

## SERUM BIOCHEMISTRY OF CAMELS (*CAMELUS DROMEDARIUS*) EXPERIMENTALLY INFECTED WITH *SARCOCYSTIS CAMELI*

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### SUMMARY

Inoculation of two camel calves with low dose ( $25 \times 10^4$ ) and high dose ( $75 \times 10^4$ ) of *Sarcocystis cameli* sporocysts obtained from dogs, produced increase activity of aspartate aminotransferase, creatine phosphokinase, lactate dehydrogenase, fructose 1,6 diphosphate aldolase and glucose-6-phosphate dehydrogenase and decrease in total protein and glucose compared to values of uninfected control. Where, the concentrations of triglycerides, phospholipids, total cholesterol and alanine aminotransferase showed no marked changes.

**Key words:** Camel, *Sarcocystis*, serum biochemistry.

### INTRODUCTION

*Sarcocystis* is one of the most prevalent protozoon parasite of the skeletal and cardiac muscles of mammals, birds and reptiles.

*Sarcocystis cameli* is a common parasite of camels (*Camelus dromedarius*) in Saudi Arabia (Hussein, 1991 and Fatani et al. 1996) and different countries of the Middle East, where camels are raised. The parasite causes severe clinical signs include anorexia, inappetence, marked loss of weight and dehydration (Fatani et al. 1996). The aim of the present investigation was conducted to study the serum biochemistry in camel calves experimentally inoculated with *S. cameli* obtained from dogs.

### MATERIAL AND METHODS

Two coccidia free (6-8 weeks of age) conventionally reared dogs were fed each 500 gm of *Sarcocystis cameli* infected musculature of camel. The dogs were raised on milk and bread to preclude infection with any coccidian parasite. Faecal samples of each dog were examined daily; until the 7th day post excretion of *S.cameli* sporocysts when the two dogs were euthanatised. The sporocysts were

obtained from the dog's intestine according to the method described by Dubey et al. (1989). They were suspended in sterile distilled water containing 1000 I.U. penicillin, 100 ug streptomycin and 500 units mycostatin per ml. The number of sporocysts per ml was counted as using haematocytometer.

Three camel calves obtained immediately after birth from a local camel breeding farm in Al-Ahsa area, Saudi Arabia were housed indoors with their dams in the absence of carnivores for 6 months period. The calves were divided into two groups, Group I and Group II. The two camels of Group I ( $C_1$  and  $C_2$ ) were inoculated with  $25 \times 10^4$  and  $75 \times 10^4$  sporocysts through the rumen respectively, while the third camel ( $C_3$ ) was kept as healthy uninfected control. Serum biochemistry of all these camels were studied at weekly intervals for a period of 4 weeks post-inoculation.

Blood samples were allowed to clot. Serum was separated and stored at  $-20^\circ\text{C}$  until analyzed for activities of aspartate amino transferase (AST), alanine aminotransferase (ALT) by the method of Reitman and Frankel (1957). Creatine phosphokinase (CK) by the method of Hughes (1962). Lactic dehydrogenase (LD), fructose 1,6-diphosphate and aldolase (FDA) and glucose-6-phosphate dehydrogenase (G6PD) were assayed by the method of King (1965). The

glucose content was estimated by the method of Dubowski (1962) and the concentration of total cholesterol, phospholipids and triglyceride in serum were determined by the method of Zlatkis et al (1969) and serum total protein concentration by biuret method.

Mean values for infected and control camels were compared statistically by the paired (t) test. Values of  $P < 0.05$  were considered to be significant deviation from the null hypothesis.

## RESULTS

The results of inoculation of *Sarcosystis cameli* sporocysts into camel calves ( $C_1$ ,  $C_2$ ) on serum biochemistry are shown in Table 1. The infection produced significantly ( $P < 0.001$ ) increased activity of AST (177%), CK (138%), LD (33%), FDA (88%) and G6PD (71%) compared to values of uninfected control ( $C_3$ ). Conversely, a significant ( $P < 0.01$ ) decrease in total protein (35%) and glucose (33%) occurred in infected calves ( $C_1$ ,  $C_2$ ) compared to uninfected control ( $C_3$ ). The concentrations of triglycerides, phospholipids, total cholesterol as ALT in infected calves ( $C_1$ ,  $C_2$ ) showed no marked changes during the experimental period. The higher dose of the inoculum produced higher effects than the lower dose though not significantly different ( $P > 0.1$ ).

Table (1): Mean ( $\pm$  SD) peripheral concentrations of some constituents and enzymes in serum of experimentally inoculated camels with *Sarcocystis camelii*.

Constituents	Uninfected control	Enteroxin Production											
		Week 1		Week 2		Week 3		Week 4					
		LD	HD	LD	HD	LD	HD	LD	HD				
Total protein (g/l)	56 $\pm$ 2.3	640 $\pm$ 2.1**	39.6 $\pm$ 2.1**	36.6 $\pm$ 1.4*	33 $\pm$ 2*	34 $\pm$ 1.6*	37 $\pm$ 1.5*	33 $\pm$ 2.1*					
Glucose (m mol/l)	4.3 $\pm$ 0.2	4.3 $\pm$ 0.2	4.3 $\pm$ 0.2	3.2 $\pm$ 0.2**	2.9 $\pm$ 0.2**	2.8 $\pm$ 0.2**	2.8 $\pm$ 0.2**	2.6 $\pm$ 0.15**					
Triglyceride (m mol/l)	0.37 $\pm$ 0.0	0.36 $\pm$ 0.03	0.36 $\pm$ 0.03	0.36 $\pm$ 0.03	0.36 $\pm$ 0.03	0.38 $\pm$ 0.03	0.34 $\pm$ 0.03	0.35 $\pm$ 0.03					
Phospholipids (m mol/l)	0.45 $\pm$ 0.04	0.42 $\pm$ 0.04	0.42 $\pm$ 0.04	0.41 $\pm$ 0.05	0.43 $\pm$ 0.03	0.39 $\pm$ 0.04	0.44 $\pm$ 0.04	0.45 $\pm$ 0.04					
Total cholesterol (m mol/l)	2.5 $\pm$ 0.31	2.5 $\pm$ 0.31	2.4 $\pm$ 0.32	2.3 $\pm$ 0.31	2.2 $\pm$ 0.32	1.9 $\pm$ 0.32	---	2.0 $\pm$ 0.31					
<b>Enzyme activity (U/L)</b>													
Aspartate aminotransferase	1.81 $\pm$ 2.1	1.81 $\pm$ 2.3	22.3 $\pm$ 3.1	56 $\pm$ 4.6*	52 $\pm$ 4.3*	68 $\pm$ 4.5*	51 $\pm$ 4.3*	85 $\pm$ 4.4*					
Alanine aminotransferase	5.1 $\pm$ 0.6	5.2 $\pm$ 0.6	---	6.1 $\pm$ 0.6	6.2 $\pm$ 0.6	---	6.0 $\pm$ 0.5	6.3 $\pm$ 0.5					
Creatine kinase	42.2 $\pm$ 4.6	45.3 $\pm$ 4.2	80.3 $\pm$ 7.4*	110 $\pm$ 8.6*	105 $\pm$ 8*	115 $\pm$ 8*	96.3 $\pm$ 7.3*	---					
Lactate dehydrogenase	395.6 $\pm$ 16.3	403 $\pm$ 17	424 $\pm$ 17.3	---	525 $\pm$ 16**	560 $\pm$ 18**	496 $\pm$ 15**	530 $\pm$ 17**					
Fructose 1,6-diphosphate aldolase	3.6 $\pm$ 0.21	3.9 $\pm$ 0.2	4.1 $\pm$ 0.2	7.4 $\pm$ 0.3**	6.7 $\pm$ 0.25**	7.9 $\pm$ 0.31**	7.3 $\pm$ 0.4**	7.6 $\pm$ 0.3**					
Glucose-6-phosphate dehydrogenase	4.2 $\pm$ 0.3	4.6 $\pm$ 0.32	---	7.8 $\pm$ 0.32**	7.3 $\pm$ 0.33**	8.2 $\pm$ 0.32**	7.8 $\pm$ 0.33**	8.1 $\pm$ 0.35**					

LD= Low dose of parasite inoculum; HD=High dose of parasite inoculum; - Denotes constituent not estimated in those samples  
\*P < 0.001, \*\*= P < 0.05.

## DISCUSSION

Significant elevation of activities of AST, LD and CK imply liver and muscle injury infected by the parasite. Similar observations have been demonstrated in calves (Mahrt and Fayer, 1975; Prasse and Fayer, 1981; and Dossouky et al. 1984). Signs of degeneration in the liver of calves and goats infected with *Sarcocystis* (Collins et al., 1980 and Hilali and Nassar 1983) will further substantiate such observation that the liver may be involved. Indeed, the increased activity of CK, a muscle specific enzyme has been elevated in this study suggesting that one of the predilection sites of *Sarcocystis* may be the muscle (Warrag, 1981 and Hussein, 1991). The activity of the enzyme also increased in bovine sarcocystosis (Fayer and Lynch, 1979 and Prasse and Fayer, 1981) muscle trauma, muscle ischaemia, nutritional myopathies (Sreekumar and Nirmalan, 1992 and Aktas et al., 1993) at and after localized intramuscular injection (Lewis and Rhodes, 1978).

The parallel increase in CK, FDA and G6PD activity in serum during *Sarcocystis* infection may be due to efflux of enzymes from parasitized muscle tissue as from repair processes or from parasite derived enzymes (Dauguschies et al., 1990). The increase in activity of FDA, D6PD and decrease in glucose concentration, and lacking effects of the parasite on total cholesterol, phospholipids and triglyceride, suggest that muscular glycolysis and energy metabolism but not lipid metabolism, were adversely affected by the parasite (Dauguschies et al. 1990 and Prickett et al., 1992). The decrease in total protein,

however, may result from anorexia and inappetence shown by the infected animals (Fatani et al. 1996).

## REFERENCES

- Aktas, M., Auguste De, Lefebvre, H.P., Toutain, P.L. and Braun, J.P. (1993): Creatine kinase in the dog: review. *Veterinary Research Communications*, 17: 353-369.
- Collins, G.H., Sutton, R.H. and Charleston, W.A.G. (1980): Studies in *Sarcocystis* species. V.A. species infecting dogs and goats; observations on the pathology and serology of experimental sarcocystosis in goats. *New Zealand Veterinary Journal*, 28: 156-158.
- Dauguschies, A. Hasche, A. and Rommel, M. (1990): Activities of selected enzymes in the blood plasma and muscles of *Sarcocystis*-infected pigs. *Mitteilungen der Osterreichischen Gesellschaft fur Tropenmedizin und Parasitologie*, 12: 131-139.
- Dossouky, M.I., Mohamed A.H., Nassar, A.M. and Hilali, M. (1984): Haematological and biochemical changes in buffalo calves inoculated with *Sarcocystis fusiformis* from cats. *Veterinary Parasitology*, 14: 1-6.
- Dobowski, K.M. (1962): An o-toluidine method for body fluid glucose determination. *Clinical Chemistry*, 8: 215-235.
- Dubey, J.P., Speer, C.A. and Fayer, R. (1989): *Sarcocystosis of Animals*. CRC Press, P: 215.
- Fatani, A., Hilali, M., Al-Atiya, S. and Al-Shami, S. (1996): Prevalence of *Sarcocystis* in camels (*Camelus dromedarius*) from Al-Ahsa, Saudi Arabia. *Veterinary Parasitology*, 62: 241-245.
- Fatani, A., El-Sebaie, A. and Hilali, M. (1996): Clinical and haematobiochemical changes in camels (*Camelus dromedarius*) experimentally inoculated with *Sarcocystis cameli*. *Journal of Camel Practice and Research*, 3 (1): 11-15.

- Fayer, R. and Lynch, G.P. (1979): Pathophysiological changes in urine and blood from calves experimentally infected with *Sarcocystis cruzi*. *Parasitology*, 79: 325-357.
- Fayer, R. and Prasse, K.W. (1981): Experimental acute *Sarcocystis bovicanis* infection in calves . I. Cellular and serologic changes. *Veterinary Pathology*, 18: 351-357.
- Hilali, M. and Nassar, A.M. (1983): Development of *Sarcocystis fusiformis* (Railliet, 1987) in experimentally infected buffalo calves (*Bubalus bubalis*). 11th Scandinavian Symposium of Parasitology, Hasseludden, Sweden, 17-19, August..
- Hughes, B.P. (1962): A method for the estimation of serum creatine kinase and its use in comparing kinase and aldolase activity in normal and pathological sera. *Clinica Chemica Acta*, 7: 597-598.
- Hussein, S.H. (1991): The prevalence of sarcocyst is infections in Saudi Arabian Najdi sheep and camels. *Biological Science*, 1: 34-56.
- King, J. (1965): *Practical Clinical Enzymology*. Van Nostrand, London.
- Lewis , H.B. and Rhodes, D.C. (1978): Effects of I.M. infections on serum creatine phosphokinase (CPK) values in dogs. *Veterinary Clinical Pathology*, 7: 11-12.
- Mahrt, J.L. and Fayer, R. (1975): Hematologic and serologic changes in calves experimentally infected with *Sarcocystis fusiformis*. *Journal of Parasitology*, 61: 967-969.
- Prasse, K.W. and Fayer, R. (1981): Hematology of experimentally acute *Sarcocystis bovinis* infection in calves. II. Serum biochemistry and hemostasis studies. *Veterinary Pathology*, 18: 358-367.
- Prickett, M.D., Prestwood, A.K. and Hoeing, M. (1992): Lipid metabolism and *Sarcocystis miescheriana* infection in growing swine. *Veterinary Parasitology*, 42 (1-2): 41-51.
- Reitman, S. and Frankel (1957): A calorimetric method for the determination of serum glutamic -oxalacetic- acid and glutamic -pyruvic transaminase. *American Journal of Clinical Pathology*, 28:56-63.
- Sreekumar, K.P. and Nirmalan, G. (1992): Normal values for certain serum enzymes of clinical value in Indian elephants. *Veterinary Research Communications*, 16: 411-414.
- Warrg, M. (1981): Studies on *Sarcocystis* and other cyst-forming Coccidia in the Sudan. M.VSc. Thesis, University of Khartoum, p:100.
- Zlatkis, A. Zak, B. and Boyle, G.J. (1969): Study of a new cholesterol reagent *Analytical biochemistry*, 29: 143-144.