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CASE REPORT

Fatal acute clinical babesiosis in an adult gelding pony living in an endemic area

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Summary

Equine piroplasmosis is a haemoprotozoal disease caused by *Theileria equi* and *Babesia caballi*. Clinical presentations vary in severity according to infectious load, host immunity and endemicity. In endemic regions, equine piroplasmosis is rarely acute or peracute in presentation. Here we report a case of a 10-year-old pony gelding presented for fever and acute inspiratory dyspnoea. Clinical signs of hypovolemic shock were observed. Blood smear examination revealed a mild anaemia and thrombocytopenia with the presence of intraerythrocytic inclusions resembling *Babesia caballi* merozoites. Acute lung injury, ischaemic myocarditis, acute renal failure, pancreatitis and myopathy were detected. We made a presumptive diagnosis of acute babesiosis, with secondary systemic inflammatory reaction and multiple organ dysfunction syndromes. Supportive therapy was initiated, but imidocarb was not administered due to potentially fatal adverse reactions in a horse in shock. Anaemia worsened, with the development of acute pulmonary oedema, respiratory distress and disseminated intravascular coagulation. The gelding was euthanised and post-mortem examination confirmed the formation of microthrombi within small vessels, leading to venous stasis and vasculitis.

KEYWORDS
horse, coagulopathy, haemolysis, piroplasmosis, SIRS

INTRODUCTION

Babesia caballi and *Theileria equi* are haemoprotozoan parasites responsible for equine piroplasmosis. Equine piroplasmosis is endemic in tropical, subtropical and temperate regions and the overall prevalence in Europe is estimated to be 2% and 25% for *B. caballi* and *T. equi*, respectively (Nadal et al., 2022). In the Camargue region in the south of France, a prevalence of 6.3% and 68.6% for *B. caballi* and *T. equi*, respectively, has recently been demonstrated (Rocafort-Ferrer et al., 2022). Depending on the host susceptibility and parasite load, the clinical presentation may vary from chronic disease and asymptomatic carrier states to acute and peracute disease (Wise et al., 2013). Acute infection is characterised by fever, anorexia, tachycardia, tachypnoea, pale and/or icteric mucous membranes and peripheral oedema (Maurer, 1962). In endemic areas, horses are regularly exposed to the parasite through tick bites. The clinical presentation is very rarely peracute and horses are generally able to clear *B. caballi* infection without treatment. This report describes a rare case of a fatal acute *B. caballi* infection in an adult pony born in an endemic area, without any other concomitant disease, but having received prior treatment with corticosteroids.

CASE HISTORY

A 10-year-old gelding pony was presented to our emergency service for the onset of fever followed by acute respiratory distress. Three days prior, the referring veterinarian examined the pony for

a high fever (39.8°C) and tachypnoea. The pony appeared moderately depressed with dysorexia but without any other abnormality on general examination. Systemic antimicrobials (penicillin and dihydrostreptomycin) combined with corticosteroids (dexamethasone, 0.05 mg/kg, by intramuscular injection, once a day) were administered for 3 days. A persistent fever, worsening tachypnoea and depression and the development of sternal oedema prompted referral to our veterinary hospital.

CLINICAL FINDINGS

On admission, the pony was weak and staggering. Initial physical examination showed a body condition score of 4/9 (estimated weight 400 kg), a normal rectal temperature (38°C), severely congested mucous membranes, subicteric sclera, an increased capillary refill time (3 s) and cold extremities. The pony was tachycardic (76 beats/min), with a holosystolic heart murmur (grade 3/6) audible on the left side. Cardiac rhythm was normal and no jugular pulse was detected. However, the facial arterial pulse was weak and a moderate sternal oedema was present. The pony was severely tachypnoeic (60 breaths/min), with flared nostrils and a superficial breathing pattern. There were no audible adventitious lung sounds and regional lymph nodes were not enlarged. Digestive borborygmi were reduced. Muscles were soft and nonpainful on palpation, and there was no evidence of clinical laminitis. No other abnormal findings were noticed, and no ticks were present.

Since the pony was in a state of hypovolemic, and potentially hypoxic, shock, intranasal oxygen was immediately administered through a nasal catheter (5 L/min). An intravenous catheter was inserted into the jugular vein and a hypertonic saline (2 mL/kg) bolus was administered, followed by an infusion of Ringer's lactate (Baxter Healthcare S/A, 10 mL/kg over 1 h). Micturition was observed; the urine was not macroscopically discoloured and the urine specific gravity was 1.018 (reference range (ref): 1.020–1.050).

Venous blood was collected from the jugular vein, with immediate haematoma formation at the puncture site. Arterial puncture from the facial artery was unsuccessful, and a haematoma developed despite adequate local compression. On subsequent arterial blood gas analysis under oxygen insufflation, PaO_2 was within the reference range (110 mmHg; ref: 80–100 mmHg), but hypocapnia (PCO_2 32 mmHg; ref: 35–45 mmHg) and metabolic acidosis (pH 7.27; ref: 7.32–7.45) with low bicarbonate (16.2 mmol/L; ref: 22–29 mmol/L) were present. Biochemistry showed severe hyperlactataemia (12 mmol/L; ref <2 mmol/L), hyperglobulinaemia (56 g/L; ref: 18–40 g/L), hypoalbuminaemia (22 g/L; ref: 28–36 g/L), increased serum amyloid A concentration (381 mg/L; ref: <5.0 mg/L), azotaemia (creatinine 622 µmol/L; ref: 80–140 µmol/L, BUN 45.5 mmol/L; ref: 3–6.5 mmol/L), hyponatraemia (128 mmol/L; ref 135–145 mmol/L), hypocalcaemia (ionised calcium concentration 1.2 mmol/L; ref: 1.35–1.45 mmol/L), increased muscle enzymes (CK 5252 IU/L; ref: 0–350 IU/L; AST 912 IU/L; ref: 190–440 IU/L), increased troponin Ic (0.18 ng/mL; ref: 0–0.06 ng/mL), increased lipase DGGR (60 IU/L; ref: 3–22 IU/L) and hyperlipaemia (triglycerides 9.32 mmol/L; ref: 0.14–0.54 mmol/L). GGT was within the reference range (27 IU/L; ref: 9.28–44.51 U/L). Complete blood count showed mild anaemia (PCV 29.4%; ref: 32%–44%; haemoglobin 12.6 g/dL; ref: 10.2–15.3 g/dL), mild neutrophilia without lymphopenia (10.2×10^9/L; ref: $2.5–7.5\times10^9$/L and 5.0×10^9/L; ref: $1.5–5.5\times10^9$/L, respectively), monocytosis (2.4×10^9/L; ref: $0.2–0.9\times10^9$/L) and mild thrombocytopaenia (83×10^9/L; ref: $90–290\times10^9$/L). Venous blood gas was not performed. Cytologic examination of the blood smear confirmed the thrombocytopenia and showed a moderate left shift and toxic changes of the neutrophils. Moreover, intraerythrocytic inclusions resembling *B. caballi* merozoites were detected in several red blood cells (Figure 1).

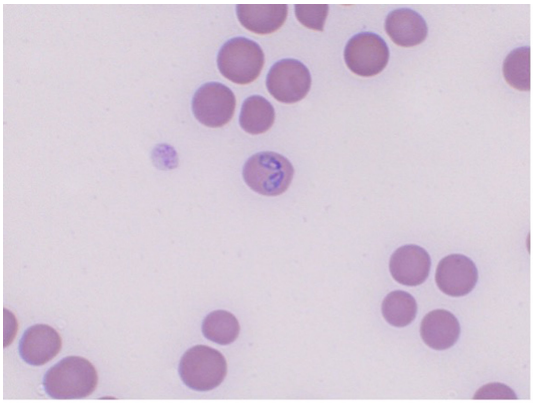

FIGURE 1 Blood smear showing intraerythrocytic *Babesia caballi* merozoites.

To further investigate the sternal oedema and hypoalbuminaemia, a thoracic and abdominal ultrasound was performed. No abdominal abnormalities were observed. A mild bilateral pleural anechoic effusion and several small, consolidated areas were visible in the thorax. A complete cardiac ultrasound examination was not performed, but there was no evidence of pericardial effusion. Thoracic radiographs showed a diffuse moderate to severe bronchointerstitial pattern, and an alveolar pattern caudal and ventral to the heart. Pleural fluid was aseptically sampled. The fluid was a modified transudate with an orange-tinged appearance, a cell count within normal limits (1.6×10^9/L cells, 69.0% nondegenerated neutrophils, 29.0% macrophages, 2.0% lymphocytes) and an increased total protein concentration (42 g/L; ref: <25 g/L). A few red blood cells were identified during cytologic examination of the pleural transudate, most of them containing merozoites (*B. caballi*). Bacterial culture of the fluid was negative.

DIAGNOSIS

Based on these findings, we made a presumptive diagnosis of acute babesiosis with secondary systemic inflammatory response syndrome, multiple organ dysfunction syndrome (acute lung injury, ischaemic myocardial injury, renal failure, pancreatic injury and myopathy) and disseminated intravascular coagulation (DIC).

TREATMENT

Supportive therapy was instituted prior to planned treatment with imidocarb diproprionate. Supportive therapy included intranasal oxygen, an infusion (3 L/h) of Ringer's lactate with 5% dextrose, calcium borogluconate (solution 10% B. Braun®, 0.5 mL/kg), furosemide (Dimazon® Intervet, 1 mg/kg) and a plasma transfusion (4 L of home-made plasma).

OUTCOME

Within 4 h, the pony's vital parameters deteriorated. He became severely depressed and recumbent, with a heart rate of 100 beats/min and a respiratory rate of 70 breaths/min, pale grey mucous membranes and a foamy haemorrhagic bilateral nasal discharge, suggestive of acute pulmonary oedema. The PCV dropped significantly to 12.1%. The platelet count was stable (89×10^9/L), but the total protein concentration also dropped (44 g/L), the metabolic acidosis worsened (venous blood pH 7.12), the hyperlactataemia was persistent and a hypoglycaemia developed (3.5 mmol/L; ref: [5.0–7.8 mmol/L]). Considering the poor prognosis and financial constraints, the owner elected euthanasia.

POST-MORTEM FINDINGS

On post-mortem examination, generalised petechial haemorrhages were present in the larynx, lungs, endocardium, intestines, kidneys, urinary bladder and adrenal glands. Four litres of red serous fluid was removed from the thoracic cavity. The spleen was diffusely enlarged with a moderately increased consistency (Figure 2). The renal cortex was diffusely softened. In the intestinal pelvic flexure, a focal transmural thickening with friable swollen mucosa was noted. Chronic ulcers and villous hepatitis and minor lesions of gastric gasterophilosis were also present.

Histopathological examination of post-mortem samples found a mild diffuse subacute centrilobular hepatic steatosis, likely secondary to hypoxia, and a mild parenchymal fibrosis. A moderate multifocal lymphoplasmacytic interstitial nephritis, and a multifocal membranoproliferative glomerulonephritis were also present. In the lungs, multifocal acute thrombosis of small vessels, along with oedema, congestion and moderate atelectasis were observed, as well as a mild neutrophilic and lymphocytic interstitial infiltrate. Diffuse acute congestion of the spleen and severe white and red pulp necrosis were also present (Figure 3).

DISCUSSION

Infection with *Babesia caballi* causes haemolytic anaemia of varying severity, which results from the physical rupture of erythrocytes during release of merozoites (intravascular) and from the removal of infected erythrocytes from the circulation by the spleen (extravascular; Donnellan & Marais, 2009). The pathogenesis of thrombocytopaenia and altered coagulation that may be observed, may include immune-mediated destruction, splenic sequestration and/or excess consumption as observed in DIC (Allen et al., 1975b; de Waal et al., 1987). *Babesia caballi*–infected erythrocytes can cause the formation of microthrombi within small

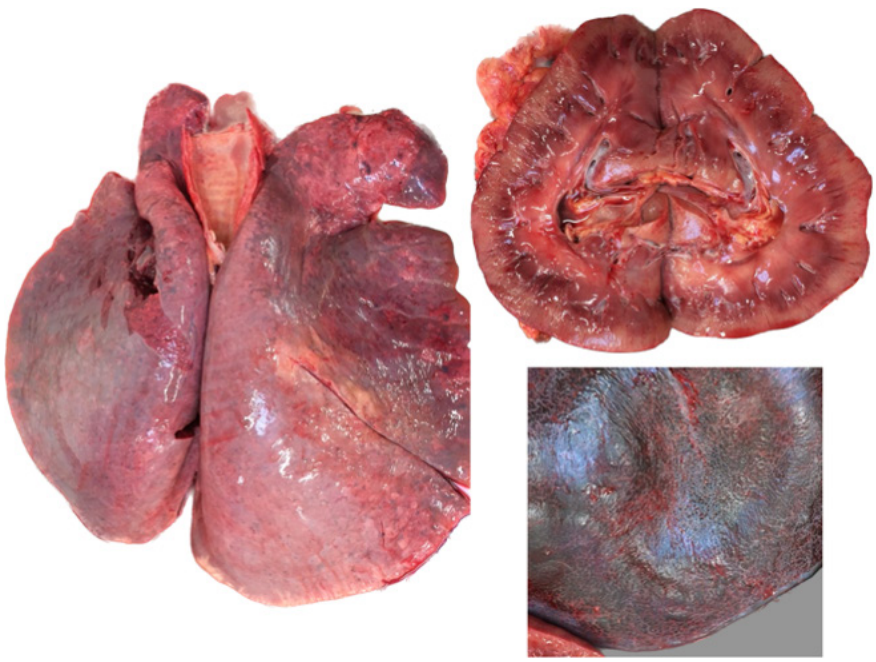

FIGURE 2 Post-mortem macroscopic examination found petechial haemorrhages on the lungs and kidneys, congestion (rounded edges) and increased consistency of the spleen.

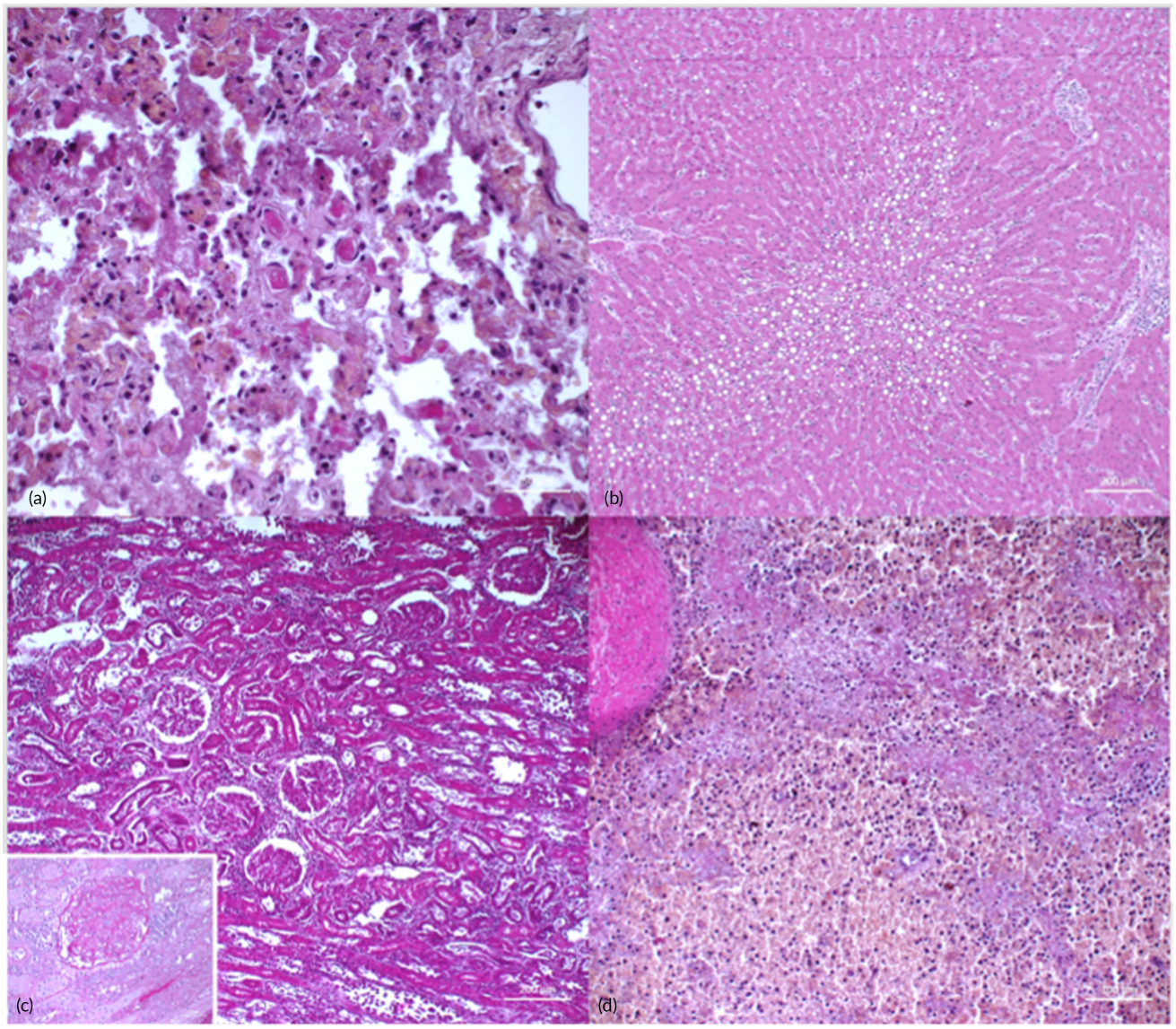

FIGURE 3 Histopathological examination of post-mortem samples (H&E staining – scale bar = 200 μm). (a) Lungs: multifocal intravascular thrombosis, diffuse congestion, low numbers of neutrophils and lymphocytes within interalveolar septa and neutrophils in alveoli. (b) Liver, mild diffuse subacute centrilobular steatosis, associated with multifocal periportal lymphoplasmacytic infiltration. (c) Kidneys, moderate lymphoplasmacytic interstitial nephritis and membranoproliferative glomerulonephritis (inset: glomeruli, Periodic Acidic Schiff stain). (d) Spleen, acute diffuse congestion and severe necrosis centred on white pulp.

vessels, leading to venous stasis and vasculitis (Allen et al., 1975b; de Waal et al., 1987). Equine piroplasmosis can also cause thrombocytopenia and alter coagulation in infected horses through unknown mechanisms (Allen et al., 1975a; Zobba et al., 2008). Thrombocytopenia is reported in 80% of *B. caballi* infections (Allen et al., 1975a; de Waal, 1992; de Waal et al., 1987) and clotting times may be prolonged or normal (Wise et al., 2014). In our case, splenic contraction, dehydration and previous corticosteroid administration might have been responsible for compensation of anaemia at admission. A few hours after initiating supportive therapy, the PCV fell sharply, probably as a result of the persistent haemolytic process induced by *B. caballi* and the dilution effect of fluid therapy.

Complications such as pulmonary oedema, central nervous symptoms, cardiac arrhythmias and gastrointestinal signs are less common in equine babesiosis (de Waal & van Heerden, 2004; Diana et al., 2007; Taylor et al., 1969; Wise et al., 2013; Zobba et al., 2008). In rare cases, acute infection can result in severe systemic inflammatory response syndrome with the development of DIC and multiple organ failure (Adam et al., 2016; Donnellan & Marais, 2009). In our case, clinical examination, laboratory and post-mortem findings were suggestive of renal failure, cardiac, pulmonary and pancreatic involvement, and the pleural effusion was suspected to be the result of acute lung injury. Bleeding diathesis was also observed in this case, suggesting a hypocoagulable state and DIC, although the thrombocytopenia was only mild and no petechiae were observed on the mucous membranes. Unfortunately, clotting times and thromboelastometry were not performed.

Acute *B. caballi* infection is characterised by pyrexia, anorexia, tachycardia, tachypnoea, pale and/or icteric mucous membranes and peripheral oedema (Maurer, 1962). Variation in severity of clinical disease depends on the region, strain of the parasite and overall health of the horse (Wise et al., 2014). Immunological naivety and increased density of infected ticks increase the risk for life-threatening clinical disease (Basset & Auger, 1931). With a competent immune system and appropriate treatment, most horses survive acute infection (Wise et al., 2014). The pony in our case had no previous history of illness and was reported to have been in good health prior to presentation. He lived in a field with other adult horses and there was no evidence or history of tick infestation. However, ticks do not remain permanently on the horse, and the absence of ticks does not rule out piroplasmosis, particularly in endemic regions. The pony's initial pyrexia was presumably caused by *B. caballi* infection. The cause of the hyperglobulinaemia present at admission is unknown but was suspected to be gastrointestinal parasitism.

The immune response secondary to infection with piroplasmids is multifactorial and complex. While the innate immune response is pivotal in cases of equine babesiosis, the precise role of the immune system is not known (Wise et al., 2013). Furthermore, information on the role of adaptive immunity, including cell-mediated immunity is limited (Banerjee et al., 1977; Zweygarth et al., 1983). The spleen plays an important role in the elimination of infected erythrocytes (Ambawat et al., 1999) because equids with intact spleens are typically able to control *T. equi* and *B. caballi* infections and survive. The survival of splenectomised horses experimentally infected with *B. caballi* depends on the infectious dose and other unknown factors, and fatalities have been reported (de Waal et al., 1988). In contrast, infection with *T. equi* causes high levels of parasitaemia in splenectomised horses and these animals invariably succumb to the disease (Knowles Jr et al., 1994; Kuttler et al., 1986).

In horses experimentally infected with *B. caballi*, production of nitric oxide, tumour-necrosis factor-α and other cytokines may play a protective role (Hanafusa et al., 1998). However, excess production of pro-inflammatory molecules can potentially contribute to pathogenicity. Horses infected with *B. caballi* also produce antibodies to rhoptry associated protein-1, a protein that seems to play a pivotal role in cattle in the induction of humoral immunity to *B. bovis* (Brown, 2001; Kappmeyer et al., 1999).

Although it is generally accepted that horses develop protective immunity after initial infection in endemic regions, studies confirming this hypothesis have not been performed. Persistent stimulation of the immune system by the parasite, reinfection or persistence of antibodies have been proposed to prevent clinical disease in previously infected horses. Clearance of *B. caballi* has been reported; however, the mechanisms underlying this clearance are currently unknown. It is also still unclear whether horses can be re-infected after a previous cleared infection (Wise et al., 2014).

The pony had no history of debilitating disease or signs suggestive of acquired immunosuppression. It is highly likely that previous administration of systemic corticosteroids caused immunosuppression with subsequent increased parasitaemia, inducing the peracute course of the disease.

The hypoalbuminemia was suspected to be secondary to protein loss in the pleural fluid and renal losses; however, we were unable to exclude protein loss in the gastrointestinal tract. Glomerulonephritis, responsible for renal protein loss, can occur secondary to acute kidney injury and tubulointerstitial disease (Olsen & van Galen, 2022). We noted glomerulonephritis on histological examination. Furthermore, an azotaemia was present, which may have been prerenal, secondary to decreased perfusion, but were most likely secondary to acute renal failure triggered by haemoglobinemia. However, we did not observe a macroscopic pigmenturia and no renal necrosis was identified during autopsy, possibly due to the presence of autolysis. Acute renal failure associated with *Babesia caballi* infection has been described previously (Adam et al., 2016). The elevated AST level was thought to be secondary to decreased blood flow to liver and muscles. Decreased hepatic blood flow may have caused the centrilobular necrosis noted in our case, as previously described (Wise et al., 2014). The increase in lipase DGGR may indicate pancreatic involvement. Secondary pancreatitis can occur secondary to hepatic disease (Edery et al., 2015; Yamout et al., 2012). However, in our case, no further investigation for liver disease was conducted, only GGT and AST levels were measured in the emergency panel. Mild increases in lipase have also been described secondary to damage of the intestinal mucosa or renal tubules (Dacre et al., 2003).

Inflammatory myopathy has been previously described in horses with chronic piroplasmosis (Pasolini et al., 2018). It is currently unknown whether myopathies may also be present in cases of acute piroplasmosis. In our case, the increased levels of muscle enzymes may have been induced by a combination of immune-mediated mechanisms and decreased muscular blood flow. It could also have been caused by prolonged recumbency, although this was not reported by the owners. Myocardial disease is a rare complication of babesiosis (Diana et al., 2007), associated with hypoxic injury secondary to hypovolemic shock. In our case, myocardial lesions were not identified on necropsy.

Our diagnosis of *B. caballi* was based on clinical signs, laboratory results and blood smear analysis. The disadvantage of blood smear evaluation is that even in the acute phase of infection, the parasitaemia levels of *B. caballi* are often very low (less than 1% of red blood cells; Friedhoff & Soule, 1996; Wise et al., 2013). PCR is the most sensitive method for diagnosis. In our case of acute piroplasmosis, several merozoites were easily identified on the blood smear as well as in the pleural fluid. High levels of parasitaemia, over 50% of red blood cells, have been previously reported in cases of abortion and neonatal piroplasmosis (Chhabra et al., 2012; De Waal & Van Heerden, 2004). No PCR was performed in this case, and a possible co-infection with *Anaplasma phagocytophilum* and/or *T. equi* was not explored. Serological tests (complement fixation test, or indirect immunofluorescence test, or complement enzyme-linked immunosorbent assay) were not carried out because they are a less sensitive diagnostic method, especially in the acute phase of the disease, and because of the delay in obtaining results compared with PCR.

Furthermore, in endemic areas, a positive result does not necessarily indicate acute infection but may instead indicate past infection (Wise et al., 2013). Nevertheless, it would have been useful to document previous exposure to *B. caballi* and/or *T. equi* in this pony by serology.

Imidocarb diproprionate has been used to treat piroplasmosis in horses and different treatment regimens have been described. Chemotherapeutic clearance of *B. caballi* can be achieved with a dosage of 4.4 mg/kg administered by intramuscular injection, given 4 times at 72-h intervals (Frerichs & Holbrook, 1974; Kumar et al., 2003; Kuttler et al., 1987; Schwint et al., 2009, Wise et al., 2014). The drug acts as an anticholinesterase and adverse reactions such as sweating, colic and diarrhoea frequently occur (Adams, 1981; Meyer et al., 2005). Clearance of the drug is achieved by the renal and hepatic routes, and necrosis in these two organs has been described in cases of intoxication (Adams, 1981; Meyer et al., 2005). There are also sporadic reports of fatal reactions in critically ill horses (Desjardins & Couroucé, 2022; Sumbria et al., 2014). The pony in this case did not receive imidocarb as there is still controversy as to whether this drug should be administered to animals in a state of shock. Adverse reactions may be prevented by intravenous administration of glycopyrrolate (0.05 mg/kg) or reversed by a single intravenous administration of atropine (0.1 mg/kg). However, adverse reactions have also been reported for these drugs (Wise et al., 2014).

In our case, supportive care was administered, including intravenous hypertonic saline followed by isotonic crystalloids to provide rapid fluid resuscitation, necessary to improve tissue perfusion. Administration of crystalloid fluids must be done cautiously, especially in cases with decreased oncotic pressure. In this pony, hypoalbuminemia was present, suggesting a decreased oncotic pressure. Therefore, a concurrent plasma transfusion was performed to provide colloid support, as well as coagulation factors and cofactors. Plasma has been previously used to treat DIC in horses (Welch et al., 1992). Transfusion-related acute lung injury is described in small animals and humans and the use of fresh frozen plasma in critically ill patients remains controversial (Beer & Silverstein, 2015). We could have administered synthetic colloids or a whole blood transfusion. Synthetic colloids increase oncotic pressure and provide more significant volume expansion; however, adverse effects like acute kidney injury and coagulopathies have been reported in human medicine (Epstein et al., 2014; Glover et al., 2014; Hayes et al., 2016). Little is currently known about these adverse effects in horses (Hepworth-Warren, 2021). Whole blood transfusion is indicated to restore the oxygen-carrying capacity of the blood in cases of anaemia. Transfusion of fresh frozen plasma was chosen in this case because the anaemia was initially mild and the incidence of transfusion reactions to commercial plasma are reported to be lower than that for whole blood transfusion (Mudge, 2015). The presence of severe anaemia on admission and/or significant thrombocytopenia would have warranted the administration of whole blood instead of plasma. In this case, one might discuss about the fact whether a whole blood transfusion would have been more indicated instead of the plasma transfusion, especially when the PCV dropped. Venous blood lactate was high, and a metabolic acidosis was present, both may indicate poor perfusion and delivery of oxygen, which is a main indication for whole blood transfusion. Venous blood gas analysis with determination of the mixed venous oxygen tension and oxygen extraction ration might have been helpful to determine the need and timing for whole blood transfusion (Hurcombe et al., 2007). To the authors' knowledge, there are no data on the benefits and need for blood transfusion in equine piroplasmosis.

In conclusion, the case shows that acute and fatal clinical babesiosis is possible in horses in endemic areas, even when no ticks are visible on the animal. Early recognition of the disease and the resulting haemolytic process is crucial to prevent subsequent organ damage. It is not known whether corticosteroids can increase the severity of clinical disease by inducing a transient immunosuppression. Furthermore, the administration of imidocarb to a horse in shock remains highly controversial, nevertheless, death may occur during the stabilisation of shock with supportive therapy, due to worsening of the haemolytic crisis. Therefore, further investigation into therapeutic strategies during clinical equine piroplasmosis is needed.

AUTHOR CONTRIBUTIONS

Lisa-Marie Hermans: Writing – original draft. **Antonin Tortereau:** Writing – original draft. **Barbara Riccio:** Writing – original draft. **Isabelle Desjardins:** Writing – original draft.

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CONFLICT OF INTEREST STATEMENT

No conflicts of interest to declare.

ETHICS STATEMENT

Not applicable.

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