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BLOOD CHEMICAL AND ENDOCRINE CHANGES IN SHEEP WITH EXPERIMENTAL CHRONIC ACIDOSIS

P. GEORGIEV & Y. NIKOLOV

Faculty of Veterinary Medicine, Trakia University,
Stara Zagora, Bulgaria

Summary

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Determinations of blood total protein, blood glucose, cholesterol, total lipids, triiodothyronine (T3), thyroxine (T4) and cortisol concentrations were performed in 5 sheet at various intervals up to the 60th day post initiation of feeding a diet producing experimental chronic rumen acidosis.

The experimental disease was manifested by various alterations at the different time intervals. The quantity of total protein did not change statistically significantly during the entire period of the study. Blood glucose increased from 2.4 ± 0.11 mmol/L prior to treatment to 4.7 ± 0.08 mmol/L by post treatment day 60.

The levels of cholesterol and total lipids decreased from baseline values of 65.3 ± 5.3 and 246.6 ± 20.3 mg/100mL to 42.2 ± 2,9 and 178.2 ± 9.2 mg/100 mL by day 60, respectively.

The levels of T3, T4 and cortisol exhibited a clear phasic pattern of changes. The pretreatment levels of hormones were 1.14 ± 0.10, 59.0 ± 7.0 and 9.4 ± 2.0 nmol/L respectively. After the treatment, they increased to 1.95 ± 0.08, 98.0 ± 10.0 and 22.0 ± 5.0 nmol/L (by post treatment day 10) and 2.03 ± 0.08, 97.0 ± 6.0 and 26.7 ± 2.7 nmol/L (by the end of the study – day 60) respectively.

Key words: adrenal glands, blood chemistry, lactic acidosis, rumen, sheep, thyroid gland

INTRODUCTION

The chronic rumen acidosis in ruminants is still among unresolved problems, especially under conditions of intensive technologies of breeding.

For elucidation of the mechanisms of that disorder, the studies of some authors have been focused on the motoric, chemical and microbiological functions of rumen. In that connection, attempts for interpretation of data for the changes in some biochemical parameters (blood sugar, total protein, lipids etc.) were performed, but an explicit statement about the etiopathogenesis of the chronic rumen lactic acidosis was not assumed (Sodhi, 1981; Abdel-Rasek, 1988; Augustinson, 1989; Pavlov *et al.*, 1994; Nikolov and Kostov, 1994; Niles *et al.*, 1998).

The published reports about the participation of some parts of the endocrine system (thyroid gland, adrenal glands, pancreas), are incomplete or interpreted without connection with the rest of the clinical and laboratory parameters (Tauwar, 1983; Owen *et al.*, 1988).

Despite that the role of T3, T4 and cortisol in anabolic and catabolic rates is established without any doubt, their direct involvement in the mechanisms of rumen acidosis and the effects resulting from this disorder still remain unclear.

The still undefined circumstances about the incidence and the development of rumen acidosis in shepp as well as the various and not always complete studies give us the reason to perform simultaneous laboratory and endocrine studies in sheep with an experimental chronic rumen lactic acidosis in order to receive more information about the interrelationships between endocrine factors and metabolic disorders.

MATERIALS AND METHODS

The experiment was performed on 5 sheep from a local breed, weighing 50–65 kg, 4–5 years old.

The chronic rumen acidosis was provoked by the following daily diet: 0.5 kg concentrated forage and free access to beet with a sugar content of 140 g/kg and to water. The daily beet consumption of each animal was about 5 kg. The diet was maintained for 60 days.

The following biochemical parameters were determined: blood glucose, total protein, cholesterol and total lipids (Ibrishimov *et al.*, 1987; Tilton *et al.*, 1992). The levels of T3, T4 and cortisol were quantitated using RIA methods (Laudat, 1988; Stockigt, 1996).

Blood for biochemical analysis was sampled from *v. jugularis* prior to the study and by days 10, 20, 30, 40, 50 and 60 after the administration of the diet.

The results were statistically processed using the Student's t-test.

RESULTS

The changes in total protein, blood glucose, cholesterol and total lipids are

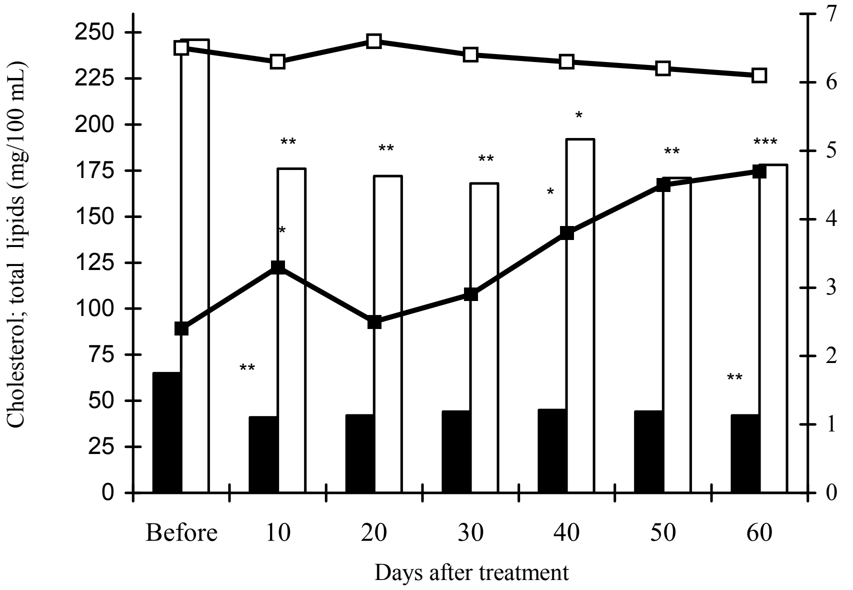

Fig. 1. Dynamics of blood cholesterol (black bars), total lipids (white bars), total protein (–□–) and blood sugar (–■–) in sheep with chronic rumen acidosis. *$p<0.05$; **$p<0.01$; ***$p<0.001$ vs baseline.

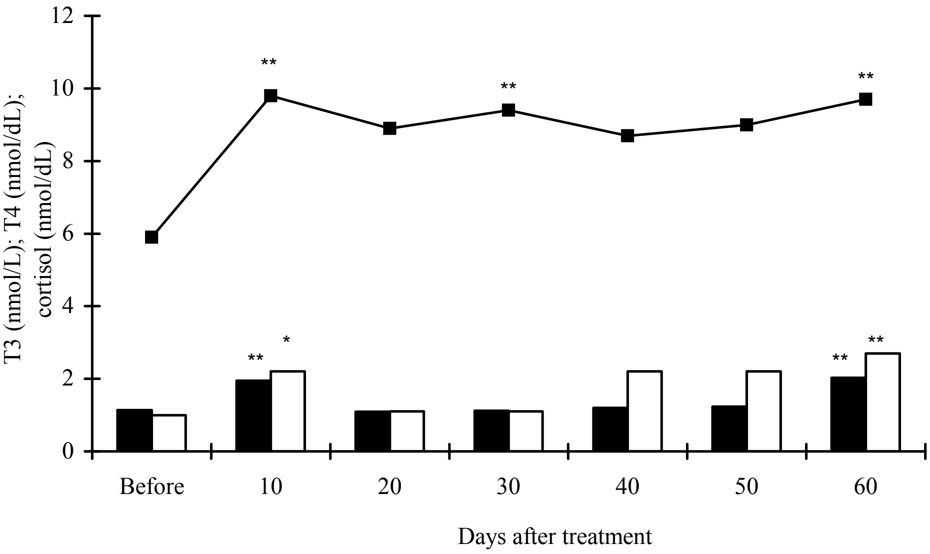

Fig. 2. Dynamics of T3 (black bars), T4 (–■–) and cortisol (white bars) in sheep with chronic rumen acidosis. *p<0.05; **p<0.01 vs baseline.

shown on Fig. 1. They differed by respect to both extent and time of appearance.

Total protein concentrations that had initial values of 65.5 ± 0.9 g/L changed insignificantly to 61.4 ± 1.1 g/L at the end of the experiment.

The most significant changes in cholesterol and total lipids levels occurred in the early periods after the treatment (day 10). Cholesterol levels decreased from 65.3 ± 5.3 mg/100 mL before the treatment to 41.0 ± 3.0 mg/100 mL by day 10. Its concentrations remained lower than baseline ones until the end of the experiment. Total lipids decreased statistically significantly from 246.6 ± 20.3 mg/100 mL (baseline) to 176.0 ± 9.8 mg/100 mL by day 10 (p<0.01). Thereafter, the parameter did not regain the initial levels and remaied within 176.0 ± 9.8 – 178.2 ± 9.2 mg/100 mL.

Blood glucose concentrations tended to increase continuously. Their pretreatment values of 2.4 ± 0.11 mmol/L were significantly increased by day 10 (3.30 ± 0.09 mmol/L; p<0.05) and reached 4.70 ± 0.08 mmol/L by day 60 (p<0.01).

The data for blood T3, T4 and cortisol cncentrations are shown on Fig. 2. As early as post treatment day 10, T3 levels were significantly (p<0.01) higher than baseline ones (1.95 ± 0.08 nmol/L vs 1.14 ± 0.10 nmol/L respectively). Between post treatment days 10 and 20, T3 decreased and by day 20 attained the initial levels. Afterwards, T3 values increased again, although at a different rate, and at the end of the study (day 60) they were 2.03±0.08 nmol/L (p<0.01). The changes in T4 were unidirectional. Compared to pretreatment values (59±7 nmol/L) they increased statistically significantly (p< 0.01) by day 10 (98 ± 10 nmol/L), day 30 (94 ± 12 nmol/L) and day 60 (97 ± 6 nmol/L).

The chronic lactic acidosis induced considerable fluctuations in cortisol levels. As early as post treatment day 10, they were higher than the initial ones (22.0 ± 5.0 nmol/L vs 9.4 ± 2.0 nmol/L, $p<0.05$). Between days 10 and 30, cortisol concentrations decreased and reached pretreatment values. After the 30th day, it increased again and at the end of the study reached 26.7±2.7 nmol/L ($p<0.01$).

DISCUSSION

The data about the dynamics of studied biochemical and endocrine parameters, obtained by us, were most probably due to systemic disorders (hepatic, thyroid, adrenal, pancreatic etc.) that appeared as a consequance of impaired physiology of forestomachs or the rumen (Pavlov *et al.*, 1994; Nikolov and Kostov, 1994).

The rumen lactic acidosis is observed in cases of single intake of carbohydrates that exceed considerably the physiological needs (acute form) or after continous ingestion of forages rich in easily digestible carbohydrates on the background of a non-balanced diet (chronic form). The physiological excess of carbohydrates in the diet causes biochemical and microbiological alterations in rumen (Ivanov, 1979; Russell and Hino, 1985; Church, 1993; Nikolov and Kostov, 1994). This is accompanied by transformation of volatile fatty acids into acetate, lactate, propionate etc. that could not be metabolized into glycogen in liver and thus, cause increased blood glucose concentrations (hyperglycaemia). The metabolic and endocrine systemic disturbances are triggered by the increased levels of lactate and histamine (Juhasz, 1968; Tauwar, 1983; Nikolov and Kostov, 1994; Stocker *et al.*, 1999).

The developing rumen acidosis is further resulting in systemic metabolic acidosis (Sugawara, 1971; Sodhi, 1981; Russel and Hino, 1985), formation and accumulation of bacterial toxins and other rumen metabolites (Sugawara, 1971; Ivanov, 1979; Chodhuri, 1980; Remond *et al.*, 1993). Absorbed in blood, they cause morphological and functional hepatic injuries and impairment of protein, lipid and carbohydrate metabolism, as evidenced by the levels of blood total protein, blood glucose, total lipids and cholesterol.

The changes in those laboratory parameters on the background of the synthesis of thyroid and adrenal hormones could hardly be interpreted as primary ones.

The phasic dynamics of T3, T4 and cortisol allow ous to assume that their early changes most probably resulted from the nutritional stress whereas the later ones were probably subsequent to the triggering of mechanisms for compensation of already existing metabolic disturbances. The increased T4 and cortisol concentrations, related to the elevated blood sugar levels could be related to impaired glycaemia or the enhanced insulin degradation. At the same time, increased T3 and T4 levels could at their turn explain the lowered total lipid and cholesterol blood concentrations.

In conclusion it could be stated that the development of the chronic rumen acidosis accompanied by impaired hepatic function (manifested by the elevated blood sugar concentrations and decreased total protein, total lipids in cholesterol), revealed a direct influence (in early stages) as well as an indirect influence (in later terms) of the chain thyroid gland–adrenal glands.

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Correspondence:
Assoc. Prof. P. Georgiev,
Faculty of Veterinary Medicine,
Trakia University,
Student's Campus,
6000 Stara Zagora, Bulgaria

Metabolic acidosis, particularly its chronic form, is a systemic process with systemic consequences that includes disturbances in the function of some endocrine systems. Such disturbances likely have short-term adverse consequences such as less sensitivity to the actions of hormones that might require higher hormone levels, e.g., insulin, to obtain needed hormonal effects. These disturbances might also have long-term consequences, however, like the association of decreased insulin sensitivity with increased cardiovascular mortality. For example, because some studies support that metabolic acid... Respiratory acidosis can be acute or chronic; the chronic form is asymptomatic, but the acute, or worsening, form causes headache, confusion, and drowsiness. Signs include tremor, myoclonic jerks, and asterixis. Diagnosis is clinical and with arterial blood gas and serum electrolyte measurements. The cause is treated; oxygen (O_2) and mechanical ventilation are often required. (See also Acid-Base Regulation and Acid-Base Disorders.) Respiratory acidosis is carbon dioxide (CO_2) accumulation (hypercapnia) due to a decrease in respiratory rate and/or respiratory volume (hypoventilation). Causes of