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Clinical Article

A CASE REPORT ON EQUINE PIROPLASMOSIS IN A THOROUGHBRED HORSE FROM DURG, CHHATTISGARH AND ITS THERAPEUTIC MANAGEMENT

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SUMMARY

Ten year old male thoroughbred horse was presented with the history of colic, inappetence, dullness, nasal discharge and dark yellowish urine. Clinical examination revealed mild fever, tachypnoea and icteric conjunctival mucus membrane. Blood smear examination revealed presence of *Thileria equi* parasite in R.B.C. Haematological examination revealed anaemia, leukopenia accompanied by lymphopenia and thrombocytopenia while important biochemical alteration was increased level of unconjugated bilirubin in affected horse. On the basis of history, clinical and laboratory examination, the case was diagnosed as equine piroplasmosis and treated successfully with Inj. Buparvaquone @ 2.5 mg I/M and supportive therapy.

Keywords: Anaemia, Icterus, *Theileria equi*

Equine piroplasmosis is an important tick borne disease mostly caused by large form of *Babesia* species i.e. *Babesia caballi* and a *Theileria* species i.e. *Theileria equi* (Rothschild and Knowles, 2007). The disease is transmitted by tick species belonging to several genera such as *Hyalomma*, *Rhipicephalus* and *Dermacenter* and is endemic in tropical and subtropical regions of the world (Sumbria *et al.*, 2016a). Usually non-specific clinical signs like fever, depression, icterus, dyspnea, tachycardia, tachypnoea, colic and in coordination in acute cases whilst distinct clinical signs like anaemia, oedema, haemoglobinuria and even death in severe cases have been reported in equine piroplasmosis (Malekifard *et al*., 2014; Sumbria *et al*., 2016b). Generally, conventional parasitological techniques (stained thin blood smears) are being considered as gold standard method for detection of piroplasms in infected equids with acute signs (Chhabra *et al*., 2011). The shape of *T. equi* organisms in the infected erythrocyte varies from spherical to ovoid/Maltese cross. The organism may be found either singly, in pairs, or in tetrads (Malekifard *et al*., 2014). Affected animals can be treated with drug like imidocarb dipropionate, buparvaquone followed by supportive therapy (Salib *et al*., 2013).

Ten year old male horse belonging to Counter Terrorism and Jungle Welfare College, Kanker, Chhattisgarh was brought to the Large Animal OPD of Teaching Veterinary Clinical Complex of College of Veterinary Science and A.H., Anjora, Durg with the history of colic, inappetence, dullness, nasal discharge and dark yellowish urine. Clinical examination revealed temperature 101.4 ºF, heart rate 44/min, respiration rate 36/min, and icteric conjunctival mucus membrane (Fig.1). For diagnosis, blood was collected and examined for presence of haemoprotozoan parasite if any and analysis of haemato-biochemical parameters.

Microscopic examination of the Giemsa stained blood smear revealed the presence of small *T. equi* parasite in the erythrocyte either singly or in pair. Haematological examination was done by using automated haematological analyser. Biochemical examination was done using semi-auto analyser. Differential Leucocytes Count (DLC) was performed manually following standard procedure (Jain, 1986).

On the basis of clinical and laboratory examination, the case was diagnosed as equine piroplasmosis. The horse was treated with Inj. Buparvaquone @ 2.5 mg I/M, a second generation of hydroxynaphthoquinone as specific therapy. Supportive therapy included Inj. Flunixin meglumine @ 1.1 mg I/M, Multivitamin preparation -15 ml I/V daily for 5 days, Inj. Haematophos with B12 @ 15 ml I/M at alternate day for 3 occasions, Livoferrol syrup 50 ml orally twice daily and Inj. DNS- 500 ml x 10 bottle I/V, Inj. RL- 500 ml x 10 bottle I/V daily for 3 days. Haematophos and livoferrol were given to correct anaemia and to enhance appetite, fluid therapy viz RL and DNS was given to correct metabolic acidosis and dehydration due to less water intake. On 3rd day animal started eating and on 7th day of treatment it recovered completely as revealed by restoration of altered haemato-biochemical values to normal level (Table 1). Post treatment (on day 7) blood smear examination could not detect *Theileria equi* in the R.B.C of the treated horse.

In the present study, affected horse had reduced values of RBC, WBC, platelet, Hb, PCV and lymphocyte

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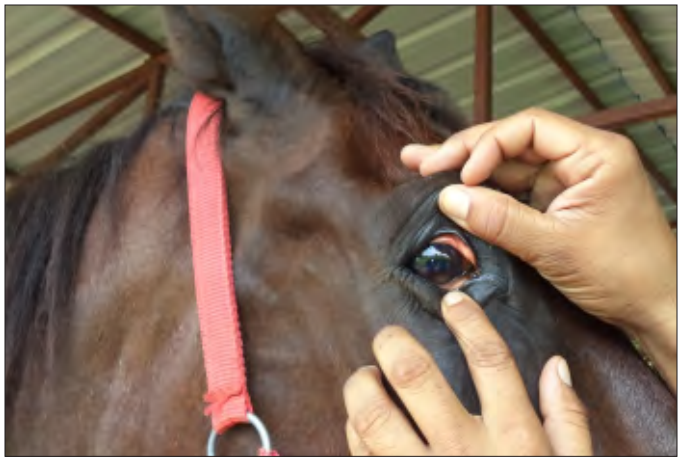

Fig. 1. Icteric conjunctival mucus membrane in horse suffering from Piroplasmosis

Table 1
Haemato-biochemical parameters of horse affected with piroplasmosis

Haemato-biochemical parameters	Pre-treatment	Post-treatment
Hb (gm/dl)	6.4	10.7
TEC (10^6/µl)	3.9	6.07
TLC (thousand/µl)	4,200	6,240
PCV (%)	22.7	33
Platelets ($\times 10^3$/µl)	73	224
Neutrophils (%)	65	56
Lymphocytes (%)	23	34
Eoosinophils (%)	9	7
Monocytes (%)	3	3
Total proteins (gm/dl)	7.03	6.6
Albumin (gm/dl)	3.03	3.4
Globulin (gm/dl)	4.0	3.2
Total bilirubin (mg/dl)	6.5	1.2
Unconjugated bilirubin (mg/dl)	6.20	0.5
Conjugated bilirubin (mg/dl)	0.30	0.7

indicating anaemia, leucopenia and thrombocytopenia. The observed biochemical alteration was increased level of unconjugated bilirubin indicating affected horse had intravascular haemolysis leading to pre-hepatic jaundice. However, it cannot be ignored that the recorded alterations in the blood of infected animal may be due to other factors, collectively; these associations suggest that the macrocytic hypochromic anaemia is the significant pathological alteration in infected horse. Similar findings have also been recorded in experimentally infected piroplasmosis in equine (Zobba *et al.*, 2008). Causes of haemolysis might be attributed to mechanical intra-erythrocyte binary fission of trophozoite, immune-mediated auto-antibodies directed against components of the membranes of infected and uninfected erythrocytes, and toxicity by haemolytic factors produced by the parasite (Mahmoud *et al.*, 2016). The cause of decreased haemoglobin is mostly due to destruction of RBCs. Haemolytic anaemia causes pre-hepatic jaundice leading to increased bilirubin. Icterus is commonly observed in infected horses due to increased serum concentration of bilirubin (Radostitis *et al.*, 2008). Present findings corroborated the earlier findings of Zaugg (1993) who reported treatment of equine piroplasmosis (*Babasia equi*) of European origin by buparvaquone. Buparvaquone, a hydroxyquinone derivative has been investigated for the treatment of leishmaniasis, cryptosporidiosis and equine piroplasmosis and is recommended as gold standard for the treatment of theileriosis. Recent studies on *Babesia* have reported the development of resistance to Diminazene aceturate and documented toxic side effects in imidocarb dipropionate treated equines (Nugraha *et al.*, 2019).

This study indicates that henceforth in Chhattisgarh, *T. equi* infection should be considered in the differential diagnosis of horses with (acute) signs of depression, inappetence, anaemia and icterus. Though several reports are available from time to time claiming presence of infection in India (Chhabra *et al.*, 2011; Kashyap *et al.*, 2014; Sumbria *et al.*, 2016a and Sumbria *et al.*, 2016b), but there is no report available from the state of Chhattisgarh. Hence, there is need of further investigations on occurrence of disease in equine population in the area using advanced diagnostics.

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