CASE REPORT

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Management of severe equine granulocytic anaplasmosis with blood transfusion and antimicrobial therapy in a mare

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Abstract

Equine granulocytic anaplasmosis is a tick-borne infection characterized by fever, weakness, thrombocytopenia, and anaemia. Severe cases may require blood transfusion—a procedure infrequently practised in equine medicine in Nigeria. A 12-year-old Sudanese Country-Bred mare weighing 400kg, presented to the veterinary team at the Ibadan Polo Club, Ibadan, Oyo State, Nigeria, with a two-week history of hyporexia and lethargy after participating in a polo tournament in Kano State, Nigeria. Clinical examination revealed intermittent fever (up to 40.3°C), pale mucous membranes, prolonged capillary refill time (> 2 seconds), slightly elevated pulse rates (54 beats/min), slightly high respiratory rates (25 breaths/min), and infestation with hard-bodied ticks. Laboratory tests showed severe anaemia (PCV 9.5%), leukopenia, thrombocytopenia, and the presence of *Anaplasma phagocytophilum* morulae within the neutrophils. Initial supportive treatment included vitamin B complex, phenylbutazone, ivermectin, and whole blood transfusion (2.5 litres). Upon confirmation of *Anaplasma phagocytophilum* infection, the horse received intravenous 5% oxytetracycline for five days, followed by two weeks of oral doxycycline. Fluid therapy was administered to correct fluid and electrolyte deficits. Complete clinical recovery was observed three weeks post-treatment, with haematologic parameters returning to near-normal levels. This case highlights the importance of early diagnosis, antimicrobial therapy, and timely blood transfusion in managing severe equine granulocytic anaplasmosis. Routine tick control and health checks before and after polo tournaments are essential preventive measures.

Keywords: Anaplasmosis, Anaemia, Blood transfusion, Intermittent fever, Ticks

Introduction

Equine granulocytic anaplasmosis, formerly equine granulocytic ehrlichiosis, is a tick-borne disease caused by a rickettsia group of gram-negative bacteria known as *Anaplasma phagocytophilum* (Bogdan *et al.,* 2024). The organism is an obligate intracellular parasite and, besides infecting horses, *A. phagocytophilum* has been known to infect other domestic animals and humans. Infection in horses is characterised by inappetence, fever, pedal oedema, depression, lethargy, and thrombocytopenia (Bogdan *et al.,* 2024). Co-infections with other tick-borne haemoparasites may occur, especially with *Borrelia burgdorferi*.

The disease was first reported among horses in the United States of America but has currently been reported in many countries where ticks are endemic. Light microscopic screening results suggest a 30% prevalence of anaplasmosis due to *Anaplasma phagocytophilum* among horses in South-south Nigeria (Eze *et al.,* 2020), while one study in South-west Nigeria reported a 14.3% prevalence of the infection among horses in the Ibadan polo stable (Alaba *et al.,* 2022). Reports of equine granulocytic anaplasmosis in Nigeria remain limited and fragmented, especially among polo horses (Eze *et al.,* 2020). Climate change has been implicated in the increasing risk of infection of horses with *Anaplasma phagocytophilum*, because climate change has significantly influenced the behaviour and distribution pattern of tick populations, as well as prolonging the duration of ticks' active seasons (Bogdan *et al.,* 2024).

Horses become infected with *A. phagocytophilum* after being bitten by ticks carrying the bacteria. The organism travels to the bloodstream after entering through the skin, where it specifically, infects granulocytes especially neutrophils. Endothelial cells in the blood vessels are also infected which contributes to the pathogenesis. The incubation period of *A. phagocytophilum* is typically 6–12 days, after which clinical signs become apparent. However, infected horses may not show obvious illness after infection and remain asymptomatic. Younger horses, especially those under four years old, usually show less severe clinical signs, whereas aged horses are more likely to experience more severe clinical manifestations of the condition characterised by fever and severe anaemia (Bogdan *et al.,* 2024).

The diagnosis of equine granulocytic anaplasmosis is based on clinical findings and blood tests (Bogdan et *et al.,* 2024). Anaplasmosis is suspected in horses if the presenting clinical signs include high fever, lethargy, hyporexia, yellowing of mucous membranes and gums, sunken eyes, swelling in the limbs, uncoordinated gaits, and the presence of ticks on the horse's body. Haematological findings typically reveal anaemia and thrombocytopenia, whereas serum chemistry often shows hyperproteinaemia and elevated liver enzyme titres (Hinson *et al.,* 2024). Although polymerase chain reaction and other molecular tests are more accurate in reaching a definitive diagnosis, microscopic demonstration of inclusion bodies or morulae within granulocytes is deemed adequate in resource-limited settings (Bogdan *et al.,* 2024).

The primary approach to the management of equine granulocytic anaplasmosis is basically the use of antibiotics and supportive care which may include fluid therapy, electrolytes replacement, blood transfusion, and antipyretics (Bogdan *et al.,* 2024). Most horses recovered fully after 2–3 weeks of treatment. However, some horses have been reported to recover fully without antibiotic treatment (Lewis *et al.,* 2009). Most untreated horses suffer from weight loss, prolonged swelling of limbs and increased susceptibility to secondary bacterial infection, which may lead to death (Bogdan *et al.,* 2024).

Whole blood transfusion is indicated in emergency cases of acute haemorrhage or severe anaemia in horses to save their lives. More than 30 factors and 8 blood groups and over 400,000 possible erythrocyte phenotypes have been identified in the horse (Magdesian, 2017). It is important to conduct cross matching and blood typing to access blood compatibility to avoid blood transfusion reactions (Jamieson *et al.,* 2022). Under a field situation and locations where pre-transfusion screening is not feasible, the equine practitioner can safely carry out blood transfusion if the horse has no previous exposure to biological products or blood transfusion with very minor risks (Jamieson *et al.,* 2022).

This report describes the management of severe equine granulocytic anaplasmosis in a polo horse in Nigeria with antimicrobial therapy and supportive blood transfusion.

Case Management

Case history

A 12-year-old Sudanese Country-Bred mare weighing 400kg, housed at the Ibadan Polo Club, Eleyele, Ibadan, Oyo State, South-Western Nigeria, was presented to the veterinary team at the same polo club with a history of continuous inappetence and

lethargy commencing about two weeks following active participation in a polo tournament held in Kano State, Northern Nigeria.

Clinical manifestations and samples collection

Hard-bodied ticks were found on the horse; pulse and respiratory rates were slightly elevated at 54 beats/min (normal range 30–44 beats/min) and 25 breaths/min (normal range 8–15 breaths/min), respectively. Intermittent fever was recorded with rectal temperatures ranging between 37.9℃ and 40.3℃ with the median value of 39.5°C (normal range 37.5℃ –38.5℃). The mucous membranes were pale; capillary refill time was prolonged (> 2 seconds).

A total of 10 mL of blood samples were collected aseptically from the jugular vein of the horse using sterile 10 mL syringe and dispensed into sample bottle containing ethylenediaminetetraacetic acid (EDTA) for complete blood count (CBC) and haemoparasite screening (5 mL of blood) and plain bottles for serum biochemistry (5 mL of blood).

Laboratory investigations

For the CBC, an automated haematology analyser (IDEXX ProCyte Dx™, IDEXX Laboratories, USA; Lot No.: 2023ABC), clinically validated for equine samples, was employed to measure parameters such as packed cell volume (PCV), haemoglobin concentration, red blood cell (RBC) count, white blood cell (WBC) count, and platelet count. Differential leukocyte counts were performed manually by examining Wright-Giemsa-stained blood smears under a light microscope to identify and quantify neutrophils, lymphocytes, monocytes, eosinophils, and basophils. Haemoparasite screening was conducted by preparing thin blood smears, which were stained with Giemsa and examined microscopically for the presence of morulae or inclusion bodies within granulocytes, specifically neutrophils, to confirm *Anaplasma phagocytophilum* infection. For serum biochemical analysis, blood samples were centrifuged to separate serum, and an automated biochemistry analyser was used to evaluate liver enzymes (ALT, AST, ALP, and GGT), renal markers (urea, creatinine), electrolytes (sodium, potassium, chloride, and bicarbonate), total protein, albumin, globulin, and glucose levels as described by Akinniyi *et al.* (2025). All procedures adhered to standard veterinary laboratory protocols, and results were interpreted in comparison to established reference ranges for horses to assess deviations indicative of disease.

Empirical management

Initial treatment included multivitamins injection (V-Multinor®, Jubaili Agrotec Ltd, Nigeria), administered at 30 mL intramuscularly for 3 days to stimulate appetite. Intravenous phenylbutazone (Phenylarthrite®, Vetoquinol, France) was given at 4.4 mg/kg for 3 days to reduce fever. Subcutaneous ivermectin (Kepromec®, Kepro B.V., Holland) was administered at 0.2 mg/kg as an endectocide against ticks.

Confirmatory diagnosis

Microscopic examination of a thin blood smear confirmed the presence of *A. phagocytophilum* morulae (Plate I); hence, a confirmatory diagnosis of equine granulocytic anaplasmosis. The patient had severe anaemia (PCV: 9.5%, Hb: 3.1 g/dL, RBC: 1.72 $\times 10^6/\mu L$), mild leukopenia, neutropenia, lymphopaenia, monocytosis, and thrombocytopenia, consistent with *Anaplasma phagocytophilum* infection (Table 1). Also, mild elevated ALP (231 U/L) and hyperproteinemia (92 g/L) were noted, likely due to inflammation. Other parameters (liver enzymes, renal function, and electrolytes) were normal (Table 2). The severe anaemia necessitated immediate whole blood transfusion to prevent the horse from going into hypovolemic shock.

Blood transfusion

A five-year old Sudanese Country-Bred stallion weighing 450kg was presented as a blood donor. Pre-transfusion haematology of the donor's blood

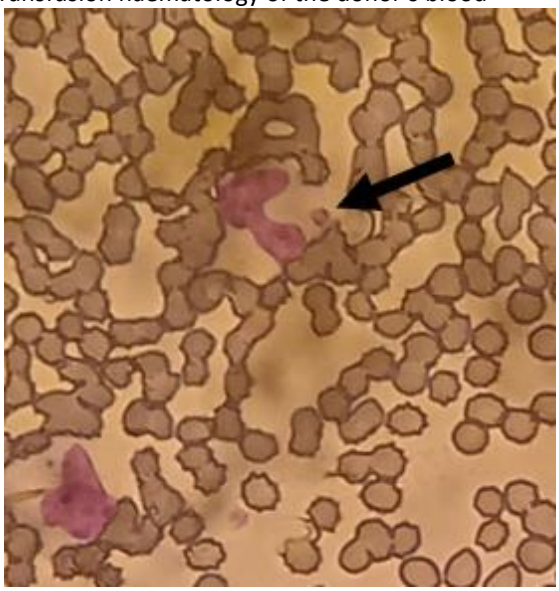

Plate I: *Anaplasma phagocytophilum* morulae (black arrow) within one of the neutrophils (1000×)

Table 1: Haematology results of the mare on presentation

Parameters	Result	Normal Range (Horse)	Interpretation
PCV (%)	9.5	32.00–53.00	Critical anaemia
Haemoglobin (g/dL)	3.1	11.0–19.0	Severe anaemia
Erythrocytes (RBC $\times10^6/\mu L$)	1.72	6.80–12.90	Severe anaemia
Leucocytes (WBC $\times10^3/\mu L$)	5.37	5.40–14.30	Mild leukopenia
Segmented Neutrophils ($\times10^3/\mu L$)	2.22 (41.3%)	2.30–9.50	Neutropenia
Lymphocytes ($\times10^3/\mu L$)	0.91 (17.0%)	1.50–7.70	Lymphopaenia
Monocytes ($\times10^3/\mu L$)	2.19 (40.8%)	0.00–1.50	Monocytosis
Basophils ($\times10^3/\mu L$)	0.04 (0.8%)	0.00–0.30	Normal
Eosinophils ($\times10^3/\mu L$)	0.01 (0.1%)	0.00–1.00	Normal
Platelets ($\times10^3/\mu L$)	80	100–400	Thromocytopaenia
MCV (fL)	55.2	37–59	
MCH (pg)	18	12.3–19.7	
MCHC (g/dL)	32.6	31.0–39.0	

Note: PCV = packed cell volume, MCV = mean corpuscular volume, MCH = mean corpuscular haemoglobin, MCHC = Mean corpuscular haemoglobin concentration. Reference intervals for haematological parameters were obtained from Reed *et al.* (2017)

Table 2: Serum chemistry results of the mare on presentation

Parameters	Result	Normal Range (Horse)	Interpretation
ALT (U/L)	17	2.7–21	Normal
AST (U/L)	160	160–412	Normal
ALP (U/L)	231	70–227	Mild elevation
GGT (U/L)	27	6–32	Normal
Total Bilirubin (µmol/L)	2.4	0–3.2	Normal
Urea (mEq/L)	18	11–27	Normal
Creatinine (µmol/L)	72.8	35–194	Normal
Total Protein (g/L)	92	56–76	Hyperproteinemia
Albumin (g/L)	42	26–41	Mild hyperalbuminemia
Globulin (g/L)	50	26–40	Hyperglobulinemia
Albumin/Globulin Ratio	0.8	–	Normal
Chloride (mmol/L)	100	98–109	Normal
Bicarbonate (mmol/L)	28	24–30	Normal
Potassium (mmol/L)	4.7	2.9–4.6	Mild hyperkalemia
Sodium (mmol/L)	134	128–142	Normal
Glucose (mg/dL)	67	62–134	Normal

Note: ALT = Alanine aminotransferase, AST = Aspartate transferase, ALP = Alkaline phosphatase, GGT = Gamma-glutamyl transferase. Reference intervals for serum biochemical parameters were obtained from Reed *et al*. (2017)

revealed a PCV of 49% and all other blood parameters were normal (Table 3). There was also absence of haemoparasites. Blood typing and cross-matching were not conducted due to field constraints however this was the first blood transfusion in the recipient horse based on history. Blood collection and transfusion was done aseptically via jugular venepuncture. Commercial blood bags containing citrate phosphate dextrose as anticoagulant were used for the whole blood collection. A total of 2.5L whole blood was collected from the donor horse in five blood bags, which was less than the estimated 7.2 litres that can be collected from a horse of 450kg with PCV of 49% based on established recommendations by Jamieson *et al.* (2022). Normal saline of equal volume to the blood collected was immediately given to the donor horse to prevent hypovolemia in the donor. Both blood collection and transfusion were gravity assisted (Plate II). Follow up haematology and serum chemistry evaluation were done for both blood donor and recipients 24 hours post transfusion. 24 hours post-donation, the donor exhibited mild decreases in PCV (44%), haemoglobin (15.0 g/dL), and RBC count (10.1 $\times10^6/\mu L$), all of which remained

within normal limits (Table 3). 24 hours post transfusion, the patient showed marked improvement. The PCV increased from 9.5% to 14%, haemoglobin from 3.1 g/dL to 4.7 g/dL, and RBC count from 1.72 $\times 10^6$/µL to 3.5 $\times 10^6$/µL, though values remained below normal ranges (Table 4).

Definitive management

The following day after a successful transfusion, drugs and intravenous fluids were administered to the patient. Intravenous 5% oxytetracycline (Tetracycline®, Vet India Pharmaceuticals Limited, Hyderabad, Andhra Pradesh, India) was given at 5 mg/kg for 5 days. Additional fluid therapy was administered to the mare to correct the fluid and electrolyte deficits: Lactated Ringer's (4 litres for 3 days) and a 50% dextrose solution (400 mL for 3 days). Initial antibiotic therapy was followed by oral administration of doxycycline capsules (Doxycap®, Hovid Pharma, Malaysia) at 10 mg/kg twice daily for 2 weeks to ensure total clearance of the parasite from the blood.

Table 3: Haematology of donor before and 24 hours after donation

Parameters	Pre-donation	24 hrs Post-donation	Normal range	Interpretation
PCV (%)	49	44	32–53	Mild decrease (still normal)
Haemoglobin (g/dL)	16.5	15.0	11–19	Mild decrease
RBC ($\times 10^6$/µL)	11.2	10.1	6.8–12.9	Mild decrease
WBC ($\times 10^3$/µL)	8.5	8.2	5.4–14.3	Stable
Segmented Neutrophils ($\times 10^3$/µL)	5.1 (60%)	4.9 (60%)	2.3–9.5	Normal
Lymphocytes ($\times 10^3$/µL)	2.6 (30%)	2.5 (30%)	1.5–7.7	Normal
Monocytes ($\times 10^3$/µL)	0.7 (8%)	0.7 (8%)	0.0–1.5	Normal
Basophils ($\times 10^3$/µL)	0.0 (0%)	0.0 (0%)	0.0–0.3	Normal
Eosinophils ($\times 10^3$/µL)	0.2 (2%)	0.2 (2%)	0.0–1.0	Normal
Platelets ($\times 10^3$/µL)	220	200	100–400	Mild decrease
Total Protein (g/L)	70	63	56–76	Mild dilution
Albumin (g/L)	35	32	26–41	Mild dilution
Globulin (g/L)	35	31	26–40	Mild dilution
MCV (fL)	43.8	43.6	37–59	
MCH (pg)	14.7	14.9	12.3–19.7	
MCHC (g/dL)	33.7	34.1	31–39	

Note: PCV = packed cell volume, MCV = mean corpuscular volume, MCH = mean corpuscular haemoglobin, MCHC = Mean corpuscular haemoglobin concentration. Reference intervals (Reed *et al*., 2017).

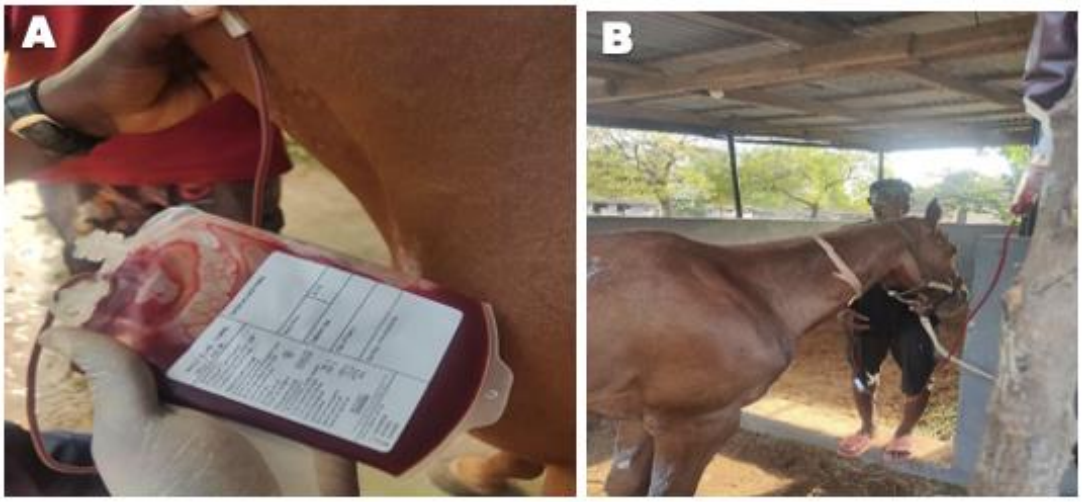

Plate II: Blood transfusion procedure: (A) Blood collection from the donor horse; (B) Administration of the blood transfusion to the sick horse

Table 4: Haematology of patient (recipient) before and 24 hours after receiving blood

Parameters	Pre-transfusion	24 hrs Post-transfusion	Normal range	Interpretation
PCV (%)	9.5	14	32–53	Improved but still anaemic
Haemoglobin (g/dL)	3.1	4.7	11–19	Improved
RBC (×10⁶/µL)	1.72	3.5	6.8–12.9	Improved
WBC (×10³/µL)	5.37	6.0	5.4–14.3	Slight improvement
Segmented Neutrophils(×10³/µL)	2.22 (41.3%)	2.5 (42%)	2.3–9.5	Slight improvement
Lymphocytes (×10³/µL)	0.91 (17%)	1.1 (18%)	1.5–7.7	Normalized
Monocytes (×10³/µL)	2.19 (40.8%)	2.0 (33%)	0.0–1.5	Resolved monocytosis
Basophils (×10³/µL)	0.04 (0.8%)	0.05 (0.8%)	0.0–0.3	Normal
Eosinophils (×10³/µL)	0.01 (0.1%)	0.1 (2%)	0.0–1.0	Normal
Platelets (×10³/µL)	80	94	100–400	Mild thrombocytopenia persists
MCV (fL)	55.2	40	37–59	
MCH (pg)	18	13.4	12.3–19.7	
MCHC (g/dL)	32.6	33.6	31–39	

Note: PCV = packed cell volume, MCV = mean corpuscular volume, MCH = mean corpuscular haemoglobin, MCHC = Mean corpuscular haemoglobin concentration. Reference intervals for haematological parameters were obtained from Reed *et al.* (2017)

Table 5: Haematology of patient after completion of 3 weeks treatment

Parameters	3 weeks post-treatment	Normal ange	Interpretation
PCV (%)	26	32–53	Near-full recovery
Haemoglobin (g/dL)	8.7	11–19	Normalized
RBC (×10⁶/µL)	5.5	6.8–12.9	Normalized
WBC (×10³/µL)	9.8	5.4–14.3	Normalized
Segmented Neutrophils (×10³/µL)	5.4 (55%)	2.3–9.5	Resolved neutropenia
Lymphocytes (×10³/µL)	3.4 (35%)	1.5–7.7	Normalized
Monocytes (×10³/µL)	0.8 (8%)	0.0–1.5	Normalized
Basophils (×10³/µL)	0.0 (0%)	0.0–0.3	Normal
Eosinophils (×10³/µL)	0.2 (2%)	0.0–1.0	Normal
Platelets (×10³/µL)	280	100–400	Resolved thrombocytopenia
MCV (fL)	42.3	37–59	
MCH (pg)	15.8	12.3–19.7	
MCHC (g/dL)	33.5	31–39	

Note: *Anaplasma phagocytophilum* morulae no longer found

Note: PCV = packed cell volume, MCV = mean corpuscular volume, MCH = mean corpuscular haemoglobin, MCHC = Mean corpuscular haemoglobin concentration. Reference intervals for haematological parameters were obtained from Reed *et al.* (2017)

Outcome

Complete recovery from clinical signs was observed three weeks after treatment. A complete blood count and serum biochemical analysis were also done, and it revealed that the patient had achieved near-full recovery. PCV (26%), haemoglobin (8.7 g/dL), and RBC count (5.5 ×10⁶/µL) approached normal ranges, indicating resolution of anaemia (Table 5).

The serum chemistry results were entirely within normal ranges. Liver enzymes (ALT: 13 U/L, AST: 171 U/L, ALP: 200 U/L), renal markers (urea: 14 mEq/L, creatinine: 80 µmol/L), and electrolytes (sodium: 137 mmol/L, potassium: 4.1 mmol/L) showed no abnormalities. Total protein (70 g/L), albumin (34 g/L), and globulin (36 g/L) levels normalized, indicating resolution of earlier imbalances (Table 6).

Discussion

Equine granulocytic anaplasmosis is an emerging concern among polo horses in Nigeria, particularly given the endemicity of ticks in the south-western region, where high humidity supports year-round tick

Table 6: Serum chemistry of patient after completion of 3 weeks treatment

Parameters	3 weeks post-treatment	Normal range (Horse)	Interpretation
ALT (U/L)	13	2.7–21	Normal
AST (U/L)	171	160–412	Normal
ALP (U/L)	200	70–227	Normal
GGT (U/L)	3	6–32	Normal
Total Bilirubin (µmol/L)	2.7	0–3.2	Normal
Urea (mEq/L)	14	11–27	Normal
Creatinine (µmol/L)	80	35–194	Normal
Total Protein (g/L)	70	56–76	Normal
Albumin (g/L)	34	26–41	Normal
Globulin (g/L)	36	26–40	Normal
Albumin/Globulin Ratio	0.9	–	Normal
Chloride (mmol/L)	99	98–109	Normal
Bicarbonate (mmol/L)	29	24–30	Normal
Potassium (mmol/L)	4.1	2.9–4.6	Normal
Sodium (mmol/L)	137	128–142	Normal
Glucose (mg/dL)	130	62–134	Normal

Note: ALT = Alanine aminotransferase, AST = Aspartate transferase, ALP = Alkaline phosphatase, GGT = Gamma-glutamyl transferase. Reference intervals for serum biochemical parameters were obtained from Reed *et al.* (2017)

proliferation (Edeh *et al.,* 2024). The seasonal peak in tick populations correlates with increased incidences of haemoparasitic infections. In this case, the horse presented in the month of August, a known high-risk period for tick activity.

Although equine granulocytic anaplasmosis has been reported to be self-limiting, especially in young horses (Bogdan *et al.,* 2024), the horse's participation in a long-distance journey of over 800 km for a polo tournament likely contributed to immunosuppression, predisposing it to severe *Anaplasma phagocytophilum* infection. Stress associated with transportation is a known risk factor for the exacerbation of latent infections or increased susceptibility to new infections.

Severe anaemia, leukopenia, and thrombocytopenia observed in this horse are classical haematological abnormalities associated with equine granulocytic anaplasmosis (Bogdan *et al.,* 2024). However, the degree of anaemia observed in this case (PCV 9.5%) was profound and uncommonly reported in previous studies (Lewis *et al.,* 2009). While mild anaemia is a recognized feature of anaplasmosis, such severe hematologic compromise suggests a chronic disease course, possibly compounded by delayed intervention following transportation-induced immunosuppression (Hinson *et al.,* 2024).

Timely whole blood transfusion was lifesaving in this case, as supported by Jamieson *et al.* (2022), who advocate for prompt transfusion in equids with critical anaemia. The subsequent antibiotic therapy with intravenous oxytetracycline ensured rapid bacterial clearance, with oral doxycycline administered to maintain therapeutic levels and prevent recrudescence. This treatment strategy aligns with current recommendations for managing equine granulocytic anaplasmosis (Bogdan *et al.,* 2024).

Follow-up laboratory testing after three weeks of treatment confirmed hematologic recovery and clearance of the pathogen. Notably, the PCV improved significantly but remained slightly below the normal range, indicating ongoing erythropoietic recovery. Complete parasite clearance was evident as the absence of neutrophilic morulae.

A key limitation of this case is the reliance solely on microscopy for diagnosis, without confirmatory molecular techniques such as PCR or sequencing. While the presence of neutrophilic morulae strongly suggests *A. phagocytophilum* infection, molecular analysis would have provided definitive species identification

This case emphasises the necessity for routine health checks and stringent tick control, particularly during seasons of high tick activity. It also underscores the need for heightened clinical suspicion for anaplasmosis in horses presenting with fever, lethargy, and haematological abnormalities, particularly in endemic regions. Regular tick control in stables has been recommended to horse owners to

prevent and reduce the occurrence of haemoparasitic infections.

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