

SEVERED HEMOPARASITOSIS COMPLICATED IN A THOROUGHBRED HORSES FROM VENEZUELA

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ABSTRACT: *Equine piroplasmosis (EP) is the disease caused by protozoan hemoparasites Babesia caballi and/or B. (Theileria equi). The aim of this study was to report of case of severed hemoparasitosis complicated in a Thoroughbred horse from Venezuela. The equine present acute abdominal pain in 24 hours and complicated post 48 hours and death in the 72 hours. Temperature oscillate 41;2°C. Icterus and hematury severed. We realized a multidisciplinary study clinical, laboratory, biochemistry, necropsy, histopathology. Hematies: 10.6 mm³, Hb: 16.4 g/dL, Hto: 48%, plaques 305mm³, Leucocytes 4.7 x/mm³, Neutrophiles 47%, Lymphocytes 44%, Monocytes 1%, Eosinophyles 7%. Total Protein 7,2g/dL, Albumine 2.8g/dL, globulin 4.4g/dL, glicemia 85mg/dL, urea 29mg/dL, creatinine 3.0 mg/dL. BT 3.07mg/dL, BD 0.33mg/dL, BI 2.74mg/dL, GOT 416UI/I, CK 183 UI/I, Na 136mmol/dL, K 3.0 mmol/dL, Cl 98 mmol/dL, fibrinogeno 300mg/dL. Giemsa staining of blood smears followed by careful microscopic examination can reveal the intraerythrocytic parasites in acute cases. B. caballi can appear pyriform-shaped and occurs in pairs whereas B. equi appears as four pyriform parasites in a Maltese-cross formation. On necropsy, were observed severed icterus oral and mucosa, xantomathosis of subcutaneous tissue. Poliserositis, ascitis, anasarca. In the abdominal cavity was observed massive hemoperitoneum severe, adhesive fibrinous peritonitis by diapedesis. Spleen with hemosiderosis severed. Peritonitis fibrinous adhesive in external surface of small intestine. Severe disseminate coagulate intravascular, hemolysis acute and bacteraemia, septicaemia. In conclusion were reported of case of severed hemoparasitosis complicated in a Thoroughbred horse from Venezuela.*

KEYWORDS: hemoparasites, equine, Piroplasmosis.

HEMOPARASITOSIS SEVERA COMPLICADA EN UN PURA SANGRE DE CARRERAS EN VENEZUELA

RESUMEN: *La Piroplasmosis equina (EP) es una enfermedad causada por el protozoo hemoparásitos Babesia caballi y / o B. (Theileria equi). El objetivo de este estudio fue reportar un caso de hemoparasitosis severa complicada en un caballo Pura Sangre de Venezuela. El equino presento dolor abdominal agudo en 24 horas y 48 horas edema de miembros posteriores y muerte en las 72 horas. La temperatura oscilo entre 41, 2 ° C, presento ictericia y hematuria. Se realizó un estudio multidisciplinario clínicos, de laboratorio, bioquímica, necropsia e histopatología. Hematies: 10,6 mm³, Hb: 16,4 g / dl, Hto: 48%, plaquetas de 305 mm³, los Leucocitos 4,7 x / mm³, neutrófilos 47%, linfocitos 44%, monocitos 1%, Eosinófilos 7%. Total de proteínas 7,2 g / dl, Albúmina 2.8g/dL, 4.4g/dL globulina, glicemia 85mg/dL, urea 29mg/dL, creatinina 3,0 mg / dl. BT 3.07mg/dL, BD 0.33mg/dL, BI 2.74mg/dL, GOT 416UI / l, CK 183 UI / l, Na 136mmol/dL, K 3,0 mmol / dl, Cl 98 mmol / dL, fibrinógeno 300mg/dL. El frotis de sangre coloreado con Giemsa seguido por el examen microscópico revelo parásitos intraeritrocíticos. B. caballi puede aparecer en forma piriforme y se produce en parejas, mientras que B. equi aparece como cuatro parásitos piriformes en una formación de Cruz-Malta. En la necropsia, se observo la mucosa oral y conjuntival icterica, xantomathosis del tejido subcutáneo. Poliserositis, ascitis, anasarca. En la cavidad abdominal se observó hemoperitoneo masivo peritonitis fibrinosa severa adhesiva por diapédesis. Bazo con hemosiderosis, Coagulación intravascular diseminada, hemólisis aguda, bacteremia y septicemia. Se reporto un caso de hemoparasitosis severa complicado en un caballo pura sangre de Venezuela.*

PALABRAS CLAVES: hemoparasitos, equino, Piroplasmosis.

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INTRODUCTION

Equine piroplasmosis (EP) is the disease caused by protozoan hemoparasites *Babesia caballi* and/or *B. (Theileria) equi*. Are obligate intracellular parasites of the phylum Apicomplexa and cause hemoparasitic diseases in horses (1). The natural transmission of these parasites is through competent tick vectors (2). Incidence of clinical infection with *Babesia* organisms is highly variable from location-to-location. Infection depends on many factors: 1) an animal reservoir; 2) a *Babesia* to which humans are susceptible, or a human host who is asplenic or otherwise immunocompromised; 3) a genus of Ixodidae (hard bodied tick) which can transmit the parasite; and 4) a suitable tick habitat. Finally, humans must put themselves at risk by frequenting a tick habitat in an endemic area in the months when infective stages of ticks are feeding. Transfusion transmission can occur anywhere and outside of tick-feeding season, because blood for transfusion can be collected in a different region of the country and asymptomatic donors may carry the parasite (3).

Horses infected with either agent have similar clinical signs. Clinical signs of EP can include fever, anemia, icterus, and anorexia. Digestive tract signs can occur, including colic, constipation, or diarrhea (2). Thus, in some regions of the world where infection is common, little or no clinical disease may be observed in native horses. However, disease is frequently observed in adult horses suddenly introduced into areas with large numbers of infected ticks (2). The aim of this study was to report of case of severe hemoparasitosis complicated in a Thoroughbred horse from Venezuela.

MATERIAL AND METHODS

History: an equine mare Thoroughbred, of 2 year old. The equine present acute abdominal pain in 24 hours and complicated post 48 hours and death in the 72 hours.

Clinical sign: Fever: Temperature oscillate 39.4 °C; 39.6 °C (24 hours), 39.7 °C (48 hours), 40.1 °C; 41;2 °C (72 hours).

Icterus: severe 48 hours.

Hematury: severe 48 hours.

Acute abdominal pain: gastric dilatation, persistent abdominal pain, intestinal motility normal, loss major colon; no refluxed in the 48 hours and abundant and severe refluxed.

Cardiac Frequency: 68, 68 (24 hours), 92, 84 (48 hours), 84, 92 (72 hours), 120, 120 lpm.

Laboratory Test: Bloods of sample were collected previous at mortem.

Treatment: emergence acute abdominal pain: acepromazine, xilacine, Dipirona, flunixin meglumine, clorure potassium, colicor, Ringer lactated 24L, 24L, 40L.

Infectious: Penicillin, enrofloxacin, diminazeno, ivermectina, vitamin B12.

Necropsy: Were examined by necropsy and samples of tissue were recollected (4, 5).

Histopathology: The tissue samples were fixed in formalin and processed by conventional H&E techniques (5).

RESULTS

Laboratory results were: Hematias: 10.6 mm³, Hb: 16.4 g/dL, Hto: 48 %, plaques 305 mm³, Leucocytes 4.7 x/mm³, Neutrophiles 47 %, Lymphocytes 44 %, Monocytes 1 %, Eosinophiles 7 %. Total Protein 7.2 g/dL, Albumine 2.8 g/dL, globulin 4.4g/dL, glicemia 85 mg/dL, urea 29 mg/dL, creatinine 3.0 mg/dL. BT 3.07 mg/dL, BD 0.33 mg/dL, BI 2.74 mg/dL, GOT 416 UI/L, CK 183 UI/L, Na 136mmol/dL, K 3.0 mmol/dL, Cl 98 mmol/dL, fibrinogeno 300mg/dL.

Giemsa staining of blood smears followed by careful microscopic examination can reveal the intraerythrocytic parasites in acute cases. *B. caballi* can appear pyriform-shaped and occurs in pairs whereas *B. equi* appears as four pyriform parasites in a Maltese-cross formation. On necropsy, were observed severe icterus oral and mucosa, xantomatosis of subcutaneous tissue. Poliserositis, ascitis, anasarca. In the abdominal cavity was observed massive hemoperitoneum severe, adhesive fibrinous peritonitis by diapedesis. Multiple hemorrhages in the adrenal cortex. Liver with hemorrhages was fibrosis chronic. Multifocal necrotic areas were present in the other lobes. Renal cortical and papillary necrosis, hematury. Spleen presented severe congestion, hemorrhage. Gastric ulcer syndrome severe. Petechiae epicardial hemorrhage. Oedema, congestion and hemorrhage pulmonary. Plastron mesenteric in abdomen cranial. Histopathology revealed severe fatty degeneration and hepatic necrosis. Hemorrhage in adrenal cortex with coagulation necrosis of zone glomerulosa, zone fasciculata and zone reticularis. Acute tubular necrosis, vacuolar degeneration and glycogen nephrosis, hemoglobinuria, glomerulonephritis membranous severe. Spleen with hemosiderosis severe, germinal center development within the lymphoid follicles should be noted as decreased. Reactive extramedullary hematopoiesis may be seen in conjunction with conditions that target the destruction of lymphocytes. Decreased cellularity of the lymphoid follicles, marginal zone and red pulp region were presented. Chronic gastritis surface, erosion focal and hyperkeratosis infiltrated of lymphocytes in the lamina propria. Peritonitis fibrinous adhesive in external surface of small intestine. Severe disseminate coagulate intravascular, hemolysis acute and bacteraemia, septicaemia.

DISCUSSION

The exact pathogenesis of equine babesiosis is not known completely, but metabolic stress placed on the parasitized erythrocytes may cause hypophosphatemia and weakening of erythrocytic cell membranes causing hemolysis (1, 7). Parasitized red cells lyse intravascularly, producing hemoglobinemia in the acute phases of the disease (1, 7). The intravascular hemolysis causes a marked hyperbilirubinemia and icterus can be pronounced in many cases. Hemoglobinuria seems to occur more frequently and severely with *Babesia equi* infections, but can be seen in horses infected with either species. In addition to regenerative hemolytic anemia, a significant monocytosis and eosinopenia may be observed in horses with babesiosis. In severe disease, the intravascular hemolysis produced by the infection may disturb capillary blood flow enough to cause disseminated intravascular coagulation (DIC) and resulting signs of coagulopathy (7). Along with appropriate clinical signs and pathologic findings of hemolytic anemia, the identification of the parasitized erythrocytes on routinely stained blood smears is diagnostic. In cases of chronic or subacute babesiosis, it may not always be possible to visualize the organisms on blood smear examination. In these situations, PCR, ELISA, serology, or a combination of these tests is used to establish a diagnosis. The most commonly used serologic tests are the complement fixation (CF) and indirect fluorescent antibody (IFA) tests.

Gross necropsy findings of horses with babesiosis include thin watery blood, icterus, effusions of the body cavities and pericardium, hepatomegaly, and splenomegaly (1, 2, 7). Several types of polymerase chain reaction (PCR) tests have been developed and are used for research purposes. What role the PCR test will play in the future for determining a horse's infection status is still being determined (2). The recent babesiosis literature continues to support the contention that the pathophysiology of babesiosis has much in common with malaria, sepsis and other systemic inflammatory states. It is now recognised that sepsis can result in both excessive and inadequate inflammation (6, 7).

A wide variety of factors may precipitate sludging: endothelial fibrin and red blood cell bound fibrinogen; strand-type knob-forming membrane changes in parasitized red blood cells; increased plasma viscosity caused by fibrin, fibrinogen, and their complexes; and erythrocyte lipid changes such as externalization of phosphatidylserine. The cellular immune system, including production of cytotoxic TNF-alpha, plays an important role in many of these clinical changes. Parasite and host derived proteases, plasma kallikrein, thrombin and thrombin-like enzymes, and anoxia cause alveolar edema and

an accumulation of neutrophils and red blood cells in pulmonary capillaries (3). Other complications of babesiosis have included retinal infarct, encephalopathy, hepatic failure, renal failure, hemophagocytic syndrome, and autoimmune hemolytic anemia (3). The reticuloendothelial system plays a vital role in nonspecific resistance (3). Specific resistance depends on both humoral and cell-mediated immunity. *Babesia* infection elevates levels of IgG and IgM in animals and humans (3). In vitro observations suggest that immunity may be influenced by humoral factors; phagocytosis of infected cells by splenic macrophages requires antibody, complement, and conglutin (3). The complex interactions between pro- and anti-inflammatory responses were elegantly illustrated in bovine babesiosis (6). A hypo inflammatory immune response might be as detrimental as excessive inflammation, and improved outcome might be achieved if individual immune statuses were known and treatment tailored accordingly (1, 2, 6, 7). This area of investigation is highly relevant to canine babesiosis. In addition to altered immune status, there can be little doubt that babesiosis is characterised by abnormal perfusion and tissue hypoxia, which has major implications for the function of every organ and system in the body. It could be convincingly argued that all critical illness can ultimately result in abnormal tissue perfusion, but it is not difficult to allow that virulent babesiosis and malaria are special cases, in that they involve large intravascular organisms that remain in the vasculature and sequester there.

Profound intravascular, vascular and perivascular pathology has been described (1). There are many potential contributing factors to tissue hypoxia in babesiosis, including microvascular sequestration, severe anemia, altered erythrocyte deformability and hemoglobin function, auto-agglutination, endothelial activation and damage, increased endothelial permeability, disseminated intravascular coagulation, hypotension, pulmonary edema and myocardial dysfunction (7).

In conclusion were reported of case of severed hemoparasitosis complicated in a Thoroughbred horse from Venezuela.

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D. Villoria y col.

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