thoroughbred mares. High degree of parasitemia with annular and maltese cross shaped parasites in erythrocytes was seen in both cases. Blood cellular changes revealed leucopenia and neutropenia with mild degenerative left shift. Anaemia was macrocytic normochromic type. Intense yellow discoloration of mucous membranes indicated jaundice. In areas endemic for equine piroplasmosis, jaundice in neonatal foal can be easily misdiagnosed as neonatal isoerythrolysis. Foals with post-partum jaundice should always be screened for equine piroplasmosis.

Keywords Babesiosis · Equine · Foal · *Theileria equi* · Transplacental-transmission

Introduction

Equine piroplasmosis, a tick-transmitted haemoparasitic disease, poses a serious threat to equine health. Out of the two species infecting equines, *Babesia equi* (*Theileria equi*) is more pathogenic and more consistent cause of haemoglobinuria and death in equines; while *B. caballi* causes a persistent syndrome of fever and anaemia. There is no confirmed report on the occurrence of *B. caballi* from India. Ixodid ticks of genera *Rhipicephalus*, *Hyalomma* and *Dermacentor* transmit these parasites (de Waal 1992). However, in India, ticks of *Hyalomma* species are considered the most common vector for the parasite (Kumar et al. 2009). The clinical signs of equine piroplasmosis are often variable and non-specific. Acute infection results in fever (40°C), depression, reduced appetite, pallor, icterus, dyspnoea, petechiation, colic, eyelid and distal limb oedema and incoordination (de Waal and van Heerden 2004). The chronic disease generally presents variable clinical signs, making the diagnosis difficult (de Waal 1992). Equine piroplasmosis can also be transmitted iatrogenically via the common use of blood contaminated syringes and needles (de Waal and van Heerden 2004). Transplacental transmission of *T. equi* has been reported in U.K. and Trinidad (Phipps and Otter 2004; Georges et al. 2011). In utero infection of foals by *T. equi* can result in abortions, full term still births or birth of the live foals with neonatal piroplasmosis (Allsopp et al. 2007; Phipps and Otter 2004). Transplacental transmission of tropical thel-eriosis in a calf has already been reported from India (Godara et al. 2009). However, to our knowledge, trans-placental transmission in natural cases of *T. equi* infection in foal has not been reported so far from India. Therefore, this article communicates the first per-acute and fatal transplacental transmission of *T. equi* in foals from India.

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Case description

Two neonate foals (foal I and II) were presented to Teaching Veterinary Hospital, Guru Angad Dev Veterinary and Animal Sciences University, Ludhiana for treatment. Foal I was born full term with normal size, vitality and physiological functions to a 10-year old thoroughbred mare. Owner reported that it was apparently in good health on day 1 of birth. It was presented when 2 days old. On clinical observation the foal was weak and icteric and was unable to suckle. Marked dyspnoea was noted. Blood sample was collected for haematological analysis using EDTA @ 2 mg/ml as anticoagulant. Wright-Giemsa stained thin blood smears revealed the presence of piroplasms in more than 50 per cent of red blood cells (Fig. 1). A stress leukogram of leucopenia, lymphocytosis with thrombocytopenia was evident (Table 1). The foal was treated by intravenous fluid, supportive and emergency medicine but died soon after initiation of the therapy. Microscopic examination of a Wright-Giemsa stained peripheral blood smear of the mare was, however, negative for piroplasms.

Foal II was borne apparently healthy after the completion of full term of pregnancy. The foal at the time of birth was normal and sucked mother's milk for 1 day. But, next day, foal started showing symptoms of weakness, yellow urination and recumbency, followed by cessation of urination and defecation. Mucous membrane turned intense yellow. The foal at the time of presentation was in lateral recumbency, comatose with stiff neck directed upward. The rectal temperature was 104.2°F and heart rate was 110/min. Polypnea with marked abdominal respiration was also evident. Ecchymotic haemorrhages and icterus were present on palpebral conjunctiva and the entire skin covering ventral abdomen appeared intense yellow. Peripheral blood picture revealed high parasitemia (>40%),

Fig. 1 Peripheral blood smear showing piroplasms in erythrocytes

Table 1 Haematological values of foals suffering from *T. equi* infection

Parameter	Reference range ^a	Foal I	Foal II
RBC ($\times 10^{12}/l$)	9.1–11.9	7.22	6.8
Hb (g/dl)	12.9–15.5	13.0	11.4
HCT (l/l)	0.38–0.46	0.43	0.38
MCV (fl)	36.0–44.0	59.5	55.8
MCHC (g/l)	32.0–360.0	302.2	300
WBC ($\times 10^9/l$)	6.0–12.0	2.2	2.5
Neutrophils (%)	58.0–79.0	56.0	58
Lymphocytes (%)	15.0–35.0	42.0	41
Monocytes (%)	1.0–7.0	2.0	1.0
Eosinophils (%)	1.0–10.0	0.0	0.0
Platelets ($\times 10^9/l$)	100.0–600.0	37.9	–

RBC red blood cell, Hb haemoglobin, HCT haematocrit, MCV mean corpuscular volume, MCHC mean corpuscular haemoglobin concentration, WBC white blood cell

^a Schalm's veterinary hematology, 6th Edn (ed., Weiss and Wardrop 2010)

macrocytic normochromic anaemia and stress leukogram with leucopenia, lymphocytosis and neutropenia (Table 1). The foal died next day, despite all possible efforts to save its life with supportive treatment.

Results and discussion

A high parasitemia was noted in the thin peripheral blood smears in both the cases. There were on average 1–4 organisms per infected RBC. Both ring forms and maltese cross were seen. *B. equi* initially invades lymphocytes producing macro- and micromeronts. These finally enter red cells. Here they reproduce by simultaneous formation of four merozoites called maltese cross. Based on the morphological features (less than 2.5 μ m in length) of the parasite, the foals were found to be infected with *T. equi* (Soulsby 1982). Parasitemia commonly range from 1–5 per cent, but up to 80% of RBCs may be infested in some cases (de Waal and van Heerden 2004; Mehlhorn and Schein 1998). Piroplasms of *T. equi* can be transmitted across the equine placenta and once a horse is infected, it appears to remain a life-long carrier, since anti-theilerial drugs suppress but do not eliminate the parasite. Carrier mares may transmit the organism to their offspring and this may result in abortion or neonatal piroplasmosis; but observations by some researchers suggest that foals may be born as carriers yet remain apparently healthy (Allsopp et al. 2007). The RBC and haematocrit (HCT) levels were on the low end of the reference interval of foals (Weiss and Wardrop 2010).

Jaundice, reduced number of red blood cells and platelets, reduction in haemoglobin concentration and leukopaenia are

common in equine babesiosis (de Waal 1992). During the first 12 h, transfusion of placental blood to the foal from the mare increases the foals RBC concentration in blood, which then decline sharply over 12–24 h (Weiss and Wardrop 2010). If icterus is not observed immediately after birth, these cases of neonatal piroplasmosis could have been mistaken for neonatal isoerythrolysis. Allsopp et al. (2007) suggested that neonatal equine piroplasmosis may be acquired during days 40–150 of gestation. In normal course, trans-stadially *T. equi* infected equine requires a minimum incubation period of 12–19 days (de Waal and van Heerden 2004) after sporozoite inoculation through tick bite. During this period, the in situ fast multiplying *T. equi* successively develops in lymphocytes as macro- and microschorizonts. Ultimately a large number of merozoites are released to invade healthy erythrocytes and multiply therein as piroplasms. Evidently, possibilities of transtadial transmission of *T. equi* were remote in the 2-day-old foal. A report of transplacental transmission of *T. equi* indicates that carrier animals may act as reservoirs to establish equine piroplasmosis in areas where competent tick vectors may be present (Phipps and Otter 2004).

From the present report, it can be concluded that in areas endemic for equine piroplasmosis, any newborn foal presented with signs of jaundice, should be investigated carefully for vertical or transplacental infection of *T. equi*. Appropriate preventive measures should be advised to the

owner for reducing losses in subsequent parturitions of the mare harbouring this insidious parasite.

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