DOI : 10.5958/2277-8934.2021.00024.2 *Vol 28 No 2, p 143-147*

ACID-BASE BALANCE, BLOOD GASES AND HAEMATOBIOCHEMICAL PROFILES IN CAMELS (*Camelus dromedarius*) WITH TRYPANOSOMOSIS

Mohamed Tharwat[1,2]

[1]Department of Veterinary Medicine, College of Agriculture and Veterinary Medicine, Qassim University, P.O. Box 6622, Buraidah, 51452, Saudi Arabia

[2]Department of Animal Medicine, Faculty of Veterinary Medicine, Zagazig University, 44519, Zagazig, Egypt

ABSTRACT

This study was carried out to investigate the status of acid-base balance and blood gases in camels with trypanosomosis compared to healthy camels. The haematobiochemical profiles were also reported in both groups. Forty-two camels with chronic weight loss, ventral oedema and ascites were examined. Passive haemagglutination test showed that 38 of the 42 camels (90%) were positive for *Trypanosoma evansi*. Compared to a value of 7.54±0.16 in healthy camels, the blood pH in diseased camels was 7.37±0.051. The partial pressure of carbon dioxide (PCO_2) was higher in camels with trypanosomosis than healthy camels. On the contrary, the oxygen partial pressure (PO_2) was lower in camels with trypanosomosis. The base excess (BE) was also lower in diseased camels than healthy ones. Similarly the bicarbonate (HCO_3) was lower in diseased camels. In a similar pattern, the total carbon dioxide (TCO_2) was lower in diseased than healthy group. The oxygen saturation (SO_2) decreased significantly in camels with trypanosomosis when compared to healthy group. Concerning the haematological parameters, leukocytosis, neutrophilia and lymphopenia was found in diseased camels. The RBCs count, haemoglobin and haematocrit decreased significantly in camels with trypanosomosis. Concerning, the biochemical parameters albumin and phosphorus decreased significantly and globulin and magnesium increased significantly in diseased camels. The serum activity of alkaline phosphatase, γ-glutamyl transferase and creatine kinase increased significantly in diseased camels compared to healthy camels. In conclusion, camels with trypanosomosis have metabolic acidosis, and the HCO_3 was lower than healthy camels. The PCO_2 was higher, while PO_2, BE, HCO_3, TCO_2 and SO_2 were lower in camels with trypanosomosis compared to healthy camels.

Key words: Acid-base balance, blood gases, camels, dromedary, trypanosomosis

Camel trypanosomosis (Surra) is caused by the protozoan parasite *Trypanosoma evansi* (*T. evansi*) and is a major threat to productivity and economic losses (Tehseen *et al,* 2015). Although surra is found in acute and chronic forms but chronic form is most common and is likely to present an association with secondary infection due to immuno-suppression caused by *T. evansi* infection (Olaho-Mukani *et al,* 1993; Olaho-Mukani and Mahamat, 2000; Ahmed, 2008; Eyob and Matios, 2013). The acid-base balance, blood gases and haemotobiochemical profiles may be altered in trypanosomosis in camels but these are least studied.

The effect of dehydration and exercise on the acid-base balance parameters has been investigated in dromedary camels (Abdoun *et al,* 2012; Okab *et al,* 2012). Another study has reported the influence of acid load with NH_4Cl on the acid-base status in young dromedary camels (Elkhair and Hartmann, 2010). In addition, the effect of tick infestation and stimulation by electroejaculation on the acid–base balance has also been reported in dromedary camels (Tharwat *et al,* 2014). Blood gas analysis has also been studied in healthy female dromedary camels, their calves and umbilical cord blood at spontaneous parturition (Tharwat, 2015). This study was carried out to determine acid-base balance, blood gases and haemotobiochemical status of camels with trypanosomosis.

Materials and Methods

Forty-two adult camels (*Camelus dromedarius*) were referred to the Veterinary Teaching Hospital, Qassim University, Saudi Arabia because of inappetance, loss of body condition, ventral or presternal oedema and abdominal distension. Duration of illness ranged from 3 days to 5 weeks. Clinical examination included general behaviour and condition, auscultation of the heart, lungs, rumen

SEND REPRINT REQUEST TO MOHAMED THARWAT email: mohamedtharwat129@gmail.com

and intestine, measurement of heart rate, respiratory rate and rectal temperature, swinging auscultation, percussion, auscultation of both sides of the abdomen and rectal examination (Wernery and Kaaden, 2002). Fifteen clinically healthy female camels were enrolled in this study as controls. From each camel, 10 mL blood sample were collected; 2 mL in EDTA tubes, 2 mL in heparinised tubes and the remaining 6 mL in plain tubes for serum harvesting. Using passive haemagglutination test, serum samples were tested for *T. evansi* antibodies (Omar *et al,* 1998). Sera from uninfected camel and an infected camel were used as negative and positive controls, respectively. Samples showing agglutination at 1:16 were considered positive.

Blood gas analyses and determination of haemato-biochemical parameters

The heparinised blood samples were used immediately to analyse the acid-base and blood gas parameters using a portable clinical veterinary analyser (I-STAT®, Abaxis, California, USA). In this way, blood pH, partial pressure of carbon dioxide (PCO_2), oxygen partial pressure (PO_2), bicarbonate (HCO_3), total carbon dioxide (TCO_2), base excess (BE), oxygen saturation (SO_2), and lactic acid (LA) were analysed immediately. A complete blood count [total and differential leukocytic count, erythrocyte count, haematocrit (HCT), haemoglobin, mean corpuscular volume (MCV), mean corpuscular haemoglobin (MCH) and mean corpuscular haemoglobin concentration (MCHC)] was carried out on the EDTA sample using the VetScan HM5, Abaxis, California, USA. An automated biochemical analyser (VetScan VS2, Abaxis, California, USA) was used to determine the serum concentrations of total protein, albumin, globulin, blood urea nitrogen (BUN), calcium, phosphorus, magnesium and cardiac biomarker troponin I (cTnI). The serum activity of γ-glutamyl transferase (GGT), aspartate aminotransferase (AST), creatine kinase (CK) and alkaline phosphatase (ALP) were also measured. Cardiac troponin I was measured in serum using a commercial available test (I-stat, cTnI, VetScan, Abaxis, CA, USA), using a two-site enzyme-linked immunosorbant assay (Tharwat, 2012; Tharwat, 2013; Tharwat *et al,* 2013a,b,c; Tharwat, 2020; Tharwat, 2121).

Statistical analysis

Data are presented as means ± SD and were analysed statistically using the SPSS statistical package, version 18, 2009. Student's t test was used for comparisons, and the significance was set at $P \leq 0.05$.

Results and Discussion

Definitive diagnosis of trypanosomosis in the infected camels was made on the basis of detecting antibodies in serum by passive haemagglutination test. The passive haemagglutination test showed that 38 of the 42 camels (90%) were positive for *T. evansi*. Hence, data of the 38 positive camels were used in this study. The most prominent clinical signs in the camels were weight loss, abdominal distension and ventral and subcutaneous presternal oedema. On clinical examination, ascites was detected by a fluid thrill on ballottment, by fluid sounds on percussion, or by the demonstration of excess fluid in the peritoneal cavity by abdominocentesis.

Compared to healthy control camels, the values of blood pH, PCO_2, PO_2, BE, HCO_3, TCO_2, SO_2 and LA means ± SD alongside the 25%, 50%, 75% and 99% percentiles in camels infected with trypanosomosis are summarised in Table 1. Compared to a value of 7.54±0.16 in healthy camels, the blood pH in diseased camels was 7.37±0.051, with a statistically significant difference (P=0.002). The PCO_2 was higher in camels infected with trypanosomosis than healthy camels (36.0±3.1 mmHg/L versus 30.0±8.1 mmHg, P=0.026). On the contrary, The PO_2 was lower in camels with trypanosomosis than healthy animals (25±2 mmHg/L versus 183±15 mmHg/L, P=0.0001). The BE was also lower in diseased camels than healthy ones (-4.3±2.3 mmol/L versus 2.4±5.3 mmol/L, P=0.0004). Similar, the HCO_3 was lower in diseased than healthy camels (20.9±1.6 mmol/L versus 24.9±2.9 mmol/L, P=0.0002). In a similar pattern, the TCO_2 was lower in diseased than healthy camels (22±1.8 mmol/L versus 25.7±2.9 mmol/L, P=0.022). The SO_2 decreased significantly in camels infected with trypanosomosis when compared to healthy control group (43±6.3 mmol/L in diseased camels versus 100 mmol/L in healthy camels, P=0.0001). In this study, camels with trypanosomosis had a significant decrease in the pH values compared to controls. This decrease could be easily justified by the increases in PCO_2 and additionally by the decreases in HCO_3 and BE values. The BE represents all basic components, not just HCO_3, and as such, is a more sensitive measure of metabolic acidosis than HCO_3 alone (Sigaard-Andersen and Fogh-Andersen, 1995). The decreased BE, HCO_3 and TCO_2 in this study could be explained as being due to the metabolic acidosis; this is the reason for the negative BE values. The LA concentration did not differ significantly between the 2 groups (1.46±1.4 mmol/L in diseased group versus 4.3±3.3 mmol/L in healthy group, P=0.11).

Table 1. Acid-base balance, blood gases and lactic acid concentration in camels with trypanosomosis versus healthy controls.

Parameters	Diseased camels (*n*=38)						Healthy camels (*n*=15)						*P* value
	Mean ± SD	Percentiles					Mean ± SD	Percentiles					
		25%	50%	75%	95%	99%		25%	50%	75%	95%	99%	
pH	7.37±0.051	7.35	7.38	7.41	7.43	7.44	7.54±0.16	7.42	7.50	7.70	7.80	7.80	0.002
PCO_2 mmHg	36.0±3.1	34.05	34.9	36.4	41.7	42.3	30.0±8.1	22.6	31.6	36.7	40.9	40.9	0.026
PO_2 mmHg	25±2	23.5	25.0	26.3	26.9	27.0	183±15	174	185	192	203.4	209.5	0.0001
BE mmol/L	-4.3±2.3	-6	-4	-2.5	-1	-1	2.4±5.3	0.0	1.0	7.5	10.0	10.0	0.0004
HCO_3 mmol/L	20.9±1.6	19.5	20.8	22	23.2	23.2	24.9±2.9	23.7	25.1	27.0	28.3	28.9	0.0002
TCO_2 mmol/L	22±1.8	20.8	22.0	23.3	23.9	24.0	25.7±2.9	24.8	26.0	27.3	29.1	29.8	0.022
SO2 %	43±6.3	40	41	44	50	52	100	100	100	100	100	100	0.0001
LA mmol/L	1.46±1.4	0.7	0.9	1.7	3.1	3.4	4.3±3.3	2.3	3.0	5.4	11.7	11.7	0.11

PCO_2, partial pressure of carbon dioxide; PO_2, partial pressure of oxygen; BE, base excess; HCO_3, bicarbonate; TCO_2, total carbon dioxide; SO_2, oxygen saturation; LA, lactic acid.

Table 2. Haematological parameters in camels with trypanosomosis versus healthy controls.

Parameters	Diseased camels (*n*=38)						Healthy camels (*n*=15)						*P* value
	Mean ± SD	Percentiles					Mean ± SD	Percentiles					
		25%	50%	75%	95%	99%		25%	50s%	75%	95%	99%	
WBCs ($\times10^9$/L)	30.4±20.2	15.8	23.5	37.1	62.8	69.1	16.8±3.9	15.7	17.9	18.6	21.3	22.3	0.007
LYM ($\times10^9$/L)	1.4±0.8	0.8	1.1	1.9	2.5	2.7	6.2±2.9	4.4	5.9	6.6	11.1	12.9	0.0001
NEU ($\times10^9$/L)	27.8±19.6	14.7	20.8	33.8	59.1	67.3	9.7±3.0	7.6	9.8	12.0	13.8	14.3	0.0003
RBCs ($\times10^{12}$/L)	7.5±1.0	6.9	7.4	7.8	8.9	9.2	11.3±1.4	10.4	11.5	12.0	13.5	13.6	0.0001
HB (g/dL)	11.5±1.3	10.6	12.1	12.2	12.9	13.1	16.4±2.8	14.6	16.0	18.0	21.0	23.0	0.0001
HCT (%)	20.4±2.0	19.1	20.3	21.2	23.3	24.2	28.9±2.7	27.4	29.0	30.5	33.0	33.2	0.0001
MCV (fl)	27.4±2.4	26.0	26.5	27.3	31.3	32.7	25.5±1.5	24.0	26.0	26.0	27.1	27.8	0.014
MCH (pg)	15.5±1.4	14.7	15.8	16.4	17.3	17.5	14.7±2.4	12.7	13.9	16.7	18.7	19.7	0.347
MCHC (g/dL)	56.8±7.2	51.0	57.4	62.3	65.2	66.0	57.6±9.0	50.6	53.7	64.3	74.3	74.9	0.829

WBCs, white blood cells; LYM, lymphocytes; MON, monocytes; NEU, neutrophils; RBCs, red blood cells; HB, haemoglobin; HCT, haematocrit; MCV, Mean corpuscular volume; MCH, Mean corpuscular haemoglobin; MCHC, Mean corpuscular haemoglobin concentration

The means ± SD of the haematological parameters in camels infected with trypanosomosis compared to healthy camels alongside the 25%, 50%, 75% and 99% percentiles are presented in Table 2. Leukocytosis was found in diseased camels compared to healthy group (30.4±20.2 $\times10^9$/L in diseased group versus 16.8±3.9 $\times10^9$/L in healthy group, P=0.007). Similar, neutrophilia was recorded in diseased group compared to healthy group (27.8±19.6 $\times10^9$/L in diseased group versus 9.7±3.0 $\times10^9$/L in healthy group, P=0.0003). However, lymphopenia was detected in diseased camels compared to healthy ones (1.4±0.8 $\times10^9$/L in diseased group versus 6.2±2.9 $\times10^9$/L in healthy group, P=0.0001). The RBCs count, haemoglobin concentration and HCT per cent decreased significantly in camels infected with trypanosomosis when compared to healthy camels (P=0.0001). Similar findings were reported (Ahmadi-hamedani *et al*, 2014; Hussain *et al*, 2018). The MCV increased significantly in diseased camels compared to healthy camels (P=0.014). The MCH and MCHC increased in diseased group compared to healthy group but the increases were not significant (P=0.34 and P=0.82, respectively). Leukocytosis encountered in this study could be explained on the basis of the chronic nature of the disease. This denotes that camels with trypanosomosis may develop concurrent and even fatal bacterial, viral and other protozoan infections as a result of immunosuppression (Aradaib and Majid, 2006). Haematological indices showed significant reduction in the haematocrit and hemoglobin indicated that affected camels had macrocytic hypochromic anaemia in an agreement to a study reported recently (Saleh *et al*, 2009).

The means ± SD of the biochemical parameters in camels infected with trypanosomosis compared to

Table 3. Biochemical parameters in camels with trypanosomosis versus healthy controls.

Parameters	Diseased camels (*n*=38)						Healthy camels (*n*=15)						*P* value
	Mean ± SD	Percentiles					Mean ± SD	Percentiles					
		25%	50%	75%	95%	99%		25%	50s%	75%	95%	99%	
TP (G/L)	70.3±9.7	65.0	70.0	75.3	80.7	81.7	67.3±4.3	63.0	67.5	68.8	74.0	76.4	0.34
ALB (G/L)	39.5±6.4	39.0	42.0	42.5	43.7	43.9	60.39±3.0	60.8	61.5	62.0	64.3	64.9	0.0001
ALP (U/L)	94.5±90.5	35.8	62.0	120.7	204.2	220.8	6.6±2.8	5.8	6.5	8.0	10.8	12.6	0.0004
AST (U/L)	101.8±46.4	79.3	92.5	115.0	155.8	164.0	79.5±16.5	69.5	80.5	85.0	104.8	118.6	0.117
CA (MMOL/L)	2.3±0.1	2.3	2.3	2.4	2.4	2.4	2.4±0.1	2.3	2.4	2.5	2.6	2.6	0.18
GGT (U/L)	63.0±104.7	9.3	12.5	66.3	189.3	213.9	12.2±5.3	8.8	12.5	13.0	19.8	26.4	0.04
GLOB (G/L)	31.0±10.6	26.0	33.0	38.0	40.4	40.9	7.0±3.8	5.0	7.0	9.0	12.5	15.3	0.0001
BUN (MMOL/L)	9.8±9.4	5.1	6.4	11.1	21.2	24.0	6.4±1.1	5.9	6.4	6.7	8.1	8.2	0.13
CK (U/L)	240.0±92.0	183.2	253.0	309.3	321.9	324.4	139.0±21.6	127.0	136.0	148.8	171.8	178.4	0.01
PHOS (MMOL/L)	2.0±0.4	1.9	2.0	2.2	2.5	2.6	2.6±0.4	2.6	2.7	2.8	3.0	3.1	0.009
MG (MMOL/L)	0.7±0.1	0.7	0.7	0.7	0.8	0.8	0.3±0.0	0.2	0.3	0.3	0.3	0.3	0.0001
cTnI (ng/mL)	0.03±0.06	0.00	0.01	0.03	0.12	0.14	0.03±0.02	0.02	0.02	0.04	0.07	0.08	0.93

TP, total protein; ALB, albumin; ALP, alkaline phosphatase; AST, aspartate aminotransferase; CA, calcium; GGT, γ-glutamyl transferase; GLOB, globulin; BUN, blood urea nitrogen; CK, creatine kinase; PHOS, phosphorus; MG, magnesium; cTnI, cardiac troponin I.

healthy camels alongside the 25%, 50%, 75% and 99% percentiles are presented in Table 3. Compared to healthy camels, the serum concentration of albumin decreased significantly in diseased camels (39.5±6.4 g/L in diseased group versus 60.39±3.0 g/L in healthy group, P=0.0001). Similar findings were reported (Ahmadi-hamedani *et al,* 2014; Hussain *et al,* 2018). Similar, the serum concentration of phosphorus decreased significantly in diseased camels (2.0±0.4 mmol/L in diseased group versus 2.6±0.4 mmol/L in healthy group, P=0.009). On the contrary, the serum concentration of globulin increased significantly in diseased camels (31.0±10.6 g/L in diseased group versus 7.0±3.8 g/L in healthy group, P=0.0001). Similar, the serum concentration of magnesium increased significantly in diseased camels (0.7±0.1 mmol/L in diseased group versus 0.3±0.0 mmol/L in healthy group, P=0.0001). The serum activity of ALP, GGT and CK increased significantly in diseased camels compared to healthy animals (P=0.0004, P=0.04 and P=0.01, respectively). Other biochemical parameters that included the serum concentrations of calcium, BUN, phosphorus, cTnI, and the serum activity of AST did not differ significantly compared to healthy camels (P=0.18, P=0.13, 0.93 and P=0.117, respectively). Hyperglobulinaemia encountered in this study could be explained on the basis of the chronic nature of the disease. This denotes that camels with trypanosomosis may develop concurrent and even fatal bacterial, viral and other protozoan infections as a result of immunosuppression (Aradaib and Majid, 2006).

It is concluded from this study that camels with trypanosomosis has metabolic acidosis when compared to healthy non-infected animals. The HCO_3 concentration was lower in camels with trypanosomosis than healthy camels. Changes in blood gases were remarkable where the PCO_2 was higher, while PO_2, BE, HCO_3, TCO_2 and SO_2 were lower in camels with trypanosomosis when compared to healthy camels.

References

Abdoun KA, Samara EM, Okab AB and Al-Haidary AA (2012). State of Acid-base Balance in Dehydrated Camels (*Camelus dromedarius*). Asian Journal of Animal and Veterinary Advances 7:420-426.

Ahmadi-hamedani M, Ghazvinian K and Darvishi MM (2014). Haematological and serum biochemical aspects associated with a camel (*Camelus dromedarius*) naturally infected by *Trypanosoma evansi* with severe parasitemia in Semnan, Iran. Asian Pacific Journal of Tropical Biomedicine 4:743-745.

Ahmed A (2008). Epidemiological studies (parasitological, serological and molecular techniques) of *T. evansi* infection in camels in Egypt. Veterinary World Journal 1:325-328.

Aradaib IE and Majid AA (2006). A simple and rapid method for detection of *Trypanosoma evansi* in the dromedary camel using a nested polymerase chain reaction. Kinetoplastid Biology and Disease 5:2.

Elkhair N and Hartmann N (2010). Studies on acid-base

status (Stewart's model) of young camels (*Camelus dromedarius*) after acid-load with NH4CI. Berliner und Münchener Tierärztliche Wochenschrift 123:153-159.

Eyob E and Matios L (2013). Review on camel trypanosomosis (surra) due to *Trypanosoma evansi*: Epidemiology and host response. Journal of Veterinary Medicine and Animal Health 5:334-343.

Hussain R, Khan A, Jahanzaib, Qayyum A, Abbas T, Ahmad M, Mohiuddin M and Mehmood K (2018). Clinico-Haematological and oxidative stress status in Nili Ravi buffaloes infected with *Trypanosoma evansi*. Microbial Pathogenesis 123:126-131.

Okab AB, Abdoun KA, Samara EM and Al-Haidary AA (2012). Acid-Base Balance in Camels (*Camelus dromedarius*): Effects of Exercise and Dehydration. The FASEB Journal. Experimental Biology Meeting Abstracts. First published: 01 April 2012 https://doi.org/10.1096/fasebj.26.1_supplement.lb739

Olaho-Mukani W and Mahamat H (2000). Trypanosomosis in the dromedary camel. In: Gahlot, T.K. (Ed.), Selected Topics on Camelids. The Camelid Publishers, Chandan Sagar Well, Bikaner, India. pp 255-270.

Olaho-Mukani W, Munyua WK, Mutugi MW and Njogu AR (1993). Comparison of antibody and antigen selection enzyme immunoassays to the diagnosis of *Trypanosoma evansi* infections in camels. Veterinary Parasitology 45:231-240.

Omer OH, Magzoub M, Haroun EM, Mahmoued OM and Abdel-hamed YM (1998). Diagnosis of *Trypanosoma evansi* in Saudi Arabian camels (*Camelus dromedarius*) by the passive haemagglutination test and Ag-Elisa. Journal of Veterinary Medicine Series B 45:627-633.

Saleh MA, Al-Salahy MB and Sanousi SA (2009). Oxidative stress in blood of camels (*Camelus dromedarius*) naturally infected with *Trypanosoma evansi*. Veterinary Parasitology 162:192–199.

SPSS (2009). Statistical Package for Social Sciences, SPSS Inc., Chicago, IL, USA Copyright© for Windows, version 16.0.

Tehseen S, Jahan N, Qamar MF, Desquesnes M, Shahzad MI, Deborggraeve S and Büscher P (2015). Parasitological, serological and molecular survey of *Trypanosoma evansi* infection in dromedary camels from Cholistan Desert, Pakistan. Parasites and Vectors 8:415.

Tharwat M (2012). The cardiac biomarker troponin I and other Haematological and biochemical variables in downer camels (*Camelus dromedarius*). Journal of Camel Practice and Research 19:123-128.

Tharwat M (2013). Ultrasonographic findings in camels (*Camelus dromedarius*) with trypanosomosis. Journal of Camel Practice and Research 20:283-287.

Tharwat M (2015). Haematology, biochemistry and blood gas analysis in healthy female dromedary camels, their calves and umbilical cord blood at spontaneous parturition. Journal of Camel Practice and Research 22:239-245.

Tharwat M (2020). The cardiac biomarkers troponin I and creatine kinase myocardial band in camels (*Camelus dromedarius*) - review. Journal of Camel Practice and Research 27:121-128.

Tharwat M, Ali A, Al-Sobayil F, Derar R and Al-Hawas A (2014). Influence of stimulation by electroejaculation on myocardial function, acid-base and electrolyte status and haematobiochemical profiles in male dromedary camels. Theriogenology 82:800-806.

Tharwat M, Al-Sobayil F and Ahmed AF (2013a). Effect of isoflurane and halothane on myocardial function in healthy dromedary camels as assessed by cardiac troponin I. Journal of Camel Practice and Research 20:289-294.

Tharwat M, Al-Sobayil F and Buczinski S (2013b). Cardiac biomarkers changes in camels (*Camelus dromedarius*) secondary to long road transportation. Journal of Veterinary Cardiology 15:15-22.

Tharwat M, Al-Sobayil F and Buczinski S (2013c). Effect of racing on the serum concentrations of cardiac troponin I and CK-MB in racing camels (*Camelus dromedarius*). Veterinary Research Communications 37:139-144.

Tharwat M (2021). Influence of 8 km training on cardiac biomarkers alongside hematobiochemical profiles in race camels. Journal of Camel Practice and Research. 28(1):79-84.

Wernery U and Kaaden O (2002). Protozoal Infections. In: Infectious Diseases in Camelids. Blackwell Science Berlin, Vienna; 2nd ed. pp 272-311.