STUDIES ON RUMINAL DISORDERS IN SHEEP

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	ABSTRACT
Received at: 27/2/2014	Fifty two adult native breed Egyptian sheep were suffering from ruminal indigestion. The animals were divided into three groups, based on clinical signs
Accepted: 18/3/2014	and ruminal pH. The number of diseased cases with ruminal acidosis were 20, ruminal alkalosis 17 and those with ruminal tympany 15. In addition, 12 clinically adult sheep were used as control. Most cases with ruminal indigestion showed partial or complete anorexia, loss of body condition, abdominal pain, cessation of rumination and abdominal distension. Such signs differ according to the disease condition. Ruminal samples were collected from all animals and transferred to the laboratory for biophysical and biochemical analysis. All samples collected from diseased cases showed changes in the microbial activity as indicated by retardation in times of SAT, MBRT and CDT. Also, the values (m1) of GFT were increased especially in cases with ruminal tympany. All cases with ruminal dysfunction showed reduction in values and percentages of total and differential protozoal count. Biochemical analysis of ruminal fluid revealed variations in the values of sodium, potassium, chlorides and lactates among the types of indigestion.

Key words: Ruminal disorders, Sheep, Microbial activity, Ruminal fluid.

INTRODUCTION

Sheep industry constitutes a major part of animal production particularly in arid and semi-arid areas which represent more than 90% of the Egyptian land (Tawfik and Dighedy, 1980). Rumen disorders had a great clinical interest because of the high morbidity and the great losses in both production and costs of treatment of affected ruminants. From the economic point of view, fore-stomach diseases result in great losses to the producers through deaths, wasted feed, delayed marketing unthriftness of the recovered animals, incomplete utilizing of diseaseprovoking food and extra labor costs of preventive and therapeutic measures (Kimberlin, 1988).

The most common rumen dysfunction is usually defined as indigestion. The term indigestion is a general term for a group of diseases that describe mainly the reticulorminal dysfunction (Radostits *et al.*, 2000). Indigestion in sheep is an abnormal functional disturbance of the fore-stomach. Such a disturbance may be due to over-feeding with some feed stubbles, sudden change in the diet, consumption of indigestible roughages as well as oral dosing with sulphonamides and antibiotics (Blood and Radostits, 1989).

Such a group of diseases are of the most serious and common problems that threaten the animal heath in our country. Therefore, the main objectives of this study were to:-

a- Recording the clinical findings associated with each type of ruminal indigestion in sheep.

b- Evaluation the role of physico-chemical and microscopical characteristics of ruminal contents as a guide and tool for diagnosing ruminal dysfunction.

MATERIALS and METHODS

Location and date:-

Animals of the present work were belonged to different governomental and private farms at various areas around Qena city, Qena governorate, Egypt. The work was done during the period from October 2012 to July 2013.

Animals:-

A total number of 52 adult native breed sheep 1-3 years old of both sexes were selected from field cases suffering from rumen dysfunction. The diseased

animals were clinically examined. Animals were divided on basis of ruminal pH and clinical signs into three groups. The number of diseased cases with ruminal acidosis were 20, ruminal alkalosis 17 and those with ruminal tympany 15. In addition, 12 sheep of the same ages and both sexes and clinically healthy were used as control.

About 100 ml of ruminal juice was collected from each animal with the help of stomach tube and suction pump. The samples were transferred to the laboratory in a box containing dry ice for the immediate laboratory analysis. Each sample was divided into two parts of 50 ml. The first part was used for physical and microscopical examinations. The second part was strained using narrow sieve pores, then centrifuged at 4000 r.p.m for 15 minutes. The clear supernatent fluid was separated into clean plastic tubes and refrigerated at -20°C until biochemical analysis (Dirksen and Smith, 1987).

Methods:-

All animals were clinically examined according to Kelly (1984). Physical examination of ruminal juice was conducted according to Coles (1986). Total protozoa count in ruminal fluid samples was carried out according to Hungate, (1966).

The differential count of rumen protozoa was done using the same slide of counting using high power lens (x40). Identification was carried out microscopically and depending upon the size of cell type, location of cilia, macro and micro nucleus, skeletal plates, caudal spines and projection of cuticle (Hungate, 1966 and Williams and Coleman, 1988).

Biochemical analysis of ruminal fluids:a-pH:-

The pH was measured immediately after collection by using pH meter (Dirksen and Smith 1987).

b- MBRT and GFT:-

Methylene blue reduction (MBRT) and glucose fermentation (GFT) tests were done according to Coles (1986).

c- Sodium (mmol/l):-

The element was estimated using flame photometer (Model Jenway PFP7. UK).

d- Potassium, Chlorides and Lactates (mmol/l):-The elements were determined colorimetrically by using test kits supplied by GMbH Com. Germany.

RESULTS

1- Clinical findings:-

a- Ruminal acidosis (pH 4.6-5.5):-

The severity of clinical signs varied according to the degree of pH value of ruminal contents. Anorexia, dullness, depression, ruminal atony (1/2 min) or complete stasis with slight tympany and congested mucous membranes were recorded in diseased cases. A splashing or tinkling sound was heard on rumen auscultation. Most of diseased sheep showed yellowish white soft faeces or even diarrhea. Grinding on teeth was a prominent clinical sign in most severely diseased cases. The body temperature, heart, and respiratory rates ranged between 37.8-39.5°C, 79-115/min., and 39-75/min. respectively.

b- Ruminal alkalosis (pH 7.2-8.3):-

Diseased cases showed anorexia with yellowish green soft faeces or even diarrhoea without systemic changes. Slightly doughy ruminal contents on palpation, dull sound on ruminal percussion and ruminal atony (1/2 min) on auscultation were recorded. Inappetance, emaciation and complete ruminal stasis were recorded in most diseased sheep with high ruminal PH (7.8 - 8.1).

C- Ruminal bloat (tympany) pH (7.1-7.5):-

The most prominant clinical signs reported in diseased cases were severe distension of the left side of abdomen at flank region with increase or complete cessation of ruminal motilities. Animals lied down and get up frequently as signs of discomfort. Tinkling or ping sound was clear on rumen auscultation in such cases especially those with severe ruminal tympany. Most of diseased cases showed rapid respiration and heart rates (48-75/min., and 78-115/min respectively). Other signs as dysponea, congestion or cyanosis of m.m were seen in some cases.

2- Ruminal analysis:-

The obtained data of the studied parameters in ruminal contents were presented in tables 1,2,3 and 4.

	Clinically healthy	Types of ruminal Dysfunction			
Parameters		Acid Indigestion	Alkaline Indigestion	Ruminal Tympany	
Colour	-Olive green -Light to drak grey -green - Yellowish brown	-Milky.grey -Greyish brown	Light to dark brown	Green	
Odour	Aromatic	-Pungent -Sour	-Putrid -Ammonia -Offensive	-fermentic -Aromatic	
Consistency	Slimy	-Slimly -Aqueous	-Thin -Aqueous	-Slimy -viscous	
SAT (min)	6.7±0.26	41.8±0.32**	22.15±0.11**	16.4±0.75	
Protozoal activity	+++	O or +	O or +	O or +	

 Table 1:- Physical characters of ruminal juice in clinically healthy sheep and those suffering from ruminal dysfunction.

SAT= sedimentation and floation time

Table 2:- Mean values ± S.E of pH, MBRT, CDT and GFT in runinal fluids of clinically healthy sheep and those suffering form runinal dysfunctions.

		Types of ruminal Dysfunction				
Parameters	Clinically healthy	Acid Indigestion	Alkaline Indigestion	Ruminal Tympany		
Ph	6.54±0.02	4.8± ^{**} 0.11	$7.4 \pm^{*} 0.31$	$7.1 \pm^{*} 0.18$		
MBRT (min)	3.8±0.15	8.9± ^{**} 0.31	$7.5 \pm^{*} 0.05$	18.1±***0.91		
CDT (hrs)	26.4±1.2	42.5± ^{**} 3.2	36.7±*3.5	39.2± [*] 4.15		
GFT(ml)	1.7±0.01	1.9±0.04	$2.9 \pm^{*} 0.07$	3.8± ^{**} 0.03		

 $\begin{array}{ll} MBRT = Methylene \ blue \ reduction \ test \\ GFT = Glucose \ Fermentation \ test \ * = P < 0.05 \\ \end{array} \begin{array}{ll} CDT = cellulose \ digestion \ test \\ ** = P < 0.01 \\ *** = P < 0.001 \end{array}$

Table 3:- Mean values ± S.E and percentages for total and differential count of ruminal protozoa in clinically healthy sheep and those suffering from ruminal dysfunction.

Condition of animals (NO.)	Total	Holotrichs spp. Abs. No and %		Oligotrichs spp. Abs. No and %			
	NO./ml and %	Isotricha Spp.	Dayst-richa Spp.	Entod-inium Spp.	Epidi-nium Spp.	Diplo-dinium Spp.	Euodip- lodinium Spp.
Clinically Healthy (12)	532341± 86125	$25072 \pm \\8642 \\ (4.7\%)$	38352± 11425 (7.2%)	295023± 36721 (55.4%)	33139± 9548 (6.2%)	71231± 12671 (12.1%)	79524± 13752 (14.9%)
Ruminal Acidosis (20)	70254 ^{***} ± 8796 (13.2%)	0.0 ^{****} - (0.0%)	0.0 ^{***} - (0.0%)	63975 ^{***} ± 12013 (91.1%)	3520± 781 (2.20%)	1101 ± 1021 (4.4%)	1658± 920 (2.4%)
Ruminal Alkalosis (17)	$ \begin{array}{r} 198476^* \pm \\ 29125 \\ (37.3\%) \end{array} $	4383± 1709 (3.4%)	14561 ± 3998 (8.4%)	130561± 20109 (65.8%)	8723% 3257 (4.6%)	25721± 8972 (13.9%)	4527± 14277 (3.1%)
Ruminal Bloat (15)	$\begin{array}{c} 101381^* \pm \\ 40107 \\ (19.1\%) \end{array}$	$\begin{array}{c} 10540^{**} \pm \\ 2520 \\ (10.4\%) \end{array}$	$\begin{array}{c} 13721^{**} \pm \\ 3100 \\ (13.5\%) \end{array}$	$\begin{array}{c} 13782^{**} \pm \\ 4210 \\ (13.6\%) \end{array}$	$20321^{**} \pm 3972 \\ (20.0\%)$	25002 ^{**} ± 9011 (24.7%)	$ \begin{array}{r} 18015^{***} \pm \\ 5123 \\ (17.8\%) \end{array} $

 $\label{eq:prod} \ensuremath{^{*}}=P < 0.05 \qquad \ensuremath{^{**}}=P < 0.01 \qquad \ensuremath{^{***}}=P < \! 0.001$

Parameters		Types of ruminal Dysfunction				
	Clinically healthy	Acid Indigestion	Alkaline Indigestion	Ruminal Tympany		
Sodium	106.12±	81.01± ^{**}	109.34±	75.92±**		
(mmol/l)	3.56	2.65	2.31	3.11		
Potassium	13.07±	12.97±	13.98± [*]	$14.54 \pm^{*}$		
(mmol/l)	0.68	1.24	0.01	0.05		
Chlorides	22.09±	37.14±**	25.12±	22.89±		
(mmol/l)	0.72	2.05	3.41	2.01		
Tactates	0.18±	7.85± ^{**}	$0.07 \pm^{*} 0.01$	0.17±		
(mmol/l)	0.01	1.96		0.04		

Table 4:- Mean values ± S.E for biochemical analysis of ruminal fluids in clinically healthy sheep and those suffering from ruminal dysfunctions.

* = P < 0.05 ** = P < 0.01

DISCUSSION

A- Ruminal acidosis:-

1) Clinical findings:-

The reported clinical signs of animals suffering from ruminal acidosis were in accordance with Radostitis (2000), Braun et al. (1992) and Abdel Raouf and Omran (1986). The observed clinical signs were explained by the fact that acidosis led to depression of all centers in central nervous system on one hand, and disintegration of Gram negative ruminal flora on the other (Radosits et al., 2000). The observed diarrhoea in most cases could be attributed to the fact that lactic acid is converted to sodium lactate which passed down to the intestinal tract producing an osmotic gradient and draws water into small intestine contributing to diarrhoea (Blood and Radostitis, 1989). The increased respiratory rate with laboured respiration could be explained by stimulation of respiratory centers by lowed PH (Radostitis et al., 2000).

The amplitude and frequency of ruminal contractions were progressively diminished with eventual stasis when the rumen pH reached 5.0 or less. The mechanism of rumen stasis was due to the involvement of hydrogen ion receptors elsewhere in the gastro- intestinal tract and or the central inhibition of gastric center by the absorbed toxic amines and amides (Slyter, 1976).

2) Ruminal Contens:-

The recorded results (tables, 1 and 2) were agreed with those reported by Radostits *et al.* (2000) and Nikolov (2000). The milkey-grey colour of ruminal contents in diseased cases arrised from excessive feeding on a concentrated diet. The aqueous

consistency and sour smell of rumen contents was due to excessive production of lactic acid (Radostits *et al.*, 2000).

The times of SAT, MBRT and CDT reported in tables, 1 and 2 are used as guides to evaluate the activity of rumen microflora (Kimberling, 1998). The decreased rumen pH in diseased cases with rumen acidosis (tables 1.2) was due to increased lactic acid (Radostits, 2000). Microscopical examination of ruminal fluid indicated marked reduction in the activity and total count of ruminal protozoa (tables 1 and3). Highly significant decrease (p<0.001) in the value of total protozoal count and percent was reached 70254±8796, 13.2%. also, the Isotricha and Dasytricha were completely disappeared while Entodinium spp. predominated (91.1%) (Table 3). The total numbers and percentages of the other ruminal protozoa were greatly reduced (table 3). These changes were due to increased lactic acid production and hypertonicity (Radostits et al., 2000 and Nagaraja et al., 1992). Most of the ruminal protozoa are highly sensitive to increased acidity of ruminal content and cannot survive under pH 6.0 with exception of Entodinium spp. which can survive as it is acid resistant (Hungate, 1966). Biochemical analysis of ruminal fluid revealed a highly significant increase (p < 0.01) in the values of both chlorides and lactates associated with significant decrease in level of ruminal sodium (p<0.01) No change was occurred in ruminal potassium. The obtained results are in agreement with Nikolov (2003). The marked increase in the concentration of ruminal lactate could be attributed to its excessive production in the rumen by excessive grain feeding (Radostitis et al., 2000). The decrease concentration of ruminal fluid sodium could be explained by dilution of ruminal contents by the flow of fluids to the rumen (Nikolov, 2003). The marked increase of chlorides concentration in ruminal

fluid can be attributed to the reflus of hydrochloric acid from abomasum (Braun *et al.*, 1992).

B- Ruminal Alkalosis:-

1- Clinical findings:-

The clinical findings of ruminal alkalosis recorded in the present work were usually varied according to the changes of rumen pH (Nema *et al.*, 2003). The changes in rumen pH towards the alkaline side $(7.4\pm0.31$, table 2) may be attributed to sight overfeeding on concentrated feed or feeding on low quality and quantity of concentrated diet, or suddn change of diet that impede ruminal activity (Rodestits *et al.*, 2000).

2- Ruminal contents:-

The changes in colour, odour and consistency of ruminal contents in cases with rumen alkalosis (table 1) may be due inactivity of rumen microflora. Such phenomena are confirmed by the observed retardation time of SAT, MBRT and CDT (tables 1 and 2). The reported changes in their values with rumen alkalosis (table 2) in diseased cases is due to the increase rate of flow of saliva to the rumen which arised from continuous and prolonged feeding on rice straw with low quality of concentrated diet.

The sluggished protozoal activity and decreased count in ruminal alkalosis or acidosis could be attributed to the changes of rumen pH towards the alkaline or acidic side (Hungate, 1966).

Concerning the microscopical examination of ruminal fluid, it revealed a marked reduction in the activity and total number of ruminal protozoa (tables 1 and 3). Also, obvious decrease was reported among all types of ruminal protozoa (table 3). The lowest percentages were found among Isotricha spp. (2.20%) and Euodiplodinium spp. (2.3%). The reported lowering in the total and differential protozoal count may be attributed to changes in ruminal pH.

Table (4) revealed no changes in ruminal sodium and chlorides values while potassium increased and lactates decrease in diseased cases with ruminal alkalosis. The reported elevation in ruminal potassium content may be caused by the prolonged and continous feeding animals on fibrous diet leads to more salivation which in turn increase potassium level in ruminal fluid. The marked decrease of ruminal lactates in diseased cases (table,4) can be resulted from excessive feeding on poor quality roughage with low quantity of concentrated diet.

C- Ruminal Bloat (Tympany):-

1- Clinical findings:-

The reported distension at the left flank of diseased cases was due to fermentation of ruminal ingesta with production and accumulation of gases in rumen. Finally, the accumulated gases leaded to cessation of ruminal motility. The resulted ruminal tinkling or ping sound is due to ruminal atony and accumulation of gases in rumen.

2- Ruminal conents:-

The reported physical characters of ruminal contents in cases with bloat agreed with Majack *et al.* (1983). The reducing capacity of rumen microbiota was greatly reduced as indicated by MBRT. Increased volume of gases by GFT test indicated severe bloat in affected cases (table,2).

Microscopical examination of ruminal fluid (table 1,3), revealed marked reduction in the activity and total number of ruminal protozoa. Significant decrease (p<0.05) in total protozoal count and percent was reached 101381±40107, 19.1%. These results coincided with Hungate (1966), who attributed the change to the slight increase in ruminal pH. Marked decrease in the values of all types of ruminal protozoa was recorded. The lowest percentage was reported in Isotricha spp. (10.4%, table3). The bioehemical changes of ruminal fluid revealed higher potassium with lower values of sodium, while there is nonsignificant change in ruminal chlorides and lactates (table 4). In mild cases of ruminal tympany, the amount of saliva increased to the degree which could interfere with buffer capacity of rumen fluid and may act to elevate the pH and potassium contents in rumen, (Radostits et al., 2000).

CONCLUSION

It could be concluded without doubt that, improper feeding, gazing or feeding on field stubbles or bad quality roughages and feeding on decayed or putrified materials as garbage are among the important factors causing ruminal disorders in sheep.

With ruminal dysfunction most of body organs or systems will suffer from illness.

As the microfuna is important for ruminal digestion, thus keeping them alive and active is very essential.

The description of biophysical and biochemical integrities of ruminal juice are used as a guide for proper diagnosis of ruminal disorders.

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دراسات عن إضطرابات الكرشى في الاغنام

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أجريت هذة الدراسة على (٥٢) رأس من الاغنام المصرية والبالغة (١-٣ سنوات) والتي تعانى من إضطرابات إكلينيكية لوظائف الكرش. تم تقسيم هذة الحيوانات المريضة الى ثلاثة مجاميع بناء على الاعراض الاكلينيكية والاس الهيدروجيني لسائل الكرش (pH). لقد بلغ عدد الحالات المريضة وتعانى من حموضة الكرش (Rumen acidosis) ٢٠ حيوان ، بينما في قلوية الكرش (Rumen alkalosis) ١٧ حاله، وبلغت حالات نفاح الكرش (Ruminal tympany) ١٥ حالة. تم اختيار ١٢ راس رمن الاغنام البالغة والسليمة والصحيحة إكلينيكيا كمجموعة ضابطة (control group). لقد إختلفت الاعراض الاكلينيكية طبقا لدرجة حموضة الكرش. تمثلت أعراض حموضة الكرش في فقدان جزئي أو كلي للشهية مصحوبا بضعف او توقف حركة الكرش وسماع صوت طرق معدني، خمول او توقف عملية الاجترار ونفاح واحتقان في الاغشية المخاطية وزيادة في بعض معدلات التنفس وضرَّبات القلب. في حالات عس الهضم القلوى لوحظ فقدان في شهية الحيوان و هز ال مع ضعف في حركة الكرش. في حالات نفاخ الكرش ، لوحظ زيادة حركات الكرش في الحالات البسيطة ولكنها تتوقف تماما في حالات النفاخ الشديد مع توقف كلي لشهية الجيوان واحتقان شديد في الاغشية المخاطية. الخواص البيولوجية لسائل الكرش مثل اللون والرائحة والقوام والاس الهيدروجيني عرضة للتغير معتمدا على طبيعة ونوعية العلائق المستخدمة في التغذية. أوضحت الدراسة ان جميع حالات سوء الهضم مصاحبة بتغيير في النشاط البكتيري في الكرش وذلك من خلال التأخر في عملية الترسيب والطفو وكذلك تأخر القدرة الاختزالية لبكتريا الكرش سجلت النتائج نقصا ملحوظا في النشاط والكثافة العددية والنوعية لبروتوزوا الكرش في كل مجاميع الاغنام المريضة. اما جنس البروتوزوا Entodinium فقد ساد سيادة مطلقة عن باقى أنواع البروتوزوا في حالات حموضة الكرش بالاضافة الى زيادة أحناس بروتوزوا Epidinium, Diplodinium and Euodiplodinium في حالات نفاخ الكرش. سجلت إختلافات في قيم محتويات الكرش من الصوديوم، البوتاسيوم ، ألكلوريدات واللكتات معتمدا على الاس الهيدروجيني لمحتويات الكرش