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A clinico pathological and therapeutic report on Babesiosis in buffaloes

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Abstract
Clinico pathological and therapeutic aspects of Babesiosis in two graded Murrah buffaloes were described. The animals were reported to Veterinary Hospital Eluru, West Godavari district, Andhra Pradesh, India during June-July 2017. Pyrexia (103.8°F) anorexia, hemoglobinuria, poor body condition scores and dehydration were prominent clinical signs and ataxia and staggering gait were also observed. Intra erythrocytic *Babesia bigemina* organisms could be found in peripheral blood smear. Hematological examination revealed anemia (Hb 6.1 & 2.7g/dl; low RBC 3.47X10*6/µl & 1.1X10*6 µl; PCV 20% & 10.7%) thrombocytopenia, lymphocytosis, monocytosis, elevated hepatic enzymes, (AST 165.6 & 286 IU/L) creatinine (2.5 & 4.6mg/dl), hypoalbuminemia (2.5 & 2.1g/dl) and hypocalcemia (8.66 & 7.65mg/dl). Both the animals were treated with diamenazene @3.5 mg/kg, along with intensive support therapy. Buffalo 1 recovered with regressed parasitemeia within 24hrs and urine color became normal in 48 hours, apatite and production could be restored after supportive treatment for 10 days. Buffalo 2 died despite intensive treatment in with significant hemato-biochemical abnormalities.

Keywords: *Babesisa bigemina*, haemoglobinuria, anemia, hemato-biochemical changes. diamenzene, anemia

1. Introduction
Of the prevalent tick born haemoprotozoan diseases among animals, infections caused by hematotropic parasites of the genus *Babesia* is important in view of their widespread nature which is second only to trypanosomosis [1]. This intraerythrocytic protozoon is prevalent among tropical and subtropical countries including India causing significant morbidity and mortality [2]. The clinical syndrome of babesiosis has considerable impact on health, production and economic viability of effected animals [3]. *B bigemina* and *B bovis*—are widespread in tropics and subtropics and as there are many common features of the diseases caused by different *Babesia*, much of the information pertaining to them can be applied to other species [4]. Infection, sub acute to chronic can cause retarded growth in calves, death, increased abortion rate, sterility, reduced milk and meat production and high costs of prevention and treatment [5]. Incidence in costal Andhra Pradesh is periodic, influenced by climatic stress, nutritional deficiencies, changing breed composition of herd, selection of geographically unadopted breeds, tick population and lack of acaricidal treatment. The objective of the present study was to discuss the varied clinico pathological and therapeutic aspects of Babesiosis in two buffaloes presented to veterinary hospital Eluru of West Godavari district.

2. Materials and Methods
Two graded Murrah buffaloes (1&2) presented to Veterinary Hospital Eluru, West Godavari district Andhra Pradesh, India, between June-July 2017, constituted the subjects of the study. History includes anorexia, depression and coffee colored urine. The animals were maintained under semi intensive housing system with poor ventilation and were stall fed. Moderate pyrexia (103.4^0F), haemoglobinuria, anorexia, reduced ruminal moments constipation were observed in both and ataxia, staggering gait, muscle tremor, constipation were recorded in the second animal. Mucus membrane are pale pink in the first and pale in the second but there is no icterus in both the animals. Tick infestation inside the ears and thighs was observed in both the animals.
Thick and thin blood smears collected from ear vein were stained with Giemsa stain [6]. Blood in K2 EDTA and serum samples were collected and subjected to hematological and bio

chemical analysis utilizing fully automatic veterinary hematological analyzer, PE6800Vet and Optimas Lab India automatic biochemical analyzer utilizing the reagent kits of ERBA. Dung samples were examined for parasitological infestation. General and body condition scores were recorded and animals were examined for co-morbidities. The hemato biochemical values are compared with those of healthy lactating Murrah buffaloes of the same locality available with the laboratory for the purpose of interpretation.

3. Results and Discussion

The general condition was poor and the body condition scores ranged from 2.0-2.5 indicating lowered energy levels and mobilization of adipose tissue [7] possibly due to existing management and poor body condition lowers the immunity and encourages the infection of animal by hemoprotozoan infections like Babesia [8]. Fever, anaemia, loss of appetite, suspended rumination, pale mucus membranes and haemoglobinurea, which are in agreement with Radostits *et al.,* (2000) [9]. Dung samples revealed no parasitic ova and there are no noticeable unrelated co morbidities allowing the attribution of pathological condition to babesisos. The blood smear of these animals revealed pyriform bodies of *B. bigemina* at an acute angle inside the erythrocytes and other pleomorphic forms of *B. bigemina* were also observed [10]. The classical microscopic examination of *Babesia* piroplasms in Giemsa stained thin blood smear is a gold standard test that is relatively cheap and quick method though in chronic infection, it has low sensitivity and usually fails to detect carrier animals [11].

Anemia, thrombocytopenia, neutrophilia, lymphopenia and monocytosis were the prominent hematologic alterations observed and in agreement with Aulakh *et al.,* (2005) [12]. Anisocytosis (spherocytes), normocytic to macrocytic hypochromic regenerative anemia with polychromatophilic RBC are indicative of hemolytic changes. Marked anemia can be due erythrolysis, indiscriminate phagocytosis of RBC by activated macrophage system [13] and suppression of erythopoietic activity of bone marrow [14]. In acute infections there is large scale destruction of erythrocytes with intravascular hemolysis, indiscriminate phagocytosis of infected / noninfected erythrocytes by activated macrophage system and suppression of erythropoietic activity of bone marrow, and these contribute to anemia [15]. In *B. bigemina* parasitemia is adept to cause a major alteration in total RBCs osmotic fragility even intact unaffected RBCs, these red cells become easily destroyed creating hemolysis [16]. Oxidative stress is a disproportion between scavenging mechanism and radical generating mechanism [17]. The present observations are similar to those of Salem *et al.,* (2016) [18] who found decrease in RBCs, HB, PCV, MCHC and platelets values along with significant increase in MCV in babesiosis in calves where in the erythrogram results advocates the presence of macrocytic hypochromic anemia. The erythrocyte peroxidation and lysis occurs in hemo-protozoan infection can be correlated to disease pathogen [19]. Decreased platelets can be due to decreased marrow production, hyperspleenism, consumption as a result of widespread endothelial damage or disseminated intravascular coagulation [20]. Thrombocytopenia is usually correlated with immune-mediated process and hemolysis [21]. The nervous symptoms can be due to sequestration of infected RBC in the brain capillaries [22].

Increase in the levels of AST, ALT and creatinine signifies harmful effect of toxic metabolites of *Babesia* sp. on hepatic and renal tissue leading to impairment and alterations of the liver and kidney enzymes and hemoglobinuric nephrosis [9, 23]. Hussein *et al.,* (2007) [24] reported the significant increase in aspartate aminotransferase, alanine aminotransferase and gamma glutamyltransferase (GGT) in babesiosis. Anemia and hemato-biochemical changes are more pronounced in Buffalo 2 where in significant hypoalbuminemia, hypoglycemia and hypocalcemia are recorded which can be attributed to anorexia and inadequate supplementation. Low albumen levels may be due to pronounced hemolytic crises, proteinuria associated with renal failure and anorexia in relation to high rise of body temperature [25] and anemic anoxia complicated by circulating immunocomplexes [12]. Reduction of albumin level probably corresponds to disturbance in liver function, urinary loss of albumin associated with proteinuria and anorexia. Babesia can cause disruption in liver function that leads to decrease albumin synthesis [26].

3.1 Treatment

Diminazene aceturate is effective against *B. bigemina*, but less effective against *B. bovis* and *B. divergens* [27]. This is locally available and economical than imidocarb. Both the animals were treated with ready to use Diminazene diaceturate inj. @3.5mg /Kg BW (Berinil RTU Hoechest) given as a single dose. Flunixine meglumine@2.2 mg/Kg (Megludyn, Virbac Pharma) BID day as an antipyretic and anti-inflammatory for 3 days. Dextrose normal saline @ 25ml/kg to combat the moderate dehydration and also to flush hemoglobin sequestrated in renal tissue. Iron sorbital 10mg /kg as a single injection (inj. Ferritas, Intas pharma) deep intramuscular to combat anemia. B-complex and liver extract preparations (Rumrec, Virbac pharma) were given @ 10ml /animal /5days as hepato regenerative interventions. Protozoa were cleared from peripheral blood after 12 hours of treatment and by the end of 24 hours the color of the urine was almost normal in both the animals and appetite was partially restored in the first animal which received continued supportive therapy with mineral supplements, concentrate feeding and oral iron sorbital for a period of 15 days. However the second animal deteriorated, recumbent and succumbed on the second day of treatment despite intensive therapy which can be attributed anemic anoxia and hepato renal damage. It is opinioned that supportive therapy is as important as specific treatment and the recovery is influenced by severity of anemia and extent of pathological changes.

Table 1: Hematobiochemical changes due to Babesisos in buffaloes.

S. No	Parameter	Buffalo 1	Buffalo 2	Normal values
1	Hemoglobin g/dl	6.1	2.7	8-15.0
2	RBC 10 *6/ µl	3.47	1.1	5.0-10.0
3	WBC 10*3/µl	3.8	2.5	4.0-12.0
4	PCV %	20	10.7	25-35
5	MCV fl	58	98.6	40.0-60.0
6	MCH pg	17.5	24.5	14.0-18.0
7	MCHC g/dl	30.5	25.2	30.0-36.0
8	Platelets 10*3/µl	127	46	100-800
9	Glucose (mg/dl)	60.8	38.5	45-65
10	Total protein (g/dl)	7.4	7.2	6.0-8.0
11	Albumen (g/dl)	2.8	2.1	2.5-3.8
12	Calcium mg/dl	8.66	7.65	9.0-11.0
13	AST IU/lt	165.5	286	60–125
14	ALT IU/lt	46.5	88	23.0-90.0
15	Creatinine.mg/dl	2.4	4.6	1.02-2.17

4. Conclusion

Babesisos as a clinical entity is comparatively rare among buffaloes though chronic and carrier states are suspected. Identification of protozoa with Giemsa stained blood smears collected at the height of pyrexia is adequate under field

conditions. The presence of a few infected ticks or even a clinically infected animal may acts as focus of infection for the other susceptible animals in the area and may be responsible for subclinical infection or carrier state threatening the health status and economic viability of animals. Evaluation of hemato biochemical changes is essential to know pathognomic effects on the animal including type and severity of anemia and extent of involvement of vital organs and their functioning and can be of immense value to predict a therapeutic outcome. Severe anemia and significant organ damage are responsible for the death despite the treatment. Early detection, specific chemotherapy and intensive supportive therapy are essential for the successful management of the clinical babesisos in buffaloes.

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