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Serum enzyme profile in post parturient bovines suffering from anorexia and ketosis

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ABSTRACT

Fifty two postparturient animals included in this study. The serum levels of Gl-6 Pase were higher in the anorectic bovines and ketotic buffaloes, particularly, around 2 months lactation. The serum levels of liver specific enzymes, viz., GOT, GPT and SAP were also recorded to be on higher side during anorexia and ketosis. However, the levels of acetylcholinesterase were low in the anorectic and ketotic animals. The treatment of the anorexic and ketotic animals with propylene glycol reversed back the enzymatic changes in the sera samples of these animals.

Key words: Postparturient bovines, serum enzymes, Anorexia and Ketosis

INTRODUCTION

Gluconeogenesis is a prime compensatory mechanism during anorexia to maintain proper supply of glucose. Involvement of liver during anorexia and ketosis is inevitable, since, gluconeogenesis mainly takes place in the liver. A depressed gluconeogenesis in the liver is associated with oxaloacatate deficiency in ketotic animals (Kaneko *et. al,* 1997). The present study was taken up in the cows and buffaloes of within a week and 2 months lactation (Peak lactation) in order to asses the status of Gl-6 Phosphatase, Fr-1,6 Diphosphatase, alkaline phosphatase, transaminases and acetyl cholinesterase during anorexia and ketosis.

MATERIALS AND METHODS

A total of 52 animals (postparturient cows - 24 and buffaloes -28 of around one week and two months lactation), belonging to the private dairy farms and College Livestock Farm, Adhartal, Jabalpur were selected for the study. The study included the non-anorectic animals, which served as control and anorectic lactating bovines including 4 ketotic buffaloes at around two months lactation.The anorectic and ketotic animals were given propylene glycol @ 220 ml/day for first two days followed by 110 ml/day for subsequent two days. The serum enzymes, viz., Glucose-6 Phosphatase (Gl-6 Pase) (Swenson, 1955), Fructose-1,6 Diphosphatase (Fr-1,6 Dipase) (McGlivery and Pogell, 1955), red cells and plasma Acetylcholinesterase (AChE) (Hestrin, 1949), Serum Alkaline Phosphatase (SAP) (Oser, 1965) and GOT and GPT (Yatzidis, 1960) were estimated. The data was statistically analysed and group differences worked out by Students 't' test as per standard procedures.

RESULTS AND DISCUSSION

The serum enzyme levels of non- anorectic (control), anorectic and ketotic animals at around a week and two months of lactation and the effect of propylene glycol has been presented in the Table 1 and 2. Among the gluconeogenic enzymes, viz., Gl-6 Pase and Fr-1,6 Dipase, the latter did not vary much in the different physio-pathological conditions of the animals, as earlier recorded in the ketotic animals (Rukkawamsuk *et al.* 1999). However, the same was higher ($P<0.05$) in anorectic animals at two months lactation in comparison to those around a week postparturitum. The serum Gl-6 Pase level was conspicuously higher in the animals where blood glucose was low and gluconeogenesis was expected to be enhanced, as reported by Fowden *et al.* (1990) in anorectic and by Veenhuizen *et al.* (1991) in ketotic animals. The values of serum Gl-6 Pase were

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Table - 1. Enzymic profile of anorectic cows and buffaloes during a week and two months postpartum treated with propylene glycol

Parameters		With in a week parturition Anorectic			Two months postpartum Anorectic		
		Non- anorectic (Control)	Before treatment	After treatment with propylene glycol	Non- anorectic (Control)	Before treatment	After treatment with propylene glycol
Gl-6 Pase	C	64.70±7.10	84.70±18.31	92.84±15.70	127.87±5.56	167.78±12.21*	109.13±16.24
(µm/L/min)	B	91.22±0.98	154.42±10.20*	115.65±5.76	133.57±2.06	162.89±9.21*	99.36±1.47
Fr-1,6 Dipase	C	66.94±3.81	30.32±5.33*	58.64±5.66	64.01±5.63	66.94±3.40	60.10±4.52
(µm/L/min)	B	68.33±1.05	69.39±2.94	66.94±4.69	62.54±1.72	74.39±1.14*	52.38±4.64
AchE (R)	C	609.99±184.17	493.76±69.95	551.90±53.56	871.43±100.62	747.14±55.37	813.27±86.13
(µm/L/hr)	B	768.05±3.48	688.64±9.32	900.40±5.25	900.40±3.79	677.61±28.14*	835.00±36.32
AchE (P)	C	406.66±58.09	365.23±58.22	392.13±83.43	493.94±69.89	348.56±63.63	464.75±43.08
(µm/L/hr)	B	493.73±8.27	464.75±8.74	479.28±58.83	450.28±39.06	405.52±39.49	435.89±35.52
Alkaline phosphatase	C	7.43±2.86	10.58±2.37	5.69±1.24	5.56±0.99	5.57±0.74	5.83±1.66
(µm/L/min)	B	10.24±0.44	4.33±0.23*	5.15±0.36	5.85±0.21	8.86±0.11	6.14±1.15
SGOT	C	116.30±10.45	136.82±8.20	164.20±24.81	136.82±7.89	164.19±23.69	117.88±38.07
(µm/L/min)	B	91.67±1.54	56.55±6.75*	120.40±1.51	165.65±3.53	157.35±5.71	155.06±7.49
SGPT	C	9.34±0.82	8.25±3.39	8.20±2.72	8.66±1.44	7.97±1.47	9,52±1.69
(µm/L/min)	B	19.22±0.66	7.94±0.32*	15.53±1.57	12. 99±0.79	10.25±0.41	11.48±0.44

* P < 0.05 C = Cows
** P < 0.01 B = Buffaloes

Table 2. Enzymic profile of clinical ketotic buffaloes treated with propylene glycol

Parameters	2 months post partum (Control)	Ketotic	After Treatment with propylene glycol
Gl-6 Pase (μm/L/min)	133.57±2.06	193..52±2.94*	123.17±7.82
Fr-1,6 Dipase (μm/L/min)	62.54±1.72	93.82±2.93*	65.23±7.79
Ach E (R) (μm/L/hr)	900.40±3.79	261.42±50.31**	784.28±112.50
Ach E (P) (μm/L/hr)	450.28±39.06	304.95±21.73*	523.06±71.21
Alkaline phosphatase (μm/L/min)	5.85±0.21	10.85±0.17*	4.62±1.86
SGOT (μm/L/min)	165.65±3.53	191.56±7.9*	164.19±5.58
SGPT (μm/L/min)	12.99±0.79	16.75±2.39*	9.91±0.94

*P < 0.05, **P<0.01.

higher in the anorectic and ketotic buffaloes at around two months lactation, which indicates the compensatory gluconeogenesis activity during this period. The enhanced levels of Gl-6 Pase in the serum might have come from the hepatocytes and kidney, the seat of gluconeogenesis and might have got leaked from these tissues. The increased permeability of the hepatic cells is evident from the enhanced concentration of liver specific enzymes, (GOT, GPT and SAP) in the sera samples of anorectic and ketotic animals. The levels of AchE in the red cell wall and plasma were lower during anorexia and ketosis, leaving more Acetylcholine (Ach) unhydrolysed to stimulate glucogenesis. The possible involvement of parasympathetic system in glucogenesis was indicated in this study also, as recorded by Mayer, (1980). The activity of AchE in the anorectic animals of two months lactation was significantly (P<0.05) higher than that of the animals during a week postpartum.The blood ketone levels were negatively correlated with the AchE activities of the red cell wall and plasma of both the species to the extent of being significant ($p < 0.01$) in the buffaloes { $r = -0.559$(R), $r = -0.287$ (P)}. One fact was again very clear that the distorted levels of these enzymes were brought back to the normalcy after administration of propylene glycol, which in the rumen got converted into glycerol and pyruvate to serve the gluconeogenesis in the anorectic and ketotic animals. This observation indicated that there occurred a deficiency of propionate, a principal glucogenic substrate during anorexia and ketosis.

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