RECURRENT EPISTAXIS WITH THROMBOCYTOPENIA SECONDARY TO BABESIOSIS IN A MURRAH BUFFALO-A CASE REPORT

Vivek Joshi^{1,*}, Umesh Dimri², Arumugam Gopalakrishnan², Bhanuprakash Asandi Govindappa² and Shahjahan Alam²

ABSTRACT

A two year old Murrah she buffalo was presented with a history of inappetence, suspended rumination, passage of dark brown urine, bleeding from the nose, sudden decrease in milk yield, and weakness for the last three days. The clinical examination revealed a high rise of rectal temperature (104.2°F), severe depression, ocular discharge, icteric mucous membranes, and hemoglobinuria. The buffalo was mildly infested with dark colored ticks, Rhipicephalus microplus. Surprisingly, recurrent episodes of epistaxis were recorded in buffalo. Hemato-biochemical analysis revealed a reduction in Hb, TEC, HCT and PLT while an increase in serum bilirubin, glucose and AST levels. Microscopic examination of Giemsa stained blood smears from the ear vein confirmed the presence of Babesia bigemina piroplasms in the erythrocytes. Buffalo babesiosis was effectively treated with diminazine aceturate at a single dose of 3.5 mg/kg body weight, deep intramuscularly, along with ancillary therapy. The buffalo was afebrile 8 h post-treatment but epistaxis disappeared only after 48 h. This seems to be the first report of recurrent epistaxis with thrombocytopenia associated with buffalo babesiosis in India.

Keywords: *Bubalus bubalis*, buffaloes, Babesiosis, diminazine aceturate, epistaxis, hemoglobinuria

INTRODUCTION

Babesiosis is one of the most important tick-borne diseases of cattle and buffaloes caused by blood parasites of the genus Babesia. The main arthropod vectors of B. bigemina and B. bovis are one-host hard ticks, Rhipicephalus spp. and incubation period often varies from 2 to 3 weeks after tick infestation (Kuttler, 1988; Talkhan et al., 2010; El-Far et al., 2014). Bovine babesiosis is prevalent worldwide in tropical and sub-tropical countries, however, buffalo babesiosis is a very rare disease being mostly recorded in India and China where it is considered to be a major health problem for the livestock sector (Zhongling et al., 1997). Buffalo babesiosis is characterized by high fever (102.5°F to 104.2°F), anorexia, lethargy, jaundice, emaciation, depression, reduced milk production, hemoglobinuria and anemia in advanced stages of infection. Haematobiochemical alterations include reduction in Hb, PCV and TEC, elevation in AST, hyperglycemia, hyperbilirubinemia and hypoprotienemia (Rani

¹Animal Health Section, Indian Council of Agricultural Research, National Research Centre on Mithun, Nagaland, India, *E-mail: joshignet@gmail.com ²Division of Medicine, Indian Council of Agricultural Research, Veterinary Research Institute, Uttar Pradesh, India et al., 2010). Death may occur within a few days in acute infection characterized by fever, anemia (PCV falls below 20%) and hypotensive shock syndrome (Urguhart et al., 1996; Homer et al., 2000; Francisco et al., 2013). Severe pathological conditions like cerebral babesiosis and respiratory distress can also lead to death (Herwaldt et al., 1996). Diagnosis of babesiosis is usually done on the basis of examination of Giemsa stained thin blood smears under oil immersion. Thin blood smears are prepared with peripheral blood from the ear vein and air dried. It is then fixed in absolute methanol for 1 to 2 minutes and stained with 10% Giemsa stain for 20 to 30 minutes. The slides are washed under running tap water, left to dry in air and examined under high power objective. If three blood smears are negative, animals is considered free from infection (El-Far et al., 2014). Mostly single and pyriform merozoites in early stage and rod-like in later stage of infection are seen in buffalo (Liu et al., 1986).

In the past few years, many researchers have studied different aspects of buffalo babesiosis and recorded distinct differences between bovine babesiosis and buffalo babesiosis. The differences are present in the pathogen, epidemiology, tick vector and immune reactions induced in animals (Zhongling *et al.*, 1997). The cases of buffalo babesiosis are less commonly encountered and rarely accompanied by recurrent episodes of epistaxis in buffalo. The present paper describes an unusual case of babesiosis in a she Murrah buffalo presented with epistaxis and its successful management. This is believed to be the pioneer report of its kind in buffalo babesiosis.

CASE PRESENTATION AND CLINICAL OBSERVATIONS

A Murrah she buffalo aged 2 years was brought to the Referral Veterinary Polyclinic, Indian Council of Agricultural Research, Indian Veterinary Research Institute, Izatnagar with the complaints of recurrent bleeding from the nose, passage of coffee coloured urine, absence of rumination, anorexia, reduced milk production, profuse ocular discharge, sudden weakness and ataxia for the last three days. On close physical examination, buffalo was found to be mildly infested with ticks, Rhipicephalus microplus (Figure 6), with the major infestation in the groin area. The clinical examination revealed the presence of tachycardia (74 bpm), elevated respiration rate (37 per minutes) and a high rise of temperature (104.2°F). Severe icterus was evident on the examination of conjunctival (Figure 1) and vaginal mucous membranes (Figure 4). Recurrent episodes of bilateral bleeding from the nose of buffalo was the only unusual clinical finding recorded, with frank and partially clotted blood being present on the external nares (Figure 2).

Heparinized blood and serum samples were collected and sent to the clinical laboratory for haemoprotozoan examination, complete blood count (CBC), and serum biochemical analysis. Giemsa stained blood smears revealed the presence of numerous paired pyriform *Babesia bigemina* piroplasms in the red blood cells of buffalo (Figure 5) and parasitemia rate was found to be about 4%. Other laboratory results indicated severe anemia, hyperbilirubinemia and jaundice, mild thrombocytopenia, leucocytosis, elevated serum AST level, hypoproteinemia, and hyperglycemia (Table 1).

Parameter	Observed values	Reference values
TLC (x 10 ³ /µL)	12.51	4-12 (Feldman <i>et al.</i> , 2000)
TEC (x 10 ⁶ /µL)	4.21	5-10 (Feldman <i>et al.</i> , 2000)
Hb (g/dl)	5.20	8-15 (Feldman et al., 2000)
HCT (%)	15	24-46 (Feldman et al., 2000)
PLT (x $10^{3}/\mu$ L)	82	100-800 (Feldman et al., 2000)
Neutrophils (%)	51	15-45 (Feldman et al., 2000)
Lymphocytes (%)	39	45-75 (Feldman et al., 2000)
Monocytes (%)	4	2-7 (Feldman et al., 2000)
Eosinophils (%)	6	0-20 (Feldman et al., 2000)
Basophils (%)	0	0-2 (Feldman et al., 2000)
Serum glucose (mg/dl)	89	45-75 (Kaneko et al., 1997)
ALT (IU/L)	35	11-40 (Kaneko et al., 1997)
AST (IU/L)	144	78-132 (Kaneko et al., 1997)
Total protein (g/dl)	6.23	6.74-7.46 (Kaneko et al., 1997)
Total bilirubin (mg/dl)	3.71	1-2 (Kaneko <i>et al.</i> , 1997)

Table 1. Haemato-biochemical constituents of a Murrah buffalo with babesiosis.



Figure 1. Icteric conjunctiva.



Figure 2. Epistaxis from left nostril.



Figure 3. Coffee coloured urine.



Figure 4. Icteric vaginal mucous membrane.

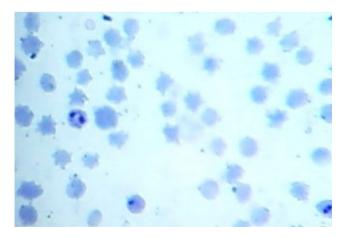


Figure 5. Piroplasms of *B. bigemina* in RBCs.



Figure 6. Rhipicephalus microplus.

TREATMENT

The affected buffalo was treated with a single dose of Diminazine aceturate (Berenil, Intervet India Pvt Ltd) at the rate of 3.5 mg/ kg body weight, by deep intramuscular route. Intravenous injection of epinephrine (Adrenaline, Gaco Pharmaceuticals Ltd.) at the rate of 10 mcg/ kg (0.25 to 0.50 ml per 45 kg) and intramuscular injection of vitamin K3 (Kapilin, Glaxo Smithkline Pharmaceuticals Ltd.) at a dose rate of 0.5 mg/kg once daily, were administered for three consecutive days. To control ticks, the buffalo was sprayed with deltamethrin (Butox, MSD Animal Health) after an adequate dilution of 2 ml/l of water. The supportive therapy included meloxicam (Melonex, Intas Pharmaceuticals Ltd.) at the dose of 0.5 mg/ kg IM, chlorpheniramine maleate (Cadistin, Zydus Animal Health Ltd.) 0.5 mg/kg IM, multivitamins (Vitakey, Zydus Animal Health Ltd.) 5 ml IM for 5 days and five injections of a hematinic (Feritas, Intas Pharmaceuticals Ltd.) at the rate of 100 mg IM on 0, 3^{rd} , 6^{th} , 9^{th} and 12^{th} day of therapy.

DISCUSSION

limited literature is available Only regarding the prevalence of babesiosis in buffalo in India. The clinical signs chiefly hemoglobinuria, anemia, and icterus may be attributed to the rapid proliferation of Babesia organisms in peripheral blood vessels resulting in extensive intravascular hemolysis, phagocytosis of infected as well as non-infected red blood cells, and bone marrow suppression leading due to diminished erythropoietic activity. These all collectively could lead to lowered values of Hb, PCV and TEC (Rani et al., 2010). Stress occurring during buffalo babesiosis may account for leucocytosis (Bhikane et al., 2001).

Increased pulse and respiratory rates might have resulted due to anemic hypoxia. This elevation may be a part of compensatory mechanism to relieve

tissue hypoxia (Ali et al., 1995; Tufani et al., 2009). Hyperglycemia may be attributed to tissue hypoxia occurring secondary to large scale intravascular hemolysis. Hyperbilirubinemia and subsequent jaundice might have resulted due to widespread destruction of red blood cells (Kumar et al., 1995). Diminazine aceturate is highy effective for the treatment of clinical cases of buffalo babesiosis and it acts by hampering the replication of DNA of Babesia parasite (Bhatt et al., 2005; Kumar et al., 2008). Epistaxis evident during buffalo babesiosis is attributed to thrombocytopenia, which may result from decreased marrow production. hypersplenism, utilisation due to disseminated intravascular coagulation, and immune-mediated platelet destruction (Pantanowitz, 2002).

REFERENCES

- Akhtar, M.Z., A. Khan, M.Z. Khan and G. Muhammad. 2007. Haemato-biochemical aspects of parturient haemoglobinuria in buffalo. *Turk. J. Vet. Anim. Sci.*, 31(2): 119-123.
- Ali, M.K., A.K. Pramanik and C. Guha. 1995. Babesiosis in calves and its treatment. *Indian Vet. J.*, 72: 1289-1290.
- Bhatt, P., S. Kumar and S. Kumar. 2005. Babesiosis in a cross bred calf: A case study. *Indian J. Vet. Med.*, **25**: 60.
- Bhikane, A.U., B.W. Narladkar, L.G. Anantwar and A.P. Bhokre. 2001. Epidemiology, clinicopathology and treatment of babesiosis in cattle. *Indian Vet. J.*, 78: 726-729.
- El-Far, A.H., N.A. Bakeir and H.M. Shaheen. 2014.
 Anti-oxidant status for the oxidative stress in blood of *Babesia* infested buffaloes. *Global Veterinaria*, 12: 517-522. DOI:

10.5829/idosi.gv.2014.12.04.8343

- Feldman, B.F., J.G. Zinkl and N.C. Jain. 2000. Schalm's Veterinary Haematology, 5th ed., Lippincott Williams and Wilkins, Philadelphia, USA.
- Francisco, A.L.S., F.V.B. Juliana, V.P. Lidiany, J.S.C. Ciro, E.A. Crica, F.B.R. Mucio, L.S. Renato and M.M.S.S. Silvana. 2013. Babesiosis and anaplasmosis in dairy cattle in Northeastern Brazil. *Pesqui. Vet. Brasil.*, 33(9): 1057-1061. DOI: 10.1590/S0100-736X2013000900002
- Herwaldt, B.L., D.H. Persing and E.A. Precigout.
 1996. A fatal case of babesiosis in Missouri: Identification of another piroplasm that infects humans. *Ann. Intern. Med.*, 124(7): 643-650. DOI: 10.7326/0003-4819-124-7-199604010-00004
- Homer, M.J., I. Aguilar-Delfin, S.R. Telford, P.F. Krause and D.H. Persing. 2000. Babesiosis. *Clin. Microbiol. Rev.*, **13**(3): 451-469. DOI: 10.1128/CMR.13.3.451
- Kaneko, J.J., J.W. Harvey and M.L. Bruss. 1997. Clinical Biochemistry of Domestic Animals, 5th ed., Academic Press, San Diego, USA.
- Kumar, A. and B.P. Joshi. 1995. Studies on blood glucose levels as a metabolic profile in clinical appraisal in buffaloes. *Indian J. Vet. Med.*, 15: 71-73.
- Kumar, B., S.P. Verma, B. Sinha and S. Shekar. 2008. Therapeutic efficacy of alum-boric acid in bovine babesiosis. *Indian Vet. J.*, 85(10): 1107.
- Kuttler, K.L., W.L. Goff, C.A. Gipson and B.O. Blackburn. 1988. Serologic response of *Babesia equi* -infected horses as measured by complement-fixation and indirect fluorescent antibody tests. *Vet. Parasitol.*, 26: 199-205.

- Liu, Z.L., L.H. Ma, G.D. Zhang and X.S. Gao. 1986. An investigation of babesiosis in buffaloes in Hubei province. Acta Veterinaria et Zootechnica Sinica, 17: 49-54.
- Pantanowitz, L. 2002. Mechanisms of thrombocytopenia in tick-borne diseases. *The Internet Journal of Infectious Diseases*, **2**(2).
- Rani, N.L., C. Sreedevi, P. Annapurna and K.A.
 Kumar. 2010. Clinical management and haemato-biochemical changes in babesiosis in buffaloes. *Buffalo Bull.*, 29: 92. Available on: http://ibic.lib.ku.ac.th/e-Bulletin/ IBBU201000021.pdf
- Talkhan, O.F.A., M.E.I. Radwan and M.A. Ali.
 2010. Cattle babesiosis and associated biochemical alteration in Kalubyia Governorate. *Nature and Science*, 12: 24-27. Available on: http://www.sciencepub.net/nature/ns0803/05_2176_mervat_ns 0803 29 36.pdf
- Tufani, N.A., A. Hafiz, H.U. Malik, F.U. Peer and D.M. Makhdoomi. 2009. Clinicotherapeutic management of acute babesiosis in bovine. *Intas Polivet*, **10**(1): 49-50. Available on: https://www.academia. edu/1058745/Clinico_therapeutic_ management_of_acute_babesiosis_in_ bovine
- Urquhart, G.M., J. Armour, J.L. Duncan, A.M. Dunn and F.W. Jennings. 1996. *Veterinary Parasitology*, 2nd ed. Book Power, Blackwell Science, Scotland. p. 243-244.
- Zhongling, L., Z. Junlong, M. Lihua and Y. Baoan.
 1997. Studies on buffalo babesiosis in Hubei province, China. *Trop. Anim. Health. Pro.*, 29(4 suppl.): 33S-36S. DOI: 10.1007/ BF02632914