CASE REPORT

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# *Babesia caballi* infection in a 6-month-old colt

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**Abstract**

On the 15th of August 2016, a 6-month-old colt weighing 118 kg was presented to the large animal clinic of Veterinary Teaching Hospital, Michael Okpara University of Agriculture, Umudike. The colt showed clinical signs including fever, weakness, inappetence, anaemia, tachycardia, dyspnoea, rough hair coat, pale mucus membranes, lacrimation, ataxia and edematous swelling of distal limbs. *Rhipicephalus eversti eversti* ticks were detected on different parts of the colt's body. Examination of peripheral blood smear showed *Babesia caballi* within the erythrocytes. Haematological analysis showed a decrease in red blood cell count, packed cell volume and haemoglobin concentration. Administration of imidocarb dipropionate and good nursing care yielded a successful result after two weeks of treatment. It is necessary to control ticks by regular use of acaricide and timely treatment of affected horses in order to reduce the devastating effects of this protozoan disease.

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## Introduction

Equine babesiosis (piroplasmosis) is a protozoan disease of horses caused by *Theileria equi* (formerly *Babesia equi*) and *Babesia caballi* (Mehlhorn & Schein, 1998). Horses, donkeys, mules and zebras are susceptible (Rothschild, 2013). The disease is transmitted by ticks that include *Dermacentor, Rhipicephalus and Hyalomma* species (Battsetseg *et al.,* 2001). Ticks are reservoirs of infection because the infection persists in ticks throughout several generations with transtadial and transovarian transmission. Horses usually remain carriers of *B caballi* for one to three years after infection and potential disseminators of the parasites (Friedhoff *et al.,* 1990; Guidi *et al.,* 2015). Babesiosis is more endemic in most tropical and subtropical areas than in temperate regions due to high ambient temperature, humidity and rainfall which favour tick development (Motlong *et al*., 2008). Case fatality rate of equine babesiosis is about 10 – 50%. Animals in endemic areas mostly survive the infection (OIE, 2009), but acutely affected horses may have little chance of survival without treatment (Morrow and

Sommardahi, 2014). Symptoms may differ from per acute to chronic forms. Horses are found dead or moribund in per acute form. In the acute form, clinical signs include: fever, anorexia, depression, anaemia, elevated respiratory and pulse rates, congested mucous membrane, icterus, hemoglobinuria, and colic (Morrow & Sommardahl, 2014). This form coincides with appearance of organism in peripheral blood and can last for 8 to 10 days. If recovery occurs, temperature returns to normal and the animal becomes a carrier. The subacute disease develops more slowly, is more prolonged, and recovery may take several weeks or months. Clinical signs seen in this form include those of acute form in addition to pale mucous membrane, mild colic, poor exercise tolerance, posterior weakness, incordination, walking in circles and mild oedematous swelling of the distal part of the limb (Zobba *et al.,* 2008). The chronic form usually appears after an acute phase; clinical signs are not specific and include loss of condition, poor exercise tolerance and slow recovery (Radostits *et al.,* 2007). Occasionally, atypical forms may include symptoms like gastro-enteritis, bronchopneumonia and abortions. Post mortem findings include intravascular haemolysis, jaundice, emaciation, enlarged spleen and liver, pale kidney and oedema in lungs. Babesiosis is usually diagnosed by demonstration of babesia parasites in the infected erythrocytes using Giemsa smears or by positive serology (Sakha, 2007). This paper reports a successful treatment of *Babesia caballi* infection in a 6-month-old colt.

**Case Report**

*History and physical examination*

On the 15th of August 2016, a 6 month old colt weighing 118 kg, belonging to the Veterinary Teaching Hospital (VTH), Michael Okpara University of Agriculture, Umudike was presented to the large Animal Clinic of VTH, Michael Okpara University of Agriculture, Umudike. Three days before presentation, the sick colt had a history of weakness, anorexia, depression, paleness of the mucous membrane, oedema of the distal part of the limbs, ataxia and difficulty in rising. There was no history of deworming and the dam died of colic when the colt was 5 months old. It was fed on wheat bran and sorghum and had access to fresh pasture and water. On examination, it was observed that heart rate was 52 beats per minute (bpm), respiratory rate, 29 cycles per minute (cpm) and temperature, 38.8$^{o}$C. Physical examination showed moderately pale mucous membrane, lacrimation, dyspnoea, ataxia and oedema of the distal part of the limbs. *Rhipicephalus eversti eversti ticks* were found on different parts of the body. A differential diagnosis of equine babesiosis, African horse sickness and equine ehrlichiosis was made.

Haematological analysis showed a decreased red blood cell count (RBC), packed cell volume (PCV) and haemoglobin concentration (Hgb) (Table 1). The white blood cell and differential counts were within normal range. Peripheral blood smear revealed *Babesia caballi* (Plate I). Tentative diagnosis was equine babesiosis based on the clinical signs and appearance of large, paired merozoites joined at the posterior ends in the erythrocytes.

*Management and treatment*

The foal was hospitalized and then treated with diminazene aceturate (Berenil®, Hoechst, Germany) given at dose rate of 3.5 mg/kg deep im twice one week apart. Ivermectin at the dose rate of 200µ/kg was given once in addition with 1.5 ml of vitamin B complex intramuscularly three times at 24 hours intervals and 1000ml of dextrose 5% in water given subcutaneously twice at 24hours apart as supportive treatment.

**Table 1**: Haemogram before treatment and after one month of treatment

| | Before initial treatment | After one month of treatment | Normal Range* |
|---|---|---|---|
| PCV (%) | 29 | 30 | 32-53 |
| RBC ($10^6$/µl) | 6.0 | 6.4 | 6.8-12.9 |
| Hb concentration (g/dl) | 9.0 | 8.6 | 11.0-12.9 |
| WBC (×$10^9$/L) | 6.9 | 7.1 | 5.4 – 14.3 |
| Neutrophils (×$10^9$/L) | 5.2 | 5.1 | 2.3 – 8.5 |
| Lymphocytes (x $10^9$/L) | 1.7 | 1.8 | 1.5 – 7.7 |
| Monocyte (x $10^9$/L) | 0.0 | 0.07 | 0 – 1.0 |
| Eosinophils (x $10^9$/L) | 0.07 | 0.07 | 0 - 1.0 |
| Basophils (x$10^9$/L) | 0 | 0.07 | 0 – 0.3 |

* Grondin & Dewitt (2010)

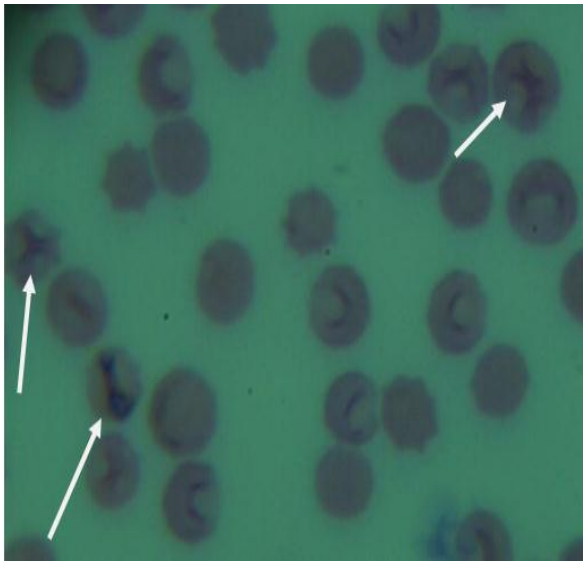

**Plate I**: Peripheral blood smear showing *Babesia caballi* merozoites (arrows) in erythrocytes (x100 oil magnification)

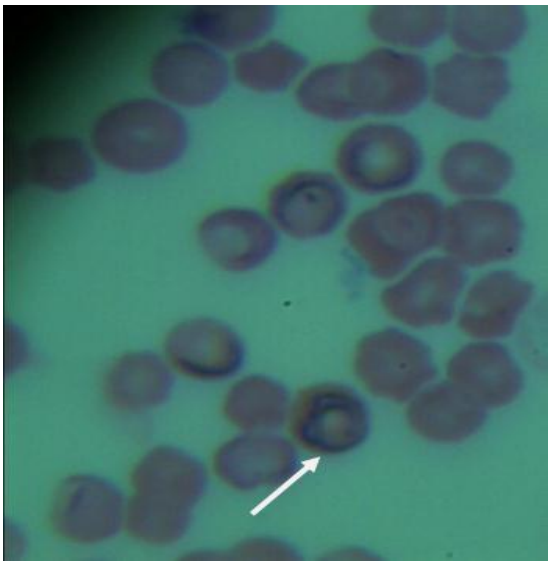

**Plate II**: Peripheral blood smear showing *Babesia caballi* merozoite (arrow) in erythrocytes (x100 oil magnification)

The foal showed no remarkable improvement after one month of treatment and could remain recumbent for a whole day in its urine and faeces during this period. Much effort was required to get the foal on its feet for physical examination. However, it was alert and had good appetite.

After one month of initial treatment, the foal was re-examined and temperature was 39.9$^{o}$C, respiratory rate, 60cpm, heart rate, 48bpm, mucus membrane was pale and body weight reduced to 107 kg. The second blood analysis still revealed decrease in RBC, PCV and Hb (Table 1) while the Giemsa stained blood smear also revealed presence of *Babesia caballi* within the erythrocytes (Plate II).

The treatment plan was therefore reevaluated and the foal was then given imidocarb dipropionate (Imizol®, Carbesia®, Mallinckrodt, United Kingdom) intramuscularly at the dose rate of 2.2 mg/kg body weight twice 24 hours apart. The vital signs, appetite and mucous membrane returned to normal, oedema on the dependent parts regressed and the foal was able to stand on its own after two weeks of the treatment.

**Discussion**

The infected foal showed varying clinical signs of the disease seen in horses with babesiosis which were in agreement with the findings of Aslani (2000), Sakha (2007), Al Saad (2009) and Garba *et al.* (2011). The appearance of large, paired merozoites (pear-shaped) joined at the posterior ends in the equine erythrocytes is diagnostic feature of *B. caballi* infection (Plates I and II), same was recorded by Butler *et al*. (2005) and OIE (2014). The haemolytic anemia seen is from the physical rupturing of erythrocytes (intravascular haemolysis) during release of merozoites, and the removal of infected erythrocytes from the circulation by the spleen (extravasular haemolysis). Haematologically, all these resulted in low erythrocytes count, haemoglobin concentration and PCV (Al Saad 2014; Mahmoud *et al.,* 2016; Mark, 2016). Kahn & Line (2006) reported that the decrease in RBC count and consequent low level of Hb concentration prevented the body tissues and organs from getting enough oxygen. The effect is the dysnoea and weakness observed in the sick colt. The decrease in RBC count, Hb concentration and PCV suggested macrocytic hypochromic anemia and these were similarly reported by Zobba *et al*. (2008) and Mahmoud *et al.* (2016). Weight loss might have been due to uptake of energy by piroplasms in form of adenosine triphosphate (ATP) and adenosine monophosphate (AMP) thereby depriving the sick colt of essential energy needed for normal anabolic process (Camacho *et al.,* 2005). The *Rhipicephalus evertsi evertsi* infestation on different parts of the body might have been responsible for the transmission of the disease. De Waal & Potgieter (1987) reported that *Rhipicephalus evertsi evertsi* transmits *B caballi* in horses. Diminazene aceturate and imidocarb dipropionate are the drugs of choice in the treatment of equine piroplasmosis (Morrow & Sommardahl, 2014). Diminazene aceturate given at

3.5 mg/kg IM twice one week apart was unable to clear *B. caballi* infection while imidocarb dipropionate 2.2 mg/kg IM given twice at 24 hours interval was able to clear persistent *B. caballi* infection in the foal. It has been reported that a relatively high dose of imidocarb dipropionate not only eliminated *B. caballi*, but also left the horses incapable of transmitting babesiosis (USDA, 2009). Reason for the failure of diminazene aceturate therapy may be due to the treatment interval. Success alleviation of clinical signs has been reported when diminazene aceturate was given at 3.5 mg/kg IM twice at 48 hour intervals (Rashid *et al.,* 2008). Ivermectine was given against ticks, dextrose water administered, provided energy to the weak foal and vitamin B complex was given to enhance appetite and boost the immune system. Maintaining appetite was critical for recovery. Hospitalization, good nursing, provision of balanced concentrate and exercise also contributed to the recovery.

In conclusion, this case report indicated a successful treatment of babesiosis due to *Babesia caballi* with the anti-protozoan drug, imidocarb dipropionate and supportive therapy. Tick control by the use of acaricides, isolation and treatment of infected and carrier animals are important measures of protecting horses from babesiosis

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