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CLINICO-PATHOLOGICAL AND HAEMATOLOGICAL FINDINGS IN EQUINE PIROPLASMOSIS IN IBADAN

R. A. M Adedokun[1], B. A. Alaba[1]*, O. O. Alaka[2] and D. Omoniwa[3]

[1]Department of Veterinary Medicine, University of Ibadan, Ibadan, Nigeria.
[2]Department of Veterinary Pathology, University of Ibadan.
[3]Department of Veterinary Medicine, Surgery & Radiology University of Jos, Jos, Nigeria.

***Corresponding Author: B. A. Alaba**

Department of Veterinary Medicine, University of Ibadan, Ibadan, Nigeria.

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ABSTRACT

Equine piroplasmosis (EP), a disease naturally transmitted by ticks is endemic and widely spread in Nigeria among horses and donkeys. This disease has remained a challenge, particularly as the movement of horses across Nigeria has increased. Hence, there is need for concerted and continuous in-depth research to check the menace of equine piroplasmosis in Nigeria. A total of Fifty-eight (58) horses from three different locations in Ibadan, Southwest Nigeria, were clinically examined in their state of rest. Haemoparasites identification and haematological parameters were evaluated according to standard procedures. Five (8.6%) of the 58 horses examined were positive for the disease. Observed clinical signs in *Babesia*-positive horses included emaciation, severe dehydration, fever, hyperpnoea, tachycardia, dullness, ecchymosis of the third eyelids, icterus, haemoglobinuria, tick infestation, oedema of the head, ventral abdominal wall, scrotal sac and fetlock joints, reluctance to move, weak hind quarters and unsteady gait were observed in clinically positive horses. Haematological examination revealed intraerythrocytic and extraerythrocytic *Theleira equi* and *Babesia caballi.* The haematology result showed that infected horses presented a significant decrease ($p<0.05$) in PCV, Hb, RBC, WBC, lymphocytes counts and significant elevation ($P<0.05$) in neutrophils and monocytes. Of the 5 (8.6%) *Babesia* positive horses, fatalities were recorded in 3(5.2%). Post mortem findings included generalized inflammation, necrosis, congestion and oedema with diffused subcapsular ecchymotic and petechial haemorrhages of the visceral organs such as lungs, heart, liver, spleen, kidney and brain. The histopathological findings of the affected organs revealed alveolar and interstitial pulmonary oedema and thrombosis of the pulmonary blood vessels. There was severe multifocal hepatic necrosis, portal fibrosis, presence of macrophages and neutrophils in the necrotic areas and presence of haemosiderin in kupffer cells. Haemosiderin laden macrophages were present in the spleen while the renal tubules were markedly dilated and contained proteinaceous casts. There were also stunting of intestinal villi, necrosis of epithelial surface, as well as oedema and haemorrhages in the lamina propia. Brain sections showed marked congestion and thickening of the meningeal blood vessel walls with moderate lymphocytic infiltrations of the meninges and margination of leucocytes in the meningeal vessels. The role of major managemental negligence such as heavy tick infestation and poor nutrition cannot be ruled out as possible causes of the predisposition of infected horses to piroplasmosis in this study. It is hereby recommended that best equine management practices are instituted.

KEYWORDS: Equine piroplasmosis, haematology, gross, histopathological lesions.

INTRODUCTION

Equine Piroplasmosis (EP) is a protozoan disease of Horses, Donkeys, Mules and Zebras caused by haemoparasitic protozoa belonging to the genera *Babesia* and *Theileria*: *Babesia caballi* and *Theilera equi* (Thank God *et al.*, 2019) and a newly reported species *Theileria haneyi* (Knowles et al., 2018). In general, *T. equi* infections are more clinically severe and common than *B. caballi* infections (de Waal, 2000; Donnellan and Marais, 2009). It is transmitted by ticks of different genera which include: *Dermacentor*, *Rhipicicephalus*, *Amblyomma*, *Hyalomma* and *Haemaphysalis* (Battseteseg *et al.*, 2001; Scoles and Ueti, 2015). Piroplasmosis is more endemic in most tropical and subtropical areas where tick vectors are known to exist than in temperate regions. This is due to high ambient temperature, humidity and rainfall which favour the development of ticks (Motlong *et al.*, 2008).

The disease occurs in three forms: the acute, subacute and chronic form. In the subacute form, clinical signs appear suddenly and may lead to death while in the acute form include fever, anaemia, inappetence,

haemoglobinuria, oedema, icterus, unsteady gait, increased respiratory, heart and pulse rates, hepatomegaly, splenomegaly and death (Rothschild *et al.,* 2013). In the chronic form, weight loss, poor performance and condition, intrauterine infections which can result in abortion and neonatal death are seen (Kouam *et al.,* 2010). The haemolysis of infected erythrocytes causes anemia in the majority of equids, regardless of the clinical manifestation of infection. The haematological profile of infected animals can be useful in diagnosis (ThankGod *et al.,* 2019). Gross pathological lesions include subcutaneous edema most especially of the eyelids and other subserosal tissues. splenomegaly, hepatomegaly, hemorrhages of the epicardial and endocardial tissues, discoloured and large kidneys and lymph nodes, and congestions of the lungs have been studied (Ueti *et al.,* 2008). Histological findings include centrilobular necrosis of the liver and microthrombi within the liver and lungs, renal tubular necrosis with haemoglobin casts, neuronal degeneration specifically in the Purkinje cells and geniestocytic astrocytes in the brain (de Waal and Van Heerden 2004; Al-Obaidi *et al.,* 2015).

Equine Piroplasmosis limits the performance of Horses and also causes serious economic losses to the equine industry which, include: treatment cost, abortions, loss of activity and death (Friedhoff *et al.,* 1996). Another factor that may contribute to an increase in Equine disease global spread is international trade of horses and other equids. Attempts to eradicate ticks have so far been efforts in futility. There are documented evidences on the occurrence, clinical and laboratory findings of EP in horses from some parts of Nigeria (Biu *et al.,* 2006; Takeet *et al.,* 2009; Ememe *et al.,* 2019; Oladipo *et al.,* 2015; Mshelia *et al.,* 2016), however, there is dearth of information on gross and histopathological lesions. This study was designed to investigate the occurrence, clinical signs and haematological parameters changes associated with *Babesia* positive horses as well as to examine the gross and histopathological lesions induced by these parasites in fatal cases.

MATERIALS AND METHODS

Fifty- eight (58) horses, randomly sampled, of different ages ranging from one and half years to nineteen years of both sex (39 males, 19 females) located in Ibadan, Southwest Nigeria were included in this study. They were clinically examined and 3ml of blood was collected via the jugular vein into a sterile bottle with EDTA anticoaggulant for haematological examination.

Clinical Assessment

The horses were examined for presence of tick infestation and emaciation. The degree of dehydration was evaluated (the presence of sunken eyes and skin elasticity). The mucous membrane was observed for anaemia and jaundice. The animals were also assessed for presence of epiphora and oedema of the fetlock joints, ventral abdomen and the scrotum, haemoglobinuria and ecchymosis of the third eyelid. Rectal temperature was done using digital thermometer to check for pyrexia.

Microscopic examination

Thin blood smear was prepared using a drop of blood on a grease free glass slide. After air drying and fixing with absolute methanol, glass slides was stained with 10% Giemsa stain in the laboratory. All slides were observed using immersion oil at 100x objective for the presence of *B. caballi* and *T. equi* using a light microscope (Jain 1986).

Haematological Analysis

The packed cell volume (PCV) was estimated by the microhaematocrit method and the haemoglobin (Hb) concentration by the cyanmethaemoglobin. Red blood cell (RBC) and white blood cell (WBC) count were determined using the new improved Neubauer haemocytometer. Differential leukocyte counts were evaluated using standard method as earlier described (Jain, 1986).

Postmortem examination

The postmortem examinations of fatal cases were carried out and tissues were processed for histological examination.

Histopathology

Tissues were collected immediately from fresh carcass and placed in a 10% formalin for proper fixation. The tissues were processed and embedded in paraffin wax. Sections of 5-6 microns in thickness were made on slides and stained with haematoxylin and eosin for histopathological examination using the method of Drury *et al*., (1976).

Data Analysis

The data obtained from this study were analyzed using descriptive statistics. The differences between the haematological parameters of infected and non-infected horses were compared using student t-test.

RESULTS

Five (8.62%) of the 58 horses examined were positive for the disease in this study while fifty-three were negative representing 91.38% of the total horse examined. All the positive horses revealed mixed infections with intraerythrocytic and extraerythrocytic *Theleira equi* and *Babesia caballi* (Fig 1a and 1b).

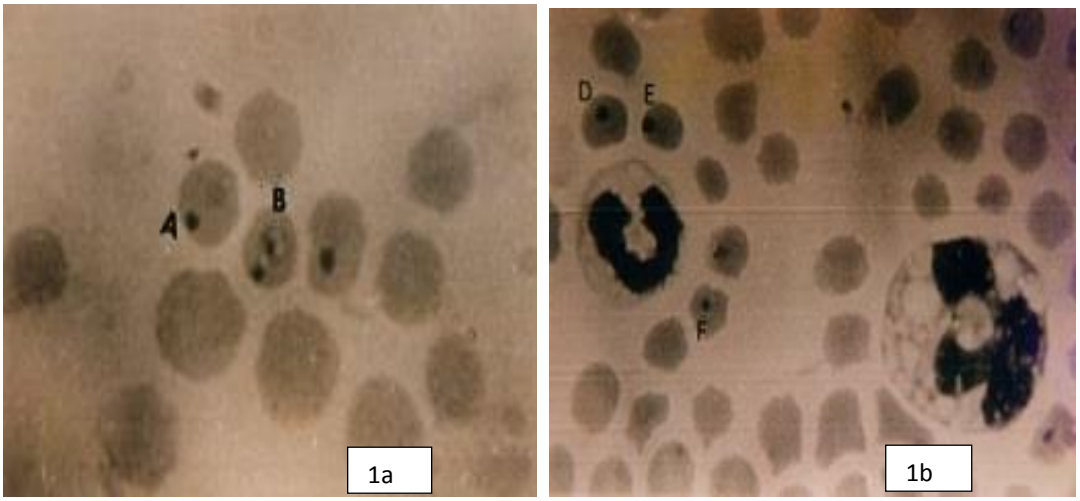


Fig (1a): A plate of mixed infections showing *T. equi* at RBC 'A' and *B.caballi* at RBC 'B'. (1b): Blood film showing *Theleira equi* in RBC 'D', 'E', and 'F'.

The *Babesia* positive horses were all male, representing 12.82% of the 39 male horses examined. Emaciation and dehydration observed ranged from subtle to severe (Fig 2a). Icterus masked pale mucous membrane was visible at the cornea, tongue, gum and the entire buccal cavity (Fig 2b). Oedema of the head, ventral abdominal wall, scrotal sac and fetlock joints, reluctance to move, weak hindquarters and unsteady gait were seen in acute cases. Ticks were recovered from mane, pinnae, perineum, base of tail, the body, scrotum and limbs (Fig 2c and 2d).

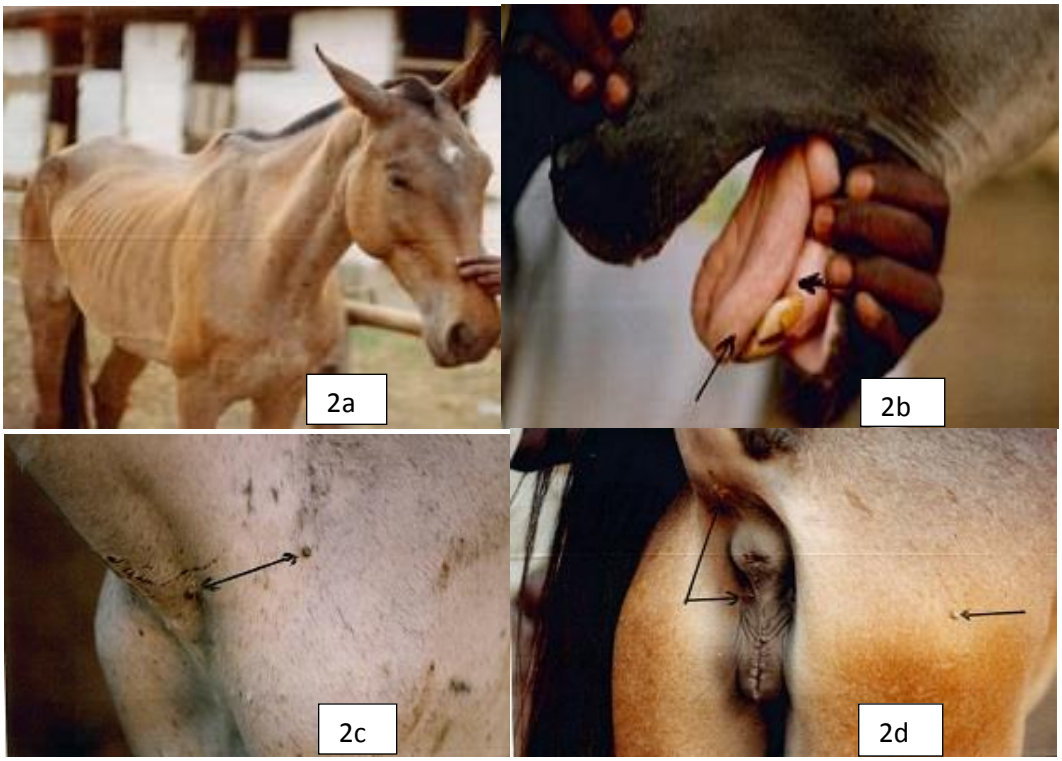


Fig. (2a): Heavily tick-infested and *babesia* positive, emaciated and dehydrated horse with prominent ribs and scapula Horse. (2b): Picture showing icteric tongue and gum. (2c and 2d): Presence of ticks at the ventral thoracic inlet, left cranial scapular, tail and perineum regions.

The overall mean of some haematological parameters of non-infected horses were: PCV (%) -34.40 ± 3.72, Hb concentration (g/dl)- 10.85 ± 1.33, RBC count (x 10^6/µL)- 11.72 ± 2.71, WBC count (x 10^3/µL)- 10.78 ± 3.05, lymphocytes (%)- 47.17 ± 10.46, eosinophils (%)- 1.61 ± 0.69, neutrophils (%)-51.40% ± 7.66 and monocytes (%)-1.13% ± 0.00 while the overall mean for infected horses were: PCV (%)- 26.00 ± 3.20, Hb concentration (g/dl)- 8.06 ± 1.51, RBC count (x 10^6/µL)- 6.43 ± 1.90, WBC count (x 10^3/µL)- 6.68 ± 1.09, lymphocytes (%)- 27.20 ± 0.71, eosinophils (%)- 1.50 ± 0.50, neutrophils (%) 67.80 ± 3.04 and monocytes (%)- 2.20 ± 0.72. (Table 1). PCV, Hb, RBC, WBC and lymphocytes count values in infected horses were

significantly ($p<0.05$) lower while neutrophils and monocytes count values were significantly ($p<0.05$) higher when compared with apparently normal horses (Table 1).

Tick infested horses were given tick bath using *diazinon dimplylate*. Clinically infected horses were treated with *Diminazene aceturate* (Berenil ®) at a dose rate of 3.5mg/kg body weight, double diluted, by deep intramuscular route at three different injection sites to prevent tissue necrosis.

Table 1: Haematological parameters of *Babesia* positive and *Babesia* negative Horses.

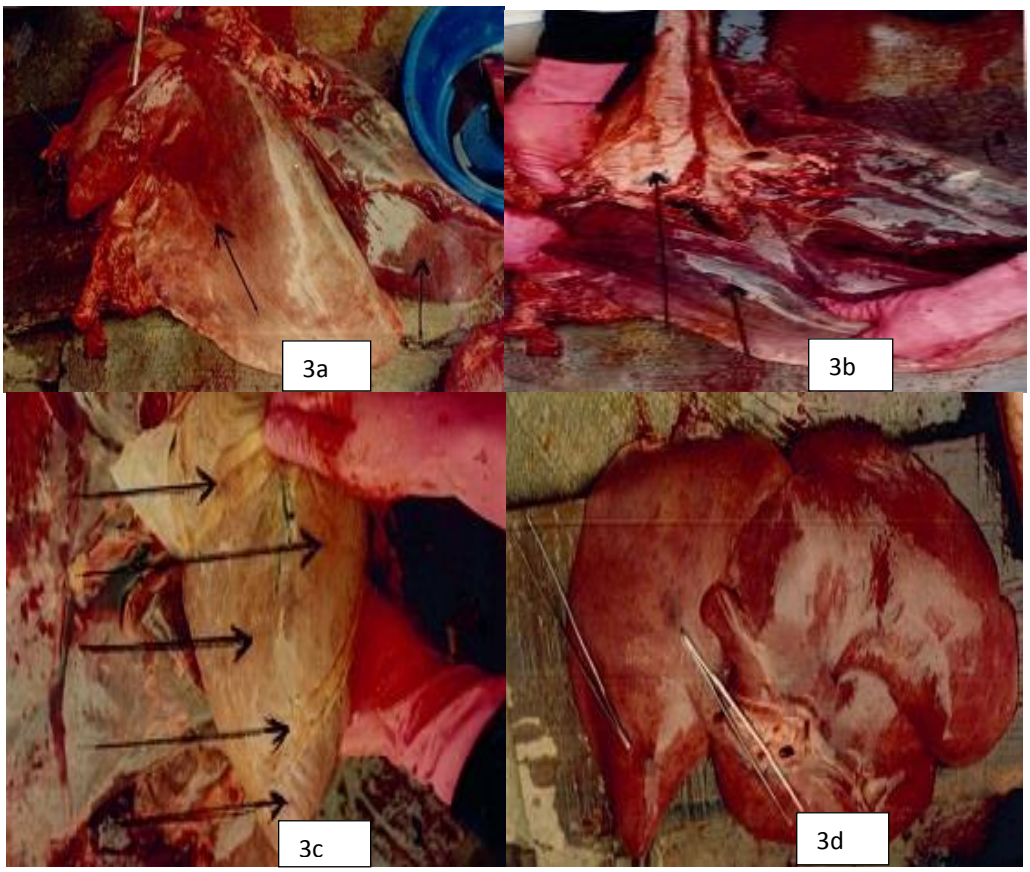
Parameters	**Values (Mean±SD)**	
	Infected	**Non- infected**
Packed cell Volume (PCV)%	26.00 ± 3.2^{b}	34.40 ± 3.72
Haemoglobin (Hb) Concentration (g/dl)	8.06 ± 1.51^{b}	10.85 ± 1.33
Red blood cell (RBC) count (x 10^{6}/µL)	6.43 ± 1.90^{b}	11.72 ± 2.71
White Blood Cell Count (WBC (x 10^{3}/µL)	6.68 ± 1.09^{b}	10.78 ± 3.05
Lymphocytes counts (%)	27.20 ± 0.71^{b}	47.17 ± 10.46
Eosinophils count (%)	1.50 ± 0.50	1.61 ± 0.69
Neutrophils count (%)	67.80 ± 3.04^{b}	51.40 ± 7.66
Monocyctes count (%)	2.20 ± 0.72^{b}	1.13 ± 0.00

$P<0.05$ is significant. Superscript b represents statistically significant difference.

Despite the treatment, three *Babesia* positive cases steadily had increasing parasitaemia and they became eventually fatal.

Postmortem examination

Carcasses of acute fatal and chronic cases were found lean and dehydrated with icteric ocular mucous membranes. Oral mucosae were markedly pale. The lungs were markedly congested and oedematous and serous fluid in the pleural and pericardial cavities (Fig 3a). Large quantity of yellowish tinged froth was present in the trachea, especially towards the tracheal bifurcation, bronchi and bronchioles with mucopurulent exudates (Fig 3b).



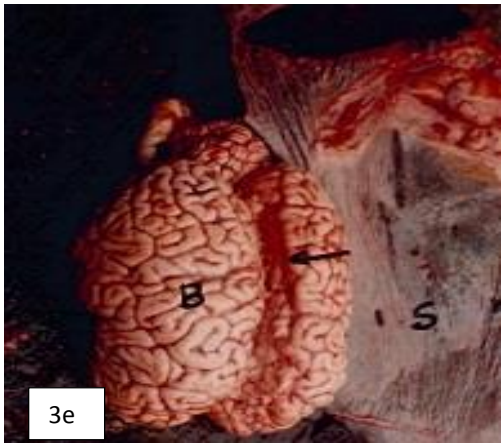
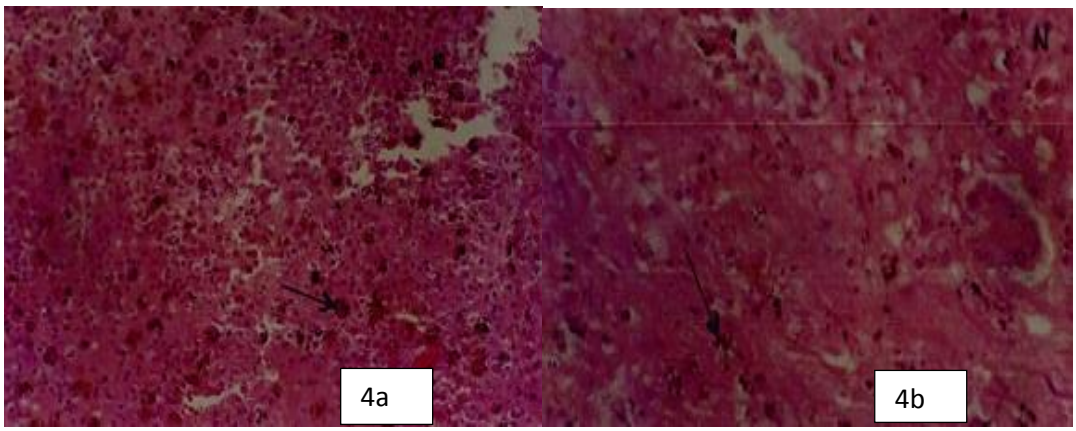


Fig. (3a): Picture showing haemorrhagic pericardium and congested and pneumonic lungs. (3b): Picture showing pneumonic lungs and mucopurulent exudates at the tracheal bifurcation and bronchi. (3c): Picture showing haemorrhagic coronary fat and haemorrhage of epicardium of the right ventricle as well as the necrosis of the apical region of the ventricle. (3d): Picture showing markedly enlarged liver, yellowish brown with diffuse petechial and ecchymotic subcapsular haemorrhages. Note the pale yellowish area. (3e): Moderate splenomegaly (S), and marked congestion of the brain, (B).

Pericardium was congested and serous atrophy of coronary fat with petechial haemorrhages (3c). There were haemorrhagic epicardium and necrosis of the ventricular apical region of the heart (Fig 3c). The right and left ventricles contained chicken fat clots while the ventricular musculature was markedly pale with ecchymotic haemorrhages on the endocardium. The liver was markedly enlarged, yellowish brown with diffuse petechial and ecchymotic subcapsular haemorrhages on the liver mixed with pale yellowish areas (Fig 3d). The spleens were moderately enlarged (Fig 3e), while the brain was markedly congested (3e).

The kidneys were very soft and congested with subcapsular cortical haemorrhages. The urinary bladders were markedly congested and distended with either straw coloured turbid urine, which contained a lot of gritty solids on the surface of the bladder's mucosa, or deep yellow to dark red coloured urine.

Histologically, the spleen had numerous haemosiderin-laden macrophages scattered within the splenic parenchyma especially in the red pulp (Fig 4a). The liver showed extensive multifocal severe hepatic necrosis with some areas of haemorrhage (Fig 4b). There were mild portal fibrosis and presence of haemosiderin in kupffer cells (Fig 4b). There was also presence of macrophages and neutrophils in the necrotic areas (Fig 4c). The kidney showed extensive coagulative necrosis of all structures with cystic dilation and markedly dilated renal tubules containing proteinacious materials and casts. Necrosis at the medulla region was mild (Fig 4d).



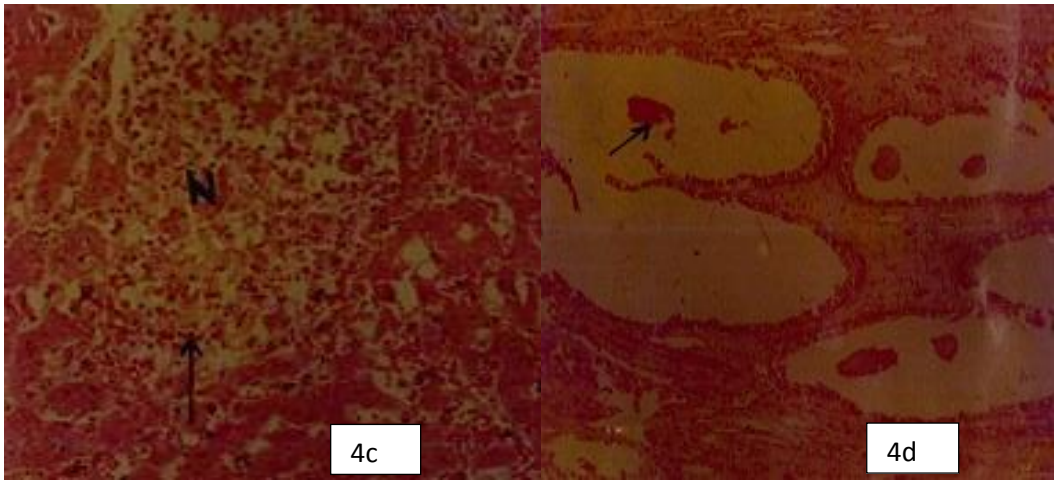


Fig (4a): Histological section of the spleen, (x100), numerous haemosiderin-laden macrophages scattered within the splenic parenchyma, especially in the red pulp. (4b) Histological section of the liver, (x160), showing hepatic necrosis (N), portal fibrosis (mild) and presence of haemosiderin in kupffer cells (arrow). (4c): Histological section of the liver, (x160) showing severe multifocal hepatic necrosis (N), with presence of macrophages and neutrophils in the necrotic areas (arrow). (4d): Histological section of the kidney, (x100), showing markedly dilated renal tubules containing proteinaceous materials/casts (arrow).

Brain sections showed marked congestion and thickening of meningeal blood vessel walls with moderate lymphocytic infiltrations of the meninges and margination of leucocytes in meningeal vessels (Fig 5a and 5b). The lungs were severely and diffusely oedematous. There were alveolar and interstitial pulmonary oedema, widening of interlobular septae by oedema fluids, and in some cases, perivascular fibrinous exudates were observed in interlobular spaces with fibroid degeneration of arterial walls (Fig 5c), and thrombosis of pulmonary blood vessels (Fig 5d).

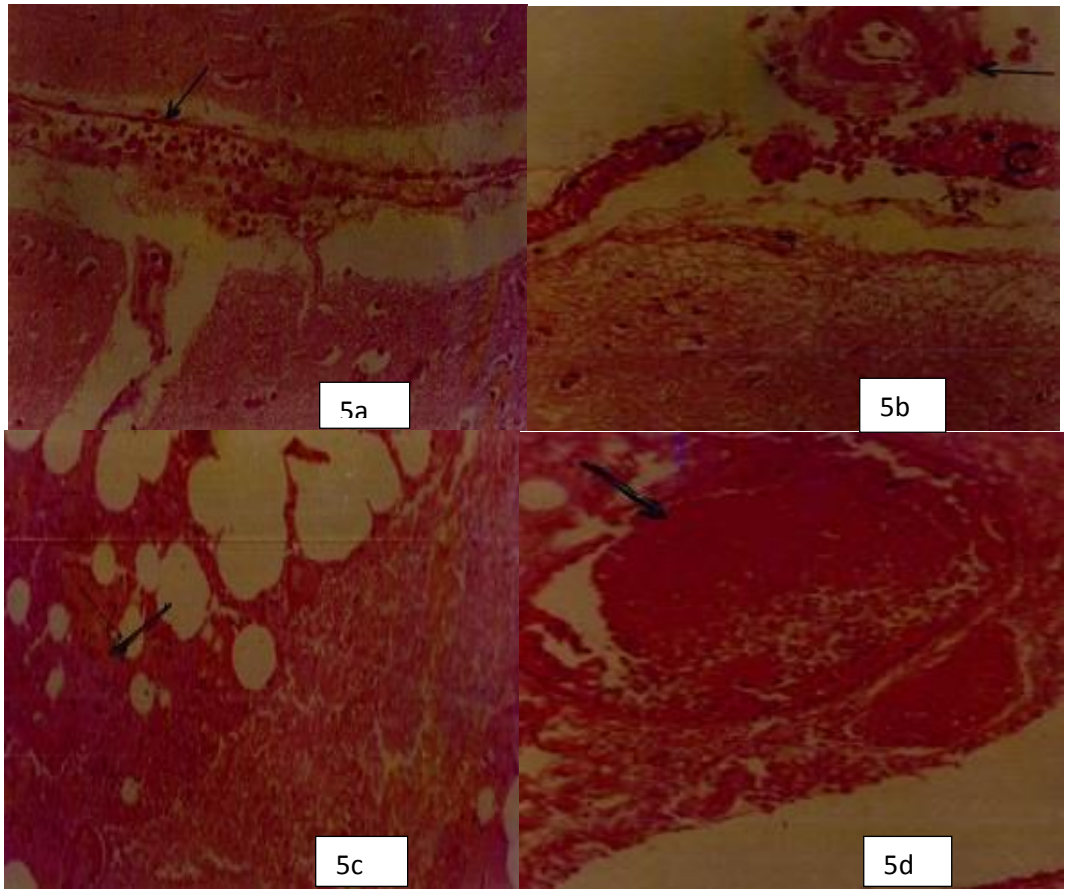


Fig (5a): Histological section of the brain, (x100), showing marginalization of leucocytes in meningeal vessels (arrow). (5b): Histological section of the brain (x100), showing markedly congestion of meningeal blood vessels

(C) with moderate lymphocytic infiltrations of the meninges (arrow). (5c): Histological section of the lungs, (x160), showing pulmonary oedema, (alveolar and interstitial), widening of the interlobular septae by oedema fluid (arrow). (5d): Histological section of the lungs, (x160), showing thrombosis of pulmonary blood vessels (arrow).

DISCUSSION

Equine piroplasmosis is endemic throughout Nigeria, and it is most prevalent during rainy season compared to dry season, which encourages vector proliferation (Tesfie et al., 2018). This study revealed a low prevalence rate of 8.62% of Equine piroplasmosis (EP) due to *T. equi* and *B. caballi* in Ibadan, Oyo State and is similar to prior report by Oladipo *et al.,* (2015). The higher prevalence in male horses than female horses agrees with the report of Ememe *et al*., (2019) but however in contrast with that of Oladipo *et al.,* (2015) in Ibadan, and Biu *et al.,* (2006) in Maiduguri, Nigeria. *T. equi, B. caballi*, and in some cases both parasites were found in the horses studied. These findings support an earlier report by Ememe *et al*., (2013) that mixed infections are common in naturally infected horses. Clinical signs observed in naturally infected horses in this study were similar to other studies reported by Radostitis *et al.,* (2007), Ibrahim *et al*., (2011) and Al-Obaidi *et al.,* 2015. Infected horses in this study were observed to be emaciated and they had heavy tick infestation. The role of major managemental negligence such as heavy tick infestation and poor nutrition cannot be ruled out as possible causes of the predisposition of infected horses to piroplasmosis in this study. Hypoprotenemia could be cause of the oedema around the fetlock joint, head, scrotal sac and ventral abdominal wall (Alsaad *et al.,* (2010) and Al-Obaidi *et al.,* (2015). The decrease in PCV, Hb and RBC of *Babesia* positive horses were similar with findings of Ememe *et al*., (2019) and Takeet *et al.,* (2009) which was due to the presence of the piroplasms, leading to the destruction of red blood cells. Anemia in piroplasmosis is caused by mechanisms which include a rapid intra-erythrocytic multiplication of the parasites as well as the phagocytosis of parasitized and non-parasitized erythrocytes by macrophages (Alsaad and Al-Mola 2006: Biu *et al.,* 2006). The leucopenia and neutrophilia observed in this study occur especially during the acute infections and this is similar to earlier reports by Zobba *et al.,* (2008) and Laus *et al.,* (2015) respectively. Also, the lymphopoenia seen in this study might have been due to a pre-erythrocytic stage of *T. equi* which includes intralymphocytic schizonts that are capable of blastogenesis and clonal expansion of predominantly T and B cells resulting in the lysis of infected lymphocytes (Homer *et al.,* 2000). Monocytosis was probably brought about by increasing need of macrophages in immune response to *Babesia* infection and may be observed in the early stages of the disease (Rosenblatt-Bin *et al.,* 1996: Rothschild and Knowles 2007). Morphological features at necropsy and histopathological findings were indicative of piroplasmosis in acute and chronic infection and in agreement with earlier reports (Rothschild and Knowles 2007, Alsaad and Al-Mola 2006, Al-Obaidi *et al.,* 2015 and ThankGod *et al.,* 2019). The non-effectiveness of the Babesiacidal drug used in this study might have been due to drug resistance due to a variety of mechanisms, including genetic alterations in target sites, reduced drug uptake and increased efflux, and metabolic regulation. The development of drug-resistant parasites has major implications for clinical cases, leading to increase morbidity and mortality (Fairlamb *et al.,* 2017).

CONCLUSION

In this study, the role of major management laxity, such as heavy tick infestation and poor nutrition, cannot be ruled out as possible causes of infected horses' susceptibility to piroplasmosis.

Further studies are required to do a molecular characterization of piroplasmosis organisms from horses in Ibadan, Nigeria and also to determine if they have resistant genes to commonly available anti-babesial drugs in Nigeria. Adequate and appropriate management practices are recommended.

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