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CLINICAL AND BIOCHEMICAL STUDIES ON MICROFILARIA AND TRYPANOSOMA INFECTED CAMELS

(With 2 Table)

By

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دراسات كيميائيه واكلينيكيه عن الجمال المصابه بالميكروفيلاريا والتربيانوسوما

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أجريت هذه الدراسة للتعرف على بعض الجوانب الكيميائيه والاكلينيكيه فى الجمال المصابه بالفلاريا والتربيانوسوما فى محافظة قنا ذات الطبيعه المناخيه الخاصه وتم القيام ببعض العلاجات باستخدام الايفرمكتين للفلاريا والترباميديم للتربيانوسوما وسجلت نتائج تأثيرها مع ذكر التغيرات التى حدثت فى نسب بعض العناصر والبروتين الكلى والتحليل الكهربى لبروتينات المصل.

MICROFILARIA, TRYPANOSOMA & CAMELS

SUMMARY

Alterations induced by microfilaria and trypanosoma migration in tissues and organs of camels are reflected on the clinical and biochemical aspects of ten sick animals suffering from both parasites. The main changes observed in the respective camels were emaciation, pale and icteric mucous membranes, high body temperature, rigid of spermatic cord blood vessels, dry skin and subcutaneous oedema on limbs and abdomen. There was also a significant drop in serum total protein, calcium and inorganic phosphorus. Diagnosis and treatment were conducted in this work and the data obtained from diseased cases were statistically analysed against apparently healthy ones, tabulated and discussed.

INTRODUCTION

Filariasis and trypanosomiasis are serious tropical diseases affecting domestic animals especially camels. Their clinical and biochemical aspects are of veterinary importance in diagnosis, prognosis and treatment. Filariasis in camel is enzootic and common in most tropical countries. Microfilaria can causes a variety of clinical syndromes characterized by localized skin lesions, severe weakness, emaciation, high body temperature and swollen of both scrotum and testis (*LOSOS, 1986; ABU-EL-MAGD* et al., *1988 and KARRAM* et al., *1991*). Trypanosomiasis is a chronic infection of camel exhibiting definite signs of debility, emaciation, intermittent fever and facial oedema (*RAISINGHANI* et al., *1980 and NASSER, 1992*).

Several studies on blood biochemistry of camels infected with microfilaria and trypanosma species, revealed that these two infections might produce disorders in blood parameters. This is due in the view of many authers, to morphological changes in the respective organs together with resultant malfunctions (*ABOUL-ELA,* et al., *1986; GAD-EL-MOULA* et al., *1987; ABU-EL-MAGD* et al., *1988; ANOSA , 1988 and NASSER, 1992*).

This investigation was conducted to study clinical and biochemical aspects of filariasis and trypanosomiasis in camels and to evaluate the use of these examinations as a possible aid in diagnosis for such infection.

MATERIAL and METHODS

Seventeen dromedary camels (7-12 years old) at Qena Governorate were used in this work. These included 7 apparently

healthy, 5 *Dipetalonema evansi* infected and 5 *Trypanosoma evansi* infected camls. All camels were clinically examined and blood samples were collected twice, i.e. before treatment and 30 days after. The history of these animals indicated that they were fed on Trifolium alexandrium and some concentrates.

Detection of microfilaria was carried out by using thick blood films prepared and stained according to *HELMY et al. (1967)*, while indentification of trypanosoma was done by using thick and thin blood smears as described by *SHUTE (1966)*.

Microfilaria infected camels were treated with Ivermectin (Ivomec/MSD). A single dose of 1.0 ml/50 Kg B.W. was given subcutaneously in the neck region. Trypanosoma infected camels were treated, on the other hand, with Trypamidium and each sick animal received 10 ml of 10 solution intravenously.

Biochemical procedures used in this work were those after *WEICHSELBAUM (1946)* and *PATTON & GROUCH (1977)* for serum total protein and urea, respectively. Aspartate aminotransferase (AST) and alanine aminotransferase (ALT) were determined after *REITMAN and FRANKEL (1957)*. Calcium and inorganic phosphorus were estimated by the methods described by *GLINDLER & KING (1972) and KILCHLING & FREIBURG (1951)*, respectively. Fractionation of serum protein electro-phoretogram was performed after *DAVIS (1964) and ORNSTEIN (1964)*. Statistical analysis of data was performed according to *SNEDECOR and COCHRAN (1974)*.

RESULTS

In this work, camels naturally infected with microfilaria showed decreased appetite, emaciation, pale mucous membraneS and high body temperature (39.4 - 39.8 C). Moreover, the spermatic cord blood vessels were found rigid and the testicles as well as the scrotum were twice normal swollen. Cases of trypanosomiasis exhibited severe clinical signs manifested themselves in the form of weakness, emaciation, lacrimation, pale to icteric mucous membraneS, dry skin and intermittent fever (39.4 - 40.0 C). Tow cases showed also subcutaneous oedema on limbs and abdomen. The biochemical values are presented in Tables 1 & 2.

DISCUSSION

In this work clinical signs on camels, naturally infected with microfilaria, come in agneement with the obsenvations of *LOSOS (1986); ABU-EL-MAGD et al. (1988) and KARRAM et al. (1991)*. These signs gradually disappeared after treatment with Ivomec. Cases of trypanosomiasis, on the other hand, exhibited severe clinical signs manifested themselves in the form of weakness, emaciation, lacrimation, pale to icteric mucous

membranes, dry skin and intermittent fever (39.4 - 40.0 C). Two cases showed also subcutaneous oedema on limbs and abdomen. Signs on individuals of this group were in accordance with those recorded by *PARKER (1980); RAISINGHANI* et al. *(1980); GEORGI (1985) and ARAFFA (1990)* in Trypanosoma evansi infected animals. Emaciation, subcutaneous oedema and other clinical signs disappeared within one or two weeks after treatment with trypanidium.

Serum biochemical alterations in microfilaria and trypanosoma infected camels, as compared with apparently healthy ones, are demonstrated in tables 1 & 2. In camels with filariasis (Table 1) there was a significant drop in serum total protein, calcium and inorganic phosphorus together with a significant elevation in urea, AST and ALT. The significant drop in total protein might be the result of hypoalbuminaemia recorded in these camels. The present increase in urea concentration is similar to the results obtained b *EL-SEIFY* et al. *(1990)* in microfilaria infected buffaloes. Increased activities of AST and ALT might be the result of degenerative changes occured during migration of adult worms in different organs and tissues. *LOSOS (1986)* discussed similar behaviour where adult worms of filaria were found in the skin, subcutaneous tissue, eyes, lymph nodes, cardiovascular system, peritoneal cavity, central nervous system, spleen and the urinary organs. *Von Lichtenberg* et al. *(1962)* observed also hepatic lesions resulted from cardiac failure in dogs with dirofilariasis. The significant increase in the mean values of calcium and inorganic phosphorus in infected camels are in close agreement with the results obtained by *SINGH* et al. *(1972)* in buffaloes with microfilariasis.

The data presented in Table 2 showed that the most characteristic alterations in serum electrophoretic pattern of microfilaria infected camels, as compared with apparently healthy ones, were significant drop in albumin and beta-1-globulin and significant elevation in gamma-1b-globulin. Such a drop in albumin values could be attributed, in the view of *SINGH* et al. *(1972),* to either a direct inhibitory effect on albumin production, a more rapid albumin catabolism or an increased globulin concentration with consequent relative hypoalbuminaemia. The observed decrease in beta-1-globulin may explain the degree of liver and kidney dysfunctions *(PESCE and KAPLAN, 1987).* Similar findings were recorded by *ABU-EL-MAGD* et al. *(1988).* While, the significant increase in gamma-1b-globulin and consequently total gamma-globulin is most likely the result of increased immunoglobulins in response to

the constant stimulation of the parasite antigens (*MOUSTAFA, et al., 1991*). *WEIL et al. (1981)* observed an increase in IgQ and IgE antibodies in dogs chronically infected with *Dirofilaria limmitis*.

Regarding trypanosomiasis, the serum analysis of sick camels with comparison to apparently healthy ones have shown hypoproteinaemia and hypophosphataemia along with an increase in the concentration of urea, AST and ALT (Table 1). The observed hypoproteinaemia in infected camels might be the result of vascular escape of serum proteins to the subcutaneous oedema. This comes in agreement with that reported by *GAD-EL-MOULA et al. (1987)*. The apparent increase in urea concentration could be attributed to the degenerative changes occured in Kidneys. *RAISINGHANI et al. (1980)* recorded glomerulonephritis and severe congestion of the medullary area with variable degenerative changes of the tubular epithelium in experimental sura in camels. Higher activities of AST and ALT were recorded in infected camels. Such elevation in transaminases was expected to occur in association with pathological changes. Similar findings were previously observed by *RAISINGHANI et al. (1980)* in different organs especially the liver of trypanosoma infected camels. *ANOSA (1980)* recoreded decreased values of phosphate in *Trypanosoma evansi* infected camels and *Trypanosoma conglense* infected cattle. These results support the data of the present study.

Electrophoretic pattern in diseased camels (Table 2) showed also hypoalbuminaemia and hypergamma-globulinaemia. The marked reduction in albumin concentration might be the result of hepatic dysfunction and the excessive protein catabolism associated with the parasite toxins and febrile condition. Similar findings were previously recorded by *RAZA et al. (1982); SINGH et al. (1982) and VARMA & GAUTAM (1982)* in trypanosoma infected animals. Fractionation of gamma-globulin revealed an increase in the concentrations of gamma-1b, gamma-1d and gamma-2b in infected animals. This increase could explain the higher increase in the concentrations of immunoglobulins. *BOID et al. (1980)* observed 5 times increase in IgM levels in camel experimentally infected with *Trypansoma evansi*, even after treatment.

Finally, it can be concluded that all animals under study were successfully cured one month after the above described treatment. This was evidenced by microscopical examination of blood films obtained after treatment and complete disappearance of clinical signs as well as amelioration of altered serum components. This also explains that both Ivomec and Trypamidium are more effective parasiticidal drugs against microfilaria and trypanosoma, respectively.

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Table (1). Serum Organic and Inorganic Parameters in Microfilaria and Trypanosoma Infected Camels.

Studied Parameters	Apparently Healthy Camels	Camels with Filariasis		Camels with Trypanosomiasis	
		Diseased	Treated	Diseased	Treated
Total Protein (gm%)	7.84 ± 0.28	oo 5.72 ± 0.07	7.12 ± 0.10	oo 6.75 ± 0.15	7.45 ± 0.08
UREA (mg%)	30.17 ± 1.14	xxx 50.04 ± 1.87	31.06 ± 1.91	xxx 44.52 ± 1.82	28.70 ± 3.24
AST (U/ml)	30.88 ± 0.78	xxx 37.00 ± 0.85	32.00 ± 0.45	x 34.00 ± 0.87	32.40 ± 1.11
ALT (U/ml)	23.69 ± 0.66	xxx 29.40 ± 0.76	24.00 ± 0.86	xx 27.20 ± 0.73	24.40 ± 0.15
Calcium (mg%)	11.53 ± 0.52	oo 8.01 ± 0.33	12.19 ± 0.43	11.46 ± 0.47	11.83 ± 0.32
Inorganic-P (mg%)	6.22 ± 0.39	oo 2.55 ± 0.07	5.12 ± 0.37	oo 2.18 ± 0.10	o 4.30 ± 0.23

x/o = Significant at: x = Increase o = Decrease

x = P<0.05
xx/o = P<0.01
xxx/oo = P<0.001

Table (2). Serum Protein Electrophoretic Pattern in Microfilaria and Trypanosoma Infected Camels.

Fractions and Subfractions (%)	Apparently Healthy Camels	Camels with Filariasis		Camels with Trypanosomiasis	
		Diseased	Treated	Diseased	Treated
Albumin:	30.98 ± 2.01	25.83 ± 0.82	30.02 ± 1.35	24.54 ± 1.29	31.86 ± 1.84
Prealbumin	0.26 ± 0.03	0.28 ± 0.08	0.21 ± 0.03	0.31 ± 0.03	0.17 ± 0.04
Albumin	30.72 ± 2.03	25.55 ± 0.78	29.81 ± 1.33	24.23 ± 1.27	31.69 ± 1.82
Alpha-globulin:	10.36 ± 0.45	12.14 ± 1.32	10.96 ± 0.68	10.46 ± 0.33	11.09 ± 0.64
Alpha-1	3.11 ± 0.16	3.61 ± 0.52	3.26 ± 0.20	3.66 ± 0.12	3.03 ± 0.41
Alpha-2	3.68 ± 0.39	4.60 ± 0.64	4.42 ± 0.38	3.25 ± 0.13	4.00 ± 0.51
Alpha-3	3.57 ± 0.24	3.93 ± 0.32	3.28 ± 0.49	3.55 ± 0.16	4.06 ± 0.60
Beta-globulin:	26.04 ± 0.73	22.69 ± 1.21	27.70 ± 1.01	25.36 ± 0.60	25.52 ± 0.89
Beta-1	12.66 ± 0.57	9.10 ± 1.18	11.96 ± 0.22	10.45 ± 0.18	11.51 ± 0.45
Beta-2a	6.75 ± 0.55	7.31 ± 0.87	7.40 ± 0.69	7.38 ± 0.49	6.61 ± 0.87
Beta-2b	6.63 ± 0.70	6.28 ± 0.87	7.34 ± 0.97	7.53 ± 0.17	7.40 ± 0.37
Gamma-globulin:	32.62 ± 1.67	39.34 ± 1.93	32.31 ± 0.90	39.64 ± 0.59	31.53 ± 1.02
Gamma-1a	5.71 ± 0.48	6.13 ± 0.60	5.77 ± 0.54	5.79 ± 0.38	5.52 ± 0.51
Gamma-1b	11.82 ± 0.83	16.16 ± 0.74	13.69 ± 0.79	15.62 ± 1.31	12.79 ± 0.67
Gamma-1c	5.84 ± 0.36	7.00 ± 1.06	4.85 ± 1.09	7.29 ± 0.47	5.35 ± 0.55
Gamma-2a	5.14 ± 0.51	5.74 ± 0.50	3.44 ± 0.77	5.36 ± 0.99	3.85 ± 0.33
Gamma-2b	4.11 ± 0.26	4.31 ± 0.22	4.56 ± 0.69	5.58 ± 0.16	4.02 ± 0.21
Total Globulins	69.02 ± 2.01	74.17 ± 0.82	69.97 ± 1.35	75.46 ± 1.29	68.14 ± 1.84
A/G Ratio	0.44 ±0.036	0.35 ± 0.015	0.43 ±0.027	0.33 ±0.025	0.47 ±0.040

x/o - Significant at: x/o = P<0.05, xx = P<0.01

x = Increase o = decrease