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INVESTIGATION ON RICKETS PROBLEM OF FATTENING CALVES AT QENA PROVINCE

(With 2 Tables & 3 Figs.)

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شملت هذه الدراسة عدد ١٦ عجل من عجول التسمين بمحطة التسمين رقم ٢ بمحافظة قنسا وظهرت عليها الأعراض الإكلينيكية للكساح على عدد ثماينة منها بينما أعتبرت الثماني وظهرت عليها الأخرى كضوابط للتجربة وقد تمت دراسة الأعراض الإكلينيكية في الحيوانات المريض ومقارنتها بالسليمة بالإضافة إلى الصورة الدموية والبيوكيميائية • كما تم علاج الحيوان المريضة بإستخدام الأملاح المعدنية وحقن فيتامين أدم هد في العضل بالإضافة إلى حقن خليط الكالسيوم والماغنسيوم في الوريد وتعريض الحيوانات للأشعة الفوق بنفسجية ثم أخذ عينات بعد العلاج بشهر شم شهرين وعمل قياس للمقارنات المختلفة •

SUMMARY

Signs of rickets on eight freisian calves under eight months old at Qena fattening station were observed. The clinical signs of rickets appeared in the form of stiffness, difficult in movement and rotation of the carpal joints.

Haemogram picture was studied. This included total erythrocytic & leucocytic count, haemoglobin concentration, packed cell volume. Mean corpscular volume and mean corpscular haemoglobin concentration were calculated.

Biochemical analysis of blood sera for calcium, inorganic phosphorus, magnesium, total protein, albumin, globulin and electrolytes were performed to confirm the diagnosis of rickets. Treatment of diseased animal have been carried out.

INTRODUCTION

Rickets is one of the most important diseases of young animals through which we can conclude some sort of disturbance in calcium, phosphorous and vit. D. metabolism (RADOSTITS and BLOOD (1985) and BONNIWELL, et al. 1982).

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Clinical signs of rickets are characterized by general unthriftness, Loss of body weight, reduced food consumption, reluctance to move, abnormal bony structure associated with enlargement of epiphysis, bented shaft and finally associated with impaired reproductive performance (CALL, et al. 1986 and 1987).

Calcium and phosphorus are essential in mineralization of bone and teeth and deficiency of one them leads to their abnormal developement. The effect of ca/p imbalance have been studied by CALL, et al. (1986); YOUSIF, et al. (1986) and HOWARD (1981).

In trial to study subclinical rickets ATTIA (1984) and EL-SHERIF and FAWZIA (1981) concluded that it can be diagnosed through biochemical analysis of blood serum. ATTIA (1984) added that the subclinical rickets have been developed when the ca/p ratio elevated above 2 without obvious clinical signs. But when the ca/p ratio was above 2 and associated with clear clinical signs this form is thus diagnosed as clinical rickets.

The aim of the present work is to confirm both the diagnosis and the role of treatment of diseased richitic calves conditions in alleviation and progress of the case.

MATERIAL and METHODS

A stock of 16 young freisian calves (under eight months) were housed indoors in Qena fattening station. Eight of them have been suffered from disturbance in movement, deformities in bony structure, stunted growth, unthriftness, loss of body weight and reluctance of movement Fig. (1,2). Other eight calves have been proved to be healthy by both clinical and laboratory methods of examination and were considered as a control group for this study.

Two blood samples were obtained from each animals through Jugular vien pincture at once and after one and two months post-treatment. First blood samples (whole blood using anticoagulent) have been used in determination of haemogram picture including total erythrocytic count (T.R.B.Cs.-T/L); total leucocytic count (T.W.B.Cs.-G/L) and haemoglobin concentration (Hb-gm/L) using electronic cell counter (CX300) and Diluter (DC.210). Packed cell volume (PCV%) mean corpscular volumed (MCV-³u) and mean corpscular haemoglobin concentration (MCHC-%) have been estimated according to the method described by COLES (1986). Second blood samples (blood sera) were used for biochemical analysis. Blood serum total protein (gm/dl); albumin (gm/dl); calcium (mg/dl), inorganic phosphorus (mg/dl) and magnesium (mg/dl) were determined using test kits supplied from Biomerieux (Bains/France) and after the methods of WEICHSELB AUM (1946); DRUPTF (1974); GINDLER and KING (1972); MORINL and PROX (1973) and GINDER and HETH (1971) respectively. Blood serum globulin and albumin/globulin (A/G) ratio were estimated mathematically.

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The concentration of blood serum sodium and potassium levels were estimated using Flame-photometer (Corning 400) meanwhile blood serum chloride level was determined using chloride meter Model 925.

The diseased animals were treated using mineral mixture and injection of Cal-D-Mag (500 ml/day I/V) and turlin (AD $_3$ E-10 ml/day I/M).

The treatment was extended to one week. Also the diseased calves were exposed to daily sunlight, second and third blood samples were taken 30 and 60 days post-examination.

Statistical analysis of the data were performed according to the method of KALTON (1967) using T-test.

RESULTS

Mean values of the haemogram picture and biochemical constituents of blood serum are illustred in tables (1 & 2) and Figs. (1, 2 & 3).

DISCUSSION

The clinical signs which have been observed in clinically diseased calves are stiffness in gait, enlargement of the limb joint especially the forelegs and the enlargement of the costochondral Junctions. (Picture 1, 2). The large bone show abnormal curveture. Delayed eruption of teeth and presence of abnormalities in it as pitting, groves and pigmention were evident. Animals also show anoroxia, unthriftness, difficulty in feeding and drooling of saliva. These clinical signs were in agreement with those previously observed by RADOSTITS and BLOOD (1985); COLES (1986) and UNDERWOOD (1979) in a similar conditions.

Regarding haemogram picture which included total erythrocytic count, haemoglobin concentration, pakeed cell volume, total leucocytic, mean corpscular volume and mean corpscular haemoglobin concentration which have been illustrated in table (1), there were non-significant variations in all studied parameters except a significant decrease in total erythrocytic count in diseased calves if compared with the healthy one. This can be attributed to anaemic condition resulting from anorexia and abnormalities in teeth. These results coincided with those previousely obtained by EL-SHERIF and FAWZIA (1984) and ATTIA (1984) in lambs.

There is a non-significant variations in studied haemogram picture either in diseased calves or after application of treatment. The obtained values are within the normal levels previously mentioned by COLES (1986) DUNCAN and PRASSE (1986) and SCHALM (1979) in young calves.

The biochemical analysis of blood sera (Table 2, Fig. 3) declared that, there were highly significant decrease (P/ 0.01) in the levels of blood serum calcium, inorganic phosphorus, magnesium, total protein and albumin in richitis calves if compared with

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the healthy ones. Hypoproteinaemia associated with hypoalbuminaemia in richitic calves were evident. This can be attributed to the unthriftness of animal due to state of anoroxia or it may be due to the role played by inorganic phosphorus in intermediam protein metabolism YOUSIF, et al. (1986); ATTIA (1984); EL-SHERIF and FAWZIA (1981) and UDALL (1972).

An evident reduction in the level of blood serum calcium, phosphorus and magnesium in richitic calves were found if compared with healthy ones. These coincided with the results previousely obtained by NORDIN (1960) and UDALL (1972) which attributed this reduction to the decrease phosporus in diet which may leads to withdrowal of both calcium and phosphorus from bone with an increased calcium excretion rate accompanied with consequent rarification of theses bones.

Blood serum electrolytes levels showed a significant decrease (P/ 0.05) in richitic calves if compared with the healthy ones. The obtained values came in agreement with those previousely obtained by COLES (1986).

The plan of treatment using cal-D-Mag, AD, E and mineral mixture-gave a good results in correcting both haemogram and biochemical constituents but the treatment can not give any progress or alleviation of bony changes.

Finally the studied parameters confirm the diagnosis of rickets and showed the role of treatment in producing alleviation in blood constituents of diseased calves to the normal physiological levels.

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Table (1)
Haemograme picture of examined calves pre and post treatment

Condition	Animal	Animal T.R.B.Cs T/L	P.C.V.	Hb gn%	M.C.V.	E.C.H.C.	T.W.B.Cs G/L
Rickets	8	6.83±1.09 32.1±1.6	32.1+1.6	11.98+1.01	48.1+8.7	37.4+4.1	13.0+1.5
Healthy	8	7.54±0.44 34.1±2.6	34.1+2.6	13.79±0.86 45.4±3.5	45.4+3.5	40.5+4.2	12.95+0.90
Rickets	8	6.23+1.09 29.0+4.12	29.0+4.12	13.06±1.7	47.75±8.79	45.67+7.72	17.18±6.13
Healtay (post 30Ds)	8	6.97±1.16	30.63+3.99	6.97±1.16 30.63±3.99 17.13±2.16	44.58±6.97	56.96+12.39	16.21+3.94
Rickets	8	5.88±0.66 28.0±1.00	28.0+1.00	14.0+18	47.62±5.60	60.66+6.0	16.80±3.05
Healtny (post 60 Ds)	œ	6.30+1.30	6.30+1.30 28.6+3.00	15.10+2.0	47.67±6.66	52.8±5.18	14.00+2.84

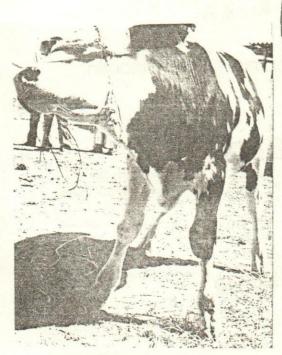




Fig. (1 & 2):

Showing clinical signs of rickets in examined Cattle.

Condition	Totul	Albumin	Globulin	1./6	Celcium	Pnosphorus	Mcgnesium Sodium	Sodium	Potessium Chloride
Condition	protein	S. C.	gmg.	ratio	He Ma	mg/8	ms %	mmol/L	mmol/L mmol/L
Rickets	5.76±0.50	3.01+0.8	2.75±0.7	1.23±0.8	7.28+1.14	5.53±0.3	1.94+0.3	1.94+0.3 136.4+5.4	5.3+0.4
	7.9 ±0.62	5.01±0.5	2.88±0.5	1.8 ±0.5	10.43±0.93	8.05±0.4	2.66+0.39	2.66±0.39 144.3±5.47 6.16±0.36 124±6.8	6.16+3.3
Rickets	5.78+1.74	2.51+0.94	2.86+1.3	1.05±0.4	8.02+1.23 6.74+0.3	6.74±0.3	2.89+3.29	2.89±3.29 132.9±5.7	5.5 +3.98 110.88+5.94
Heal tay	8.26+1.26	5.2 ±0.76	3.06+0.74 1.76+0.4	1.76±0.4	10.73+1.29 10.43+1.24	10.43:1.24	3.29+0.64	3.29+3.64 156.6+12.39 6.84+0.52 131.12+5.30	6.84+0.5
(post 30Ds)				-		4			
Rickets	7.58±0.78	4.44±0.3	3.09+0.6	0.98+0.5	9.6 +1.14	8.7 ±0.5	3.0 ±0.4	120 ±6.8	5.0 ±0.6 112.12±6
Heal tay	8.36+1.38	5.1 ±0.66	3.28±0.9	1.8 ±0.5	10.0 ±0.56	8.9 ±6.2	3.10±0.5	140.6±4.9	5.8 ±3.5 125.9±5.0
(post 60Ds)									
1									

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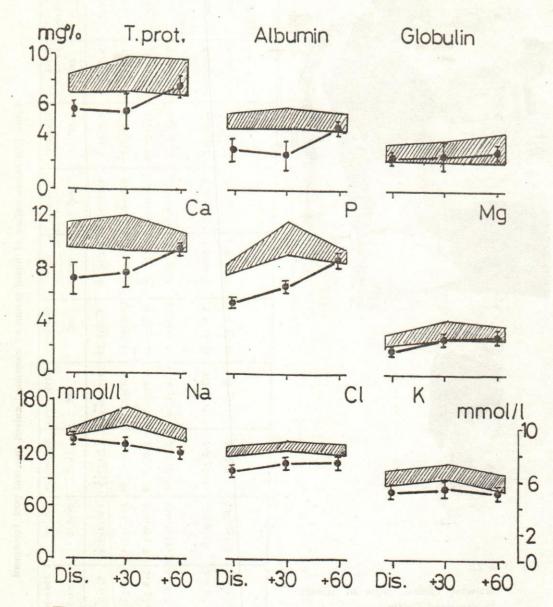


Fig.3. Mean values of studied blood serum parameters. \$\overline{x} \tilde{x} \tilde{s} \tilde{S} \tilde{D}\$ Healthy calves \$\overline{x} \tilde{x} \tilde{S} \tilde{D}\$ Diseased calves .

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