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بعض التغيرات البيوكيميائية والهستوباثولوجيه المصاحبة لحموضة الكرش في الأغنـــام

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أجريت هذه الدراسة على عشرون رأس من الأغنام منها اثنتا عشرة غنمة مصابة بحموضه الكرش نتيجة لتناولها كميات كبيرة من الحبوب عرضا أما باقي الحيوانات فكانت سليمه ظاهريو واكلينيكيا واستخدمت للمقارنة .

اجريت التحاليل المعملية لعينات من الدم جمعت من الحيوانات المصابة والحيوانات السليمة ، كما أجريت الصفة التشريحية والفحوص الهستوباثولوجية على عينات من بعض الاعضاء (الكبد ـ الكلى ـ الرئتين والطحال) من ثلاثة حالات اثنتان منها ذبحت اضطراريا والثالثة نفقت قبل ذبحها وقد أوضحت النتائج مايلي:

ـ أسفرت تحاليل مكونات الدم عن وجود زيادة معنوية في مستوى كل من الصفراء في الدم الكرباتنين، بولينا الدم ، انزيمات (SGOT , SGPT and LDH) والبوتاسيوم بينما وجد نقص ملحوظ في مستوى كل من سكر الدم، أنزيم الفوسفاتيز القاعدي والصوديوم .

كما أوضح الفحص الهستوباثولوجي عن وجود مايلي:

* اضحلال في خلايا الكبد والكلى.
* أوديما وتمدد في الحويصلات الهوائية بالرئه .
* تضخم في الطحال ووجود حبيبات الهيموسيدرين به .

ويتضح من هذه الدراسة أن لحموضة الكرش أثر جانبي ضار على مكونات الدم والاعضاء الحيوية الهامة مثل(الكبد ـ الكلى ـ الرئتين والطحال)، كما وضح من التغيرات في نتائج مكونات الدم وثبت تأكيده من الفحوص الهستوباثولوجيه . ولهذا ينصح بالحد من كميات الحبوب عند تكوين العلائق كلما أمكن مع التغيير تدريجيا .

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SOME BIOCHEMICAL AND HISTOPATHOLOGICAL CHANGES ACCOMPANIED RUMEN ACIDOSIS IN SHEEP

(With 2 Tables and 4 Figures)

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SUMMARY

Twenty sheep were included in this study, 12 of which were suffering from rumen acidosis while the rest served as control.

Blood samples were collected from normal as well as diseased sheep for biochemical analysis. In addition to that, specimens from liver, kidneys, lungs and spleen were taken from two affected cases which were emergancy slaughtered and one died ase for histopathological examination. The obtained results were as follows:

- Biochemically, acidosis in sheep was accompained by significant increase in serum bilirubin, creatinine, urea nitrogen, serum transaminases, lactic dehydrogenase and serum potasium. On contrast blood glucose, serum alkaline phosphatase and serum sodium were significantly decreased.

- Histopathological studies revealed various degenerative changes were occurred in liver and kidneys. Lung oedema and emphysema, besides to lymphoid hyperplasia and haemosiderosis in spleen were also observed.

- From this study, it was concluded that rumen acidosis has an adverse effect on blood metabolites and vital organs as revealed from blood changes that was also confirmed histopathologically.

INTRODUCTION

Diseases of sheep whatever the causes, may constitute the main factor of direct and indirect losses which reduce the productivity of these animals. Acidosis is one of these diseases which affect production in sheep.

Acute rumen acidosis is a problem in ruminants resulting from excessive consumption of fermentable carbohydrates which causes metabolic disturbances and non-physiological reduction in ruminal pH due to production of lactic acid by alteration of ruminal flora from anerobic to aerobic acid producers (HUNGATE et al., 1952; GNANAPRAKASAM, 1970; HUSSIEN, 1974 and SLYTER, 1976).

DAIN et al. (1955) found that, the animals become fatally ill when the acidity reached pH below 4.5 with histamine production upto 70 ug/ml blood. Clinically, this syndrome is manifested by severe toxaemia, dehydration, complete stasis and high mortality rate (JENSEN, 1974 and BLOOD et al., 1979).

ROBINSON et al. (1951), JENSEN et al. (1954), JENSEN and MACKEY (1971) and Brent (1976) stated that lactic acidosis was accompanied by ruminitis, which in turn leads to liver abscesses because the causative microorganisms can cross the devitalized mucosa of the rumen, invade blood vessels and metastasize into the liver through portal circulation.

Biochemically, DIRKSEN (1965) mentioned that, rumen acidosis in cattle was accompanied by significant rise in blood urea nitrogen, SGOT, and serum bilirubin, while alkaline reserve, serum calcium and chloride showed tendency for decreases. PROCOS et al. (1970) stated that, sheep experimentally affected with rumen acidosis showed marked drop in plasma glucose level and insignificant changes in plasma electrolytes. Moreover, significant rise in blood urea nitrogen, serum transaminases, serum icterus index together with significant fall in blood glucose were observed in experimental rumen acidosis (NAURIYAL and BAXI, 1978).

EL-HAMAMSY (1979) recorded an increase in the levels of serum bilirubin, SGOT, LDH and alkaline phosphatase and decrease in serum chloride, potassium and blood glucose in buffaloes due experimental rumen acidosis.

DIRKSEN (1965) mentioned that, in rumen acidosis the most marked pathological changes occurred in rumen mucosa and kidney. Moreover experimental rumen acidosis in goats was accompanied by severe degenerative changes in liver, kidneys, oedema of the lung and congestion of the meningeal blood vessels, in addition to that neurophagia and slight perivascular infiltration of mononuclear cells were evident in crebral cortex (SEN et al., 1984).

The present investigation was planned to study the metabolic disturbances resulting from rumen acidosis and its correlation with histopathological studies.

MATERIAL and METHODS

This study was conducted on twenty sheep which were divided into two groups:

Group (1): Consists of 8 sheep clinically normal and used as control.

Group (2): Consists of 12 sheep showing signs of rumen acidosis with history of accidental overfeeding on grains. Animals in both groups were of both sexes, nearly of the same age and were parasitic free.

Blood samples for biochemical analysis were collected from both normal and diseased groups. Blood glucose was determined as by FOLIN and WU, (1920), while serum bilirubin, creatinine and urea nitrogen were determined according to the methods of MALLOY and EVELYN (1937), HUSDAN and RAPOPORT (1968) and MARCH et al. (1965), respectively. The methods adopted by REITMANS and FRANKEL (1957), CABAUD and WROBLEWSKI (1958) and BELFIELD and GOLDBERG (1971) were used for determination of serum transaminases, lactic dehydrogenase and alkaline phosphatase activity. Serum chloride (SCHALES and SCHALES, 1941), while serum sodium and potassium were determined by using EEL flame photometer and according to the technique adopted by OSER (1965).

Specimen from liver, kidneys lungs and spleen from three affected cases two from which were emergency slaughtered and one was died were taken and preserved in 10% formol saline. Section of 3 microns thickness were prepared from these organs, stained by Haematoxylin and Eosin and examined microscopically (CARLETONS et al., 1967).

Statistical analysis of obtained data were done according to the method described by SNEDECOR and COCHRAN (1967).

RESULTS

I- Biochemical changes :

The mean values for blood glucose, serum bilirubin, creatinine, urea nitrogen, serum enzymes in clinically normal sheep and those affected with rumen acidosis were recorded in table (1). It is evident that rumen acidosis was accompanied by significant increase in the levels of serum bilirubin, creatinine, urea nitrogen, serum transaminases and lactic dehydrogenase, while blood glucose and serum alkaline phosphatase showed significant decrease.

Table (2) revealed that hyponatraemia and hyperkalaemia was a prominant feature in affected sheep, although the decrease in the level of serum chloride was marked but insignificant.

Table (1): Effect of rumen acidosis on some liver and kidney function tests of sheep.

Blood parameter	group (1)	Group (2)
	Clinically normal sheep	sheep affected with acidosis
Blood glucose mg/100 ml.	61.40±2.15	45.80±3.02 **
Serum bilirubin mg/100 ml.	0.357±0.056	0.906±0.095 ***
Serum creatinine mg/100 ml.	1.63±0.08	2.46±0.15 **
Serum urea nitrogen mg/100 ml.	17.43±1.13	25.42±2.49 **
SGOT. RFU/ml.	44.57±4.31	78.71±4.96 **
SGPT. RFU/ml	16.43±1.57	30.57±2.50 **
Lactic dehydrogenase Units/ml.	764.00±52.31	1108.00±74.26 *
Alkaline phosphatase I.U/L.	80.43±7.62	56.29±5.04 *

SGOT = Aspartate amino transferase
SGPT = Alainine amino transferase
RFU = Reitman Frankel unit.

* , P <0.05
** P <0.01
*** P <0.001

Table (2): Serum electrolytes values in normal sheep and those affected with rumen acidosis.

Electrolyte		Group (1) Clinically normal sheep	Group (2) Sheep affected with acidosis
Sodium	meq./L.	142.57±7.32	121.14±6.98*
Potassium	meq/L.	4.67±0.18	6.34±0.25**
Chloride	meq./L.	105.28±5.06	96.57±3.32

* P/ 0.05 ** P/ 0.01

II- Pathological changes :

1. Liver: Macroscopically the liver appeared congested and enlarged in size with greyish areas on the surface. Microscopically, the hepatic cells showed variable degenerative changes as cloudy swelling. Other hepatic cells showed coagulative necrosis with slight leucocytic infiltration (Fig. 1).

2. Kidneys: Macroscopically, the kidneys appeard enlarged in size, paller in colour and soft in consistency, while microscopical examination revealed that, renal tubules showed various degenerative changes as cloudy swelling (Fig. 2).

3. Lungs: Grossely the lungs of affected sheep appeared enlarged and congested. Histopathologically the lungs showed areas of oedema with faint pink stained fluid in the air sacs and some R.B.Cs were seen in some air sacs. Allveolar emphysema was also seen in some areas of the lungs (Fig. 3).

4. Spleen: Grossly, the spleen appeared enlarged and dark red in colour. Microscopically lymphoid hyperplasia was evident. Haemosiderosis was also seen (Fig. 4).

DISCUSSION

I- Biochemical changes :

The values of blood glucose, serum bilirubin, creatinine, urea nitrogen, serum enzymes in clinically normal and acidotic sheep were recorded in table (1). It was clear from results in the table that, hypoglycaemia was prominant feature that occured in case of rumen acidosis affected sheep. This result was coincided with those obtained by ARMSTRONG and BLAXTER (1957) and PROCOS, et al. (1970). KRONFELD et al. (1959) concluded that in rumen acidosis the energetic efficiency of volatile fatty acids and glucose metabolism were diminished and this tends to lower the blood glucose level. On the other hand, significant increase was observed in the levels of serum bilirubin, creatinine, urea nitrogen, serum transaminases and lactic dehydrogenase in affected sheep. Similar results were reported in cattle or buffaloes due to natural or experimental rumen acidosis by DIRKSEN (1965), NAURIYAL and BAXI (1978) and EL-HAMAMSY (1979). MULLEN (1976) mentioned that, hepatic cell damage and liver dysfunction occurred in cattle being placed on grain ration even with control of daily intake and the dietary adaptation occurred in 2-3 weeks and this suggests in changes in serum bilirubin and enzymes activity. HUBER (1976) stated that lactic acidosis was accompanied by reduction in glomerular filtration rate and renal failure and consequently alteration in urea nitrogen and serum creatinine occurs.

Table (2) denotes the values of serum electrolytes in normal as well as in affected sheep. Normally these findings were in agreement with those reported by PROCOS et al. (1970). While RAKALSKA et al. (1973) observed similar decrease in serum sodium and chloride with an increase in serum potassium in cattle affected with severe acidosis. TELLE & PRESTON (1971) mentioned that ovine lactic acidosis was accompained by increase in the concentration of blood potassium, while serum sodium level was relatively constant. BLOOD et al. (1979) attributed alteration in serum electrolytes to dehydration and sequestration in the rumen.

II- Pathological changes :

The main pathological changes arrising from acidosis in sheep depends upon its effects on metabolism and the amount of toxin produced. The lesions which depended on the metabolic disorders were seen in liver and kidneys which mainly degenerative changes. These results

were in agreement with those of SABA (1966) and SEN et al. (1984). The lesions which depended on the toxins were localized in lung, and spleen which includes oedema and lymphoid hyperplasia. These results were similar to those obtained by SEN et al. (1984).

From this study, it is concluded that rumen acidosis has an adverse effect on blood metabolities and vital organs as revealed from blood changes and was also conirmed by histopathological examination.

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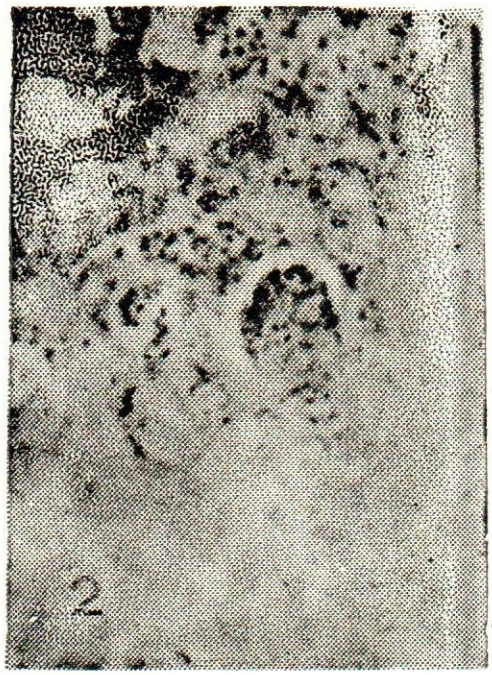
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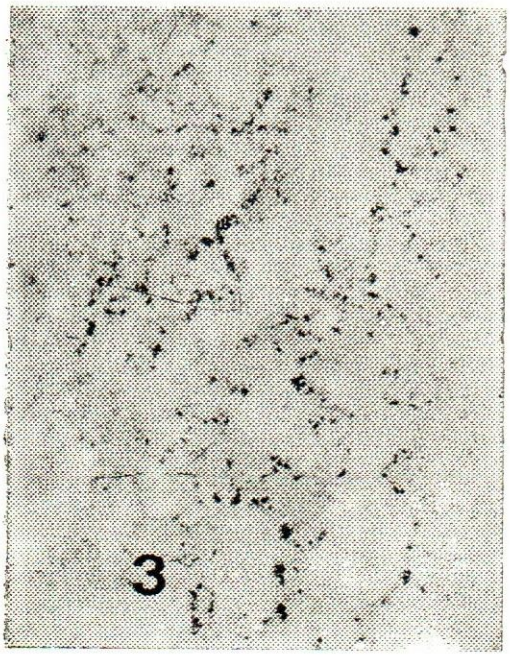
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