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IMPACT OF SUBCLINICAL ANAPLASMOSIS ON HEMATO-BIOCHEMICAL PARAMETERS OF NATURALLY INFECTED CAMELS (*Camelus dromedarius*)

Muhammad Zaeem Abbas[1,*], Muhammad Saqib[1], Farrah Deeba[1] and Muhammad Sohail Sajid[2]

[1]Department of Clinical Medicine and Surgery, University of Agriculture, Faisalabad-38400, Pakistan;[2] Department of Parasitology, University of Agriculture, Faisalabad-38400, Pakistan
***Corresponding author's e-mail: zaeemabbas45@gmail.com**

Anaplasmosisis a disease of circulatory cells that might impose drastic effects on different blood parameters and thereby camel's health. The present study describes the hemato-biochemical changes in camels naturally infected with subclinical anaplasmosis. The study comprisedof2 groups *viz.*, group-I(15 *Anaplasma* positive camels identified by PCR)and group-II (15 healthy- PCR negative camels) which served as negative control. The student t-test as statistical tool revealed significant (p<0.05) decrease in hemogram [red blood cells (2.78±1.08), HGBconc. (7.41±2.78), HCT(11.22±3.52)] with accelerated (p<0.05)leukocytes count(20.29±6.08) in infected animals. Remarkable serum biochemical alterations were increased liver enzymes (GGT 18.0±1.0; LDH 68.67±27.024) with significantly low iron. The study concluded significant alterationsin blood parameters of camels naturally infected with subclinical anaplasmosis thatrequired further studies to be conducted in order to set a reference guide of blood parameters for the diagnosis of anaplasmosis in camels.
Keywords: Anaplasmosis, camel, diagnosis, hemato-biochemical parameters.

INTRODUCTION

Tick borne diseases (TBDs) are a great threat toproduction potential of livestock in Pakistan,as the agro-ecological and geo-climatic conditions of the country favors the growth and multiplication of different tick vectors (Karim *et al.,* 2017; Sudan *et al.*, 2014).Among TBDs, anaplasmosis is animportant disease of domestic ruminants, caused by*Anaplasma* spp.(intra-erythrocytic,pleomorphic bacteria)mainly includes*A. marginale, A. centrale, A.ovis* and *A. bovis*. Clinicalmanifestations of anaplasmosis in camels may include fever, anorexia, depression, lethargy, pale mucous membrane,varying degree of anemia, edema,lacrimation, diarrhea and abortion(Ismael *et al.*, 2016; Lbacha *et al.*, 2017). Camel mostly develops latent infection (Sudan *et al.,* 2014; Ghazvinian and Khodaiean, 2016) and serve as carrierof the disease for healthy stock. These sort of inapparent diseases in camels cause economic losses in terms of production, reproduction and traction power. Occurrence of tick vector, *Rhicephalusmicroplus* and *Hyaloma*spp have been reported in camels from different parts of the Pakistan (Karim *et al.*, 2017) which underscore the risk of anaplasmosis in camels.Virtually all studies onhemato-biochemical alterations in camel anaplasmosis are from Arabian and African countries.

Hematological and biochemical alterations are the indicators of disease severity and are considered to be the excellent tool for the diagnosis and prognosis of effective therapy (Col and Uslu, 2007).To the best of our knowledge, the information on hemato-biochemical parameters of anaplasmosis in camel is scanty. The present study wasconducted to assess the pattern of changes and relativevalue of hemato-biochemical parameters in camels naturally infected with subclinical anaplasmosis. This study may providea baseline data for further understanding on the pathogenesis of thesub-clinical anaplasmosis in camels.

MATERIALS AND METHODS

***Animals and diagnosis*:** In a survey of TBDs in province Punjab-Pakistan, 15 of 728 (2.06%) camelswere presumptively diagnosed to have *Anaplasma*in stained blood smears (Fig. 1). The infection was confirmed by PCR (Polymerase chain reaction) targeting 16SrRNA region(F, 5´-TACCTCTGTGTTGTAGCTAACGC-3´; R, 5´-CTTGCGACATTGCAACCTATTGT-3´) as described previously (Seong *et al.*, 2015). The size of amplified product was 429bp (Fig. 2). The DNA samples were prepared from ethylene diamine tetra acetic acid blood using commercially kit (Favorprep™ Blood genomic DNA extraction kit, Favorgen Biotech Corp, Taiwan). The samples were lysed and mixed with guanidine-isothiocyanate buffers. The lysates were passed through spin-columns, washed with the buffers, and subsequently pure DNA had eluted using 20 ul buffer. The PCR amplifications were carried out 20 uL volume with following conditions: 95˚C for 15 min; 40 cycles of 95˚C for 10 sec, 58˚C for 30 sec, and 72˚C for 30 sec; and final extension at 72˚C for 5 min. The positive and negative controls of *Anaplasma* were run in tandem.

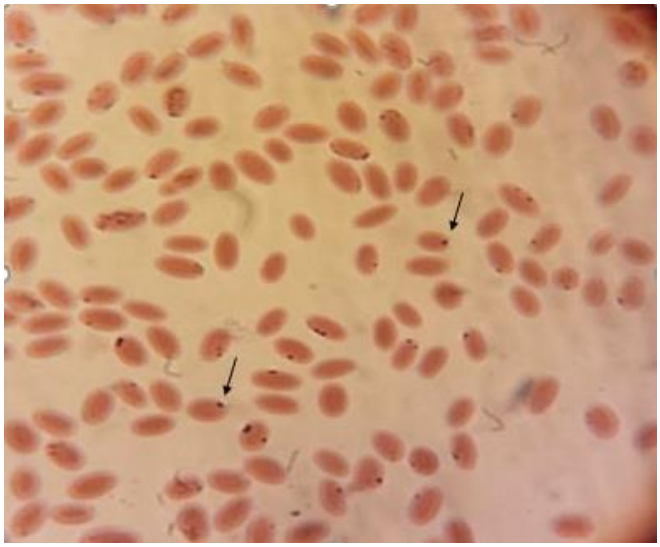

Figure1. Photomicrograph of Giemsastained thin blood smears,black arrows indicating*Anaplasma* sp.located inside the RBCs (Arrow) X100.

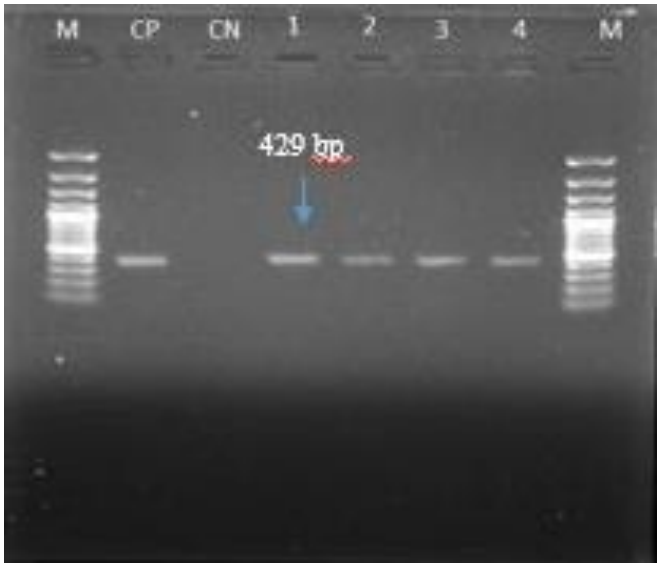

Figure2. Agarose gel electrophoresis of PCR products, M= Marker (100bp), CP= Control Positive, CN = Control Negative, 1 to 4 = Positive samples for *Anaplasma.*

Signalments and body conditions of *Anaplasma* positive camels have been shown in the Table 1. History of camels revealed gradual loss of body condition, decreased milk production and work insufficiency. As none of camels showed overt signs of clinical disease (fever, anorexia, pale mucous membrane, edema, lachrymationand diarrhea etc.),the infection was dubbed as subclinical anaplasmosis.

Table 1. Signalments and body condition of *Anaplasma* positive camels (n=15).

Signalment	Variables	Number
Sex	Male	6
Female	Female	9
Age	<3 years	2
	3.1-7 years	6
	7-10 years	3
Breeds	Marecha	8
	Beralla	7
Body Conditions	Good	1
	Moderate	12
	Poor	2

***Determination of hematological parameters*:** Red blood cells(RBCs), total leucocytes (TLC), Platelets count (PLT), hemoglobin (HGB) and packed cell volume (PCV) were determined by an electronic counter (Medonic, Sweden). Mean corpuscular volume (MCV), mean corpuscular hemoglobin (MCH) and meancorpuscular hemoglobin concentration (MCHC) were calculated according toformulae of Coles (Coles, 1986). Air-dried thin blood smears were fixed in methanol followed by Giemsa staining for differential TLC count and observed under light microscope.

***Biochemical assays and analysis*:** Serum biochemical analysis including total protein was measured by Biuret method, albumin by bromocresol green method (Tietz, 1995), urea and BUN by urease method (Burtis and Ashwood, 1994), total bilirubin by Jendrassik-Grof method (Jendrassik and Grof, 1938) and creatinine by Jaffemethod (Swanson *et al.*, 1993). The serum analyzed for gamma glutamyl transferase (GGT) by kinetic method based on the rate of 2-nitro-5-amino benzoic acid formation (Tietz, 1995). The levels of aspartate aminotransferase (AST) and alanine aminotransferase (ALT) were determined by colorimetric method of Kaplan (Kaplan, 1984), activities of lactate dehydrogenase (LDH) and alkaline phosphatase (ALP) were determined colorimetrically as described by Thomas (Thomas, 1998). Serum iron concentration was measured by chromazurol B (CAB) method (Garcic, 1979).

***Statistical analysis*:** Atotal of 15PCR positive samples and 15 control (PCR negative) samples for anaplasmosis were included for hemato-biochemical analyses. All results of hemato-biochemical parameters were represented as mean ± standard error of mean (SEM). The obtained data were analyzed using Student's t test at 5% probability ($p<0.05$) using SPSS statistical software version 22.

RESULTS

***Hematological findings*:** Camels infected with *Anaplasma* revealed a significant reduction ($p<0.05$) in RBC count (2.786±1.08), platelets counts (129.33±4.04), HGB conc. (7.41±2.78), HCT (11.225±3.52) and MCV (28.58±3.07), while significantly ($p<0.05$) increased WBC count (20.295±6.08), lymphocytes% (58.725±8.51) and MCHC (70.34±13.35) were recorded. Other selected hematological parameters showed non-significant ($p>0.05$) correlation with anaplasmosis (Table 2).

Table 2. Hematological parameters (Mean±SE) of infected (PCR Positive) andnon-infected (PCR negative) camels.

Parameters (Units)	Groups		*p*-value
	Non-infected	Infected	
WBC (10^9/L)	12.50±0.30	20.29±6.08	0.04
LYM (10^9/L)	7.50±1.00	7.56±6.30	0.98
MON (10^9/L)	0.63±0.21	0.69±0.25	0.69
GRA (10^9/L)	5.13±3.25	6.27±2.33	0.45
LYM (%)	44.01±1.72	58.72±8.51	0.00
MON (%)	3.93±1.72	5.06±1.79	0.32
GRA (%)	28.00±12.95	46.99±13.06	0.03
RBC (10^{12}/L)	8.33±2.08	2.78±1.08	0.00
HGB Conc. (g/dl)	14.36±0.60	7.41±2.78	0.00
HCT (%)	33.67±1.53	11.22±3.52	0.00
MCV (fl)	41.67±5.77	28.58±3.07	0.00
MCH (pg)	25.67±1.40	29.15±9.18	0.52
MCHC (gm/l)	56.03±2.11	70.34±13.35	0.08
RDW (%)	16.33±2.49	14.33±4.65	0.47
PLT (10^9/L)	160.05±26.88	129.33±4.04	0.00
MPV (fl)	5.27±0.06	4.83±0.77	0.34
PCT (%)	0.07±0.02	0.04±0.025	0.10
PWD (%)	11.03±0.71	8.97±2.71	0.21

**Significant (p<0.05);* WBCs: White blood cells, LYM: Lymphocytes, MON: Monocytes, GRA: Granulocytes, RBCs: Red blood cells, HGB: Hemoglobin concentration, HCT: Hematocrit, MCV: Mean corpuscular volume, MCH: Mean corpuscular hemoglobin, MCHC: Mean corpuscular hemoglobin concentration, RDW: Red distribution width, PLT: Platelets, MPV: Mean platelet volume, PCT: Plateletcrit, PDW: Platelet distribution widths

***Biochemical findings*:** A significant ($p<0.05$) reduction in the serum iron (58.7±4.1) was detected in the infected camels in comparison with the controls (Table 2). While significant ($p<0.05$) increase in the mean values of GGT (18.00±1.00), total bilirubin (0.4833±0.07024), protein (7.0333±0.32146), albumin (3.967±0.30551), globulin (3.3667±0.30551) and LDH (687.6667±27.024) was detected in infected camels. Further, it was found that anaplasmosis have non-significant ($p>0.05$) impact on urea, creatinine, ALT, AST, ALP (Table 3).

Table3. Biochemical parameters (Mean±SE) of infected (PCR positive) andnon-infected (PCR negative) camels.

Parameters	Groups		*p*-value
	Non-infected	Infected	
Protein (g/dl)	5.93±0.08	7.03±0.32	0.00
Albumin (g/dl)	3.36±0.09	3.96±0.30	0.03
Globulin (g/dl)	2.69±0.08	3.36±0.30	0.02
Ratio	1.25±0.05	1.18±0.12	0.42
GGT (µ/ml)	12.33±1.52	18.00±1.00	0.00
AST (µ/ml)	61.00±6.24	64.00±18.00	0.79
ALT (µ/ml)	10.43±0.40	15.66±3.51	0.06
ALP (µ/ml)	114.33±1.52	109.33±8.08	0.35
Total bilirubin mg/dl)	0.33±0.04	0.48±0.07	0.03
Direct bilirubin (mg/dl)	0.18±0.02	0.26±0.05	0.06
Indirect bilirubin (mg/dl)	0.16±0.02	0.22±0.02	0.03
LDH (µ/ml)	515.33±39.95	687.66±27.02	0.00
Creatinine (mg/dl)	1.14±0.21	1.34±0.04	0.17
Urea (mg/dl)	31.33±3.05	28.33±4.04	0.36
BUN (mg/dl)	14.60±1.37	13.80±2.38	0.64
Iron (µg/ dl)	113.67±12.89	58.67±4.50	0.00

**Significant (p<0.05);* GGT: gamma-glutamyl transferase, AST: aspartate aminotransferase, ALT: alanine aminotransferase, ALP: Alkaline Phosphatase, BUN: blood urea nitrogen, LDH: lactate dehydrogenase.

DISCUSSION

Most of the preceding studies on the hemato-biochemical findings of anaplasmosis are on cattle and other animals species, whereas, the information on camels is scanty. In the present study, hematological and biochemical parameters of the clinically healthy camels were in normal range, while significant ($p<0.05$)alterations were observed in infected camels which corroborate with findings of previous studies (Mal *et al.*, 2001; Ayoub *et al.*, 2003; Ghazvinian and Khodaiean, 2016).These alterations in various parameters might serve as indicators to diagnosis and to explore the severity of disease. In this study, significant ($p<0.05$) reduction was found in HGB conc., HCT and RBCs, reflecting microcytic normochromic type of anemia. The findings were similar to those of reported previously in camel anaplasmosis in different countries (Maghaddar, 2002; Mohammed *et al.*, 2007; Alsaad, 2009; Rabana *et al.*, 2011; Abdalla *et al.*, 2017; Azmat *et al.*, 2018). The reduction in HGB, HCT and RBCs were probably due to phagocytosis of erythrocytes by the reticuloendothelial system, short life span of erythrocytes and suppression of hemopoitic system (Mahran *et al.*, 2004; Mohammed *et al.*, 2007; Alsaad, 2009). Similar findings were reported by Egbu *et al.*, (2013), where a strong negative correlation was found between the level of infection and values of RBCs, HCT and HGB concentration. In the present study, significant decrease in platelets count was observed. Thrombocytopenia is well reported findings in infection with pathogens of *Anaplasmatace* (Li, *et al.*, 2015) that is

speculated due to the overproduction of WBCs in bone marrow that suppress the production of platelets in infected camels. The level of MCV was found significantly ($p<0.05$) lower in infected camels as compared to that of normal healthy animals. The lower MCV of infected camels vis-a-vis healthy ones may be ascribed to phagocytosis of erythrocytes (Aboaziza *et al.*, 2017). The similar findings were observed in Egypt and Iran (Derakhshanfar *et al.*, 2010; Aboaziza *et al.*, 2017).

The current study revealed a significant ($p<0.05$) increase in WBCs and LYM% that is in line with many other studies reported previously (Rezakhani *et al.*, 1997; Middleton, 1999; Alsaad, 2009; Ismael *et al.*, 2016). The increase in WBC may be due to stimulation of lymphoid tissue and stem cells in the bone marrow by the parasites and their toxins (Alsaad, 2009). The lymphocytosis was marked during the formation of antibodies in response to antigen and *Anaplasma* infection (Mahran *et al.*, 2004). Values of MCHC in the current study were observed significantly ($p<0.05$) higher in infected camels as compared to those of healthy camels that can be ascribe to the release of hemoglobin into plasma due to erythrolysis.

Hypoproteinemia is well known in animals anaplasmosis and development of edema due to low serum protein has been documented in sub-clinically *Candidatus A. camelii* infected camels (Lbacha *et al.*, 2017). Low total serum protein levels in sub-clinically infected camels of this study corroborate with earlier findings. The findings of high values of serum globulin in *Anaplasma* infected camels are congruent with those of previous studies conducted in Egypt (Aboaziza *et al.*, 2017). This might be due to hepatic degeneration and damages accompanied with hypoxia, or might be due to parasite antigens (Azza, 2008; Enwezorand Sackey, 2005).There was significant increase in mean values of GGT and LDH of infected camels. The elevated level of LDH is suggestive of damage to the myocytes, cardiomyocytes and hepatic tissues (Kataria and Bhatia, 1991). Slight hyper-bilirubinemia was found in this study that is in line with the findings published earlier (Omer *et al.*, 2003; Alsaad, 2009; Khan *et al.*, 2011; Agaar and Jassem, 2015). The increased bilirubin can be attributed to massive RBCs destruction and hepatocellular damage (Kataria and Bhatia, 1991; Qarawi, 1999). The serum iron level was found significantly lower in infected camels than those of healthy. It may be the outcome of massive engulfment of RBCs by reticuloendothelial system (RES) that ensures in compartmentation of iron in RES and a penultimate decrease in serum iron level. Furthermore, it is well established that in clinical or sub-clinical cases of anaplasmosis there is loss of appetite that also decreases the supply of iron to the body.

***Conclusion*:** Present study reveals that sub clinical anaplasmosis in camels lead to decreased hemogram and bring significant serum biochemical alterations. Sub-clinical anaplasmosis seriously affects the health of camels that could impose a socioeconomic burden especially on desert nomadic tribes in terms of production and work insufficiency. The results of hemato-biochemical parameters of camel anaplasmosis could serve as the guidelines for future studies in dromedaries under experimental and natural conditions in Pakistan and worldwide. Still more investigations are required to describe the worth of these selected parameters in anaplasmosis, and to explore new diagnostic tools for the management of this disease. Investigation of causative agent at Genera level is a limitation of this study that demands further studies on camel anaplasmosis at specie level.

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