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HEPATIC AND RENAL DYSFUNCTIONS IN EXPERIMENTAL OVINE PREGNANCY TOXAEMIA

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Considerable economic loss occur to sheep and wool industry owing to pregnancy Toxaemia (PT). Earlier studies have described abnormalities of carbohydrate and lipid metabolism characterized by decreased blood glucose, elevation of blood ketones, hypercortisolemia and marked impairment of hepatic and renal functions in pregnancy toxaemia in ewes (Mc Causland *et al.*, 1974). The present study reports the clinico-biochemical features in pregnant sheep in whisch PT was induced by the administration of PZ Insulin and semi-starvation.

Materials and Methods

Sixteen, 7-8 yrs old, Muzzaffarnagari ewes in third trimester of gestation (115 ± 10 days) were used for this study. Ewes were grouped into six animals of group I and II and four animals of group III as control. Pregnancy toxaemia was induced by administration (S/C) of protamin zinc insulin (M/S Boots Co. India, Bombay) @ 40 and 20 units per ewe on alternate days in Gr. I and II, respectively. The liver function and kidney function tests were carried out in all the experimental and control group on 0, Ist, III and VI weeks of the experiment. The percentage of retention of BSP was determined for individual ewes as per the method of Davidson and Wells (1963). Sulfobromophthalein dye (@ 5 mg/kg body weight) was injected slowly into the jugular vein. Blood samples were collected from the vein of the opposite side after 3 and 10 mts of the dye injection. Blood glucose, ketone bodies (acetone), cholesterol, total protein, BUN and creatinine were analysed in the serum at 0, I, III and VI weeks in the animals of experimental and control groups.

Results and Discussion

The results of biochemical observations including liver and kidney function tests are presented in Table 1. The experiment lasted till full gestation period (3 wks in Gr. I and 6 wks in Gr. II & III) or lambing/death of the ewes due to pregnancy toxaemia.

The animals of experimental groups (Gr.I and II) exhibited moderate to marked degree of clinical signs/symptoms of pregnancy toxaemia after administration of Insulin followed by semi starvation. The symptoms of PT exhibited by experimental animals (Gr. I and II) were anorexia, dullness, depression, mild to moderate symptoms of staggering gait, incoordination of movements, lowering of head, ataxia, convulsions, sternal/lateral recumbency, development of ketonuria and proteinuria, detection of sweat odour of ketones in breath, inability to get up and expell their foetii out during lambing. These findings are in conformity with earlier

Table 1 Biochemical profile of ewes with induced pregnancy toxaemia

	Parameters	Group with No. of animals	Values at different duration of experiment			
			Pre-exptl. (0 wk)	1 week	3 week	6 week
1.	Blood glucose (mg/dl.)	I (6)	58.0±3.86	45.5±5.86*	20.4±4.20**	-
		II (6)	54.3±4.11	49.5±5.77*	32.0±5.67**	22.0±5.20**
		III (4)	62.7±4.38	60.5±5.21	59.0±3.88	57.2±6.55
2.	Serum ketone bodies/ acetone (mg/dl)	I (6)	3.5±0.16	6.5±0.72*	28.1±4.21**	-
		II (6)	3.8±0.13	5.8±0.84	12.6±2.84**	31.4±6.11**
		III (6)	4.0±0.26	4.2±0.37	4.7±0.48	6.1±0.77
3.	BSP clearence (% retention)	I (6)	3.0±0.28	4.2±0.78*	18.2±3.17**	-
		II (6)	2.8±0.18	3.1±0.26	7.4±1.44**	26.4±3.86**
		III (4)	3.1±0.24	2.8±0.16	3.3±0.37	5.6±0.86
4.	Serum total protein (gm/dl)	I (6)	6.6±0.27	6.1±0.43*	5.3±0.93**	-
		II (6)	6.4±0.18	6.2±0.51	5.6±0.68*	4.9±1.88*
		III (6)	6.5±0.21	6.6±0.26	6.4±0.32	6.3±0.38
5.	Serum cholesterol (mg/dl)	I (6)	96.0±5.71	116.0±7.57	192.0±19.57**	-
		II (6)	90.0±5.12	106.0±5.88	158±14.82**	286±21.66**
		III (4)	86.0±4.98	94.0±5.08	105.0±6.70	128±8.43
6.	BUN (mg/dl)	I (6)	14.3±1.81	19.4±3.66	37.8±6.98**	-
		II (6)	15.5±1.26	18.5±2.84	26.6±4.65**	44.5±5.66**
		III (4)	16.2±1.32	17.0±1.55	19.5±2.41	20.8±2.88
7.	Serum creating (mg/dl)	I (6)	1.1±0.08	1.4±0.28	3.36±0.68**	-
		II (6)	1.2±0.16	1.3±0.21	2.1±0.51*	4.10±1.47**
		III (4)	1.2±0.12	1.1±0.09	1.2±0.19	1.3±0.26

* Significant at $P < 0.05$ and ** Significant at $P < 0.01$

Mean comparisons represents, significance along columns within control (III) and experimental (I & II) groups

observations (Mc Causland *et al.*, 1974; Ranaveera, 1980; Vihan and Rai, 1984; Sigurdsson, 1988). The course of the clinical illness ranged from 3 to 9 days. Severity of symptoms was more in Gr. I animals, but the course of the clinical illness was shorter in the ewes of the Gr. I than Gr. II as reported by Reid, (1968).

A drop in blood glucose level became evident from the 1st week in the animals of Gr. I and II, and marked fall (20.4 and 22.0 mg/dl) was observed during 3rd and 6th week of experiment. Similar findings have been reported in experimental pregnancy toxaemia in ewes (Mc Clymont and Setchell, 1955, Vihan and Rai, 1984). It has been

postulated that nervous signs observed are due to inhibition of glucose uptake/utilization by the brain as a result of hypercortisolemia. The levels of ketone bodies (acetone) significantly increased at the onset of clinical symptoms of PT in experimental ewes and was maximum (28.1 and 31.4 mg/dl in Gr. I and II, respectively) at 3rd and 6th week of the experiment, as observed by Reid, (1968) and Vihan and Rai (1984). The ketone bodies were detected in urine also.

Liver Function Test

A significant ($P < 0.05/0.01$) increase was observed in the per centage retention (18.2 ± 3.7 and 26.4 ± 3.84 respectively) of BSP during the 3rd and 6th week in the experimental animals of Gr. I & II, when compared to control (Gr. III). An increase in the BSP retention in the present study indicated disturbance in the excretory function and hepatocellular damage. The levels of serum total cholesterol also increased (192 and 286 mg/dl) significantly and progressively at the onset of clinical symptoms in the animals of Gr. I and II. Mobilization of fat from its depots as a source of energy would have been responsible for hypercholesterolemia. A significant fall occurred in total serum protein (5.3 and 4.9 gm/dl) during the 3rd and 6th week of the experiment in Gr. I and II, which can be attributed to the depletion of fat depots in the last stage of the ailment as this would have compelled the toxaemic pregnant sheep to depend upon their protein source for energy resulting in increased protein catabolism (gluconeogenesis) of body proteins (Reid, 1968).

Kidney Function Test

A progressive and significant ($P < 0.05/0.01$) increase in BUN concentration was observed from the Ist week onwards in experimental ewes (Gr. I and II). The peak reached in 3rd week (37.8 mg/dl) and 6 wk (44.5 mg/dl) in the animals of Gr. I and II, respectively. Mean serum creatinine level was comparable in experimental and control groups. Subsequently a significant ($P < 0.05/0.01$) increase occurred in the levels during the 3rd and 6th week (3.36 and 4.10 mg/dl) in Gr. I and II. These observations are in agreement with the findings of Saba *et al.* (1966) Mc Causland *et al.* (1974) and Hill *et al.* (1984), suggesting impairement of renal functions in ewes with PT.

A decrease in the total protein concentrations in the present study would have been due to marked proteinuria (albuminuria) as a result of renal damage/dysfunction as it usually occurs in PT (Mc Causland *et al.*, 1974; Hill *et al.*, 1984). According to Reid (1968) and Mc Causland *et al.* (1974) renal dysfunctions are apparent in terminal stages of the disease and contribute to the development of clinical signs and fatal outcome. Present observations indicated that hepatic and renal dysfunctions started at the onset of clinical symptoms of PT.

Summary

Induced pregnancy toxaemia in ewes showed marked hepatic and renal dysfunctions and clinical symptoms were characterized by anorexia, dullness, depression, nervous symptoms and proteinuria. Liver and kidney function tests revealed significant increase in per cent

retention of BSP dye, levels of serum ketones, serum total cholesterol, BUN and creatinine. Significant reduction in the levels of blood glucose and total serum protein, was also observed.

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