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ACCIDENTAL UREA POISONING IN FATTENING BUFFALO-CALVES
(With One Table)

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التسمم العارض باليوريا في عجول التسمين الجاموس

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لوحظ تسمم باليوريا بين ٢٥ عجل تسمين جاموس نتيجة زيادة نسبة اليوريا في العلف المصنع (٤ر٢٪) وعدم كفاءة خلط اليوريا المضافة بمعدلات ثابتة . نفق ثلاث حيوانات بعد تناول العليقة فجأة في الحظائر وتم ذبح ستة آخرين بينما ظهرت أعراض التسمم على الباقيين التي شملت انخفاض أنشطة الحيوانات لفترة ثم تبعها ارتعاشات عضلية لاارادية وسيولة اللعاب وسرعة التنفس وعدم اتزان وانتفاخ الكرش ثم تبع ذلك انقباضات عضلية شديدة وإغماء . كانت التغيرات الكيميائية المميزة هي زيادة اليوريا في الدم (٣ر٧٨ + ٠٨ر مجم٪) ارتفاع نسبة الأمونيا في مصل الدم (٢ر٠٣٠ر مجم٪) والجلوكوز بنسبة (٦ر٩٥مجم٪) .

SUMMARY

Urea poisoning due to accidental access of large quantities of improperly mixed concentrate of high urea content (4.2%) was diagnosed in 25 fattening buffalo calves. Three animals died and 2 were emergency slaughtered. Dullness, followed by muscular tremors, excessive salivation, rapid respiration, incoordination, moderate bloat, finally convulsions and opithotonus were the significant clinical signs observed. The characteristic biochemical changes were increased blood urea (78.3±0.8 mg%), serum ammonia (1.2±0.03 mg%) and serum glucose levels (95.6±0.2 mg%).

INTRODUCTION

Urea is currently used as a cheap non, protein nitrogenous compound in ruminant ration, this usage arised from the ability of ruminal micro-organisms to hydrolyse urea to ammonia and carbon dioxide and reuse of ammonia for amino acid synthesis

(McDONALD, et al. 1981). PAYNE (1977) cited that the speed of microbial hydrolysis of urea is many times faster than the maximum rate of bacterial uptake of liberated ammonia and much of it passes through the ruminal wall especially in high pH to reach the liver. CALDOW and WAIN (1991) recorded that most of the absorbed ammonia is rapidly detoxified in the liver and urea poisoning can occur only if the rate of ammonia production exceeds both the consumption ability of the ruminal microorganisms and the detoxification capacity of the ruminal wall and liver.

Ammonia in contrast to urea is a toxic substance capable of causing encephalopathy in chronic poisoning and sudden death from respiratory and cardiac failure in acute intoxication (WORD, et al. 1969; CHO & LEIPOLD, 1977 and SMITH, 1990).

As a result of widespread use of urea in ruminant nutrition, the incidence of toxicity could occur periodically. Although many studies were conducted on experimental urea poisoning in ruminants (WORD, et al. 1969; MORRIS & PAYNE, 1970; SALLEM ABDULLAH, et al. 1986 and FRENANDEZ, et al. 1990), there were few reports on accidental urea poisoning (CHO & LEIPOLD, 1977). CALDAW & WAIN (1991) recorded an accidental urea intoxication with death of 17 of 29 suckler cows within six hours after the contamination of their drinking water with urea fertilizer.

This study represents the clinical and biochemical findings of acute accidental urea poisoning in fatening buffalo calves.

MATERIAL and METHODS

A twinty five steers out of 150 fattening buffalo steers 18-24 months old, their weights ranged from 400-500 Kg put in a separte yard in the animal farm station of the Faculty of Vet. Med., Suez Canal University, were observed to suffer from severe nervous manifestations thirty minutes after an evening meal related to a new lot of commercial concentrate mixture. Two of the affected calves died and three were emergency slaughtered in the yard, while the others were subjected to complete clinical investigations.

Blood samples were obtained from both affected animals and five healthy herdmates. The determination of serum calcium inorganic phosphrous, magnesium, urea, ammonia and serum glucose were carried out using test kits* after GINDLER and KING (1972); EL-MERZABANI, et al. (1977); GINDLER (1971); CHANEY and MARBACH (1972); FENTON (1962) and TRINDER (1969), respectively.

* Bio-Merieux, Mains. France.

Samples of the concentrate ration were taken for determination of urea concentration using test kit after CHANY and MARBACH (1962).

P.M. examination was carried out on the two emergency slaughtered animals and ruminal pH was measured using an electronic pH meter (Orion, SA, 720).

The statistical analysis were carried out using T-test according to KALTON (1976).

RESULTS

Clinical Findings:

After 30 minutes of the evening meal, five buffalo-calves were observed in the yard with severe dyspnoea and lateral recumbeney. A moderate bloat was noticed. Muscular tremors of all body muscles were obvious. Hyperaethesia, severe convulsions and opithotonous developed. Salivation and froth at the mouth were observed in some animals. Three animals died and two were emergency slaughtered.

One hour later, twenty animals appeared dull, aimlessly wandering and appeared to be sleepy. Incoordination and tremors of head & neck were firstly observed. The animals response to noise or sudden movements were exaggerated followed by convulsions, opithotonous and nystagmus.

Post mortum findings:

Gross examination revealed no significant changes and the pH of the rumen contents revealed levels between 6.8-7.9.

Laboratory findings:

Results of serum biochemical analysis are shown in table (1). There were no significant changes in the mean values of serum calcium, magnesium and phosphorus. Significant elevations of serum urea (78.3±0.8 mg%), serum ammonia (1.2±0.03 mg%) and serum glucose (95.6±0.2 mg%) were recorded in affected animals, in comparison with control group.

The urea concentration in the food sample were 4.2%.

DISCUSSION

The rapid onset of the clinical signs after feeding, which appeared from the clinical examinatiin and laboratory findings were diagnostic for urea poisoning.

The occurrence of clinical signs after about thirty minutes of feeding the concentrate diet, in strong heavy weight fattening buffalo-calves, suggested the cause to be accidental access to large quantities of improperly mixed or distributed diet rich in urea as a non protein nitrogen. This may be the cause of sudden production of large quantities of ammonia in the rumen and toxicity occured.

High urea concentration (4.2%) was recorded in the concentrates offered to these animals, and this confirms the occurrence of urea toxicity. BLOOD and RADOSTITIS (1989) and SMITH (1990) stated that urea concentration in ruminant feed must not exceeds 3%. These data emphasizes the presence of urea toxicity.

The clinical signs observed were similar to those described in experimental urea intoxication in steers (WORD, et al. 1969; BARTTEY, et al. 1976 and FRENADEZ, et al. 1990) and in accidental urea poisoning in suckler cows after the contamination of this drinking water with urea fertilizer (CALDAW and WAIN, 1991).

Although blood urea in systemic circulation don't have ill effect even in high concentration, the obtained high serum urea levels in affected animals may be attributede to the attempts by the liver to metabolise ammonia for reducing the poisoning (PAYNE, 1977 and LLOYD, 1986). A high serum urea concentration (10.6-13.3 m mol/l) was recorded by CALDAW and WAIN (1991) in four suckler cows with urea poisoning.

The obtained value of serum ammonia concentration of affected calves (1.2±0.03 mg%) was diagnostic of ammonia toxicity. WORD, et al. (1969) stated that toxicity symptoms appeared when blood ammonia nitrogen averaged 1.0 mg%. Furthermore, BARTTEY, et al. (1976) reported that clinical signs of urea poisoning appear in cattle when blood ammonia nitrogen concentration reaches 0.7-0.8 mg%. The pH of ruminal contents of two emergency slaughtered animals were 6.8 and 7.9. This was of diagnostic significance as urea ingestion can rise ruminal pH to above 7.5 (LLOYD, 1986 & BLOOD & RADOSTITS, 1989). The elevated peripheral ammonia concentration were the result of ruminal ammonia bypassing hepatic circulation and detoxification rather than the inability of the liver to metabolize incoming ammonia (BARTTEY, et al. 1981 and FRENANDEZ, et al. 1990).

The hyperglycaemia recorded in affected calves may be attributed to decreased glucose utilization by prepheral tissues (FRENANDEZ, et al. 1990) who added that, pancreatic insulin release tended to be inhibited by ammonia.

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Table (1) : Serum biochemical analysis in clinically healthy and fattening buffalo-calves with urea poisoning.

Parameter / Animal condition	Nr.	Calcium mg %	Inorganic phosph. mg %	Magnesium mg %	Urea mg %	Ammonia mg %	Glucose mg %
Diseased	20	9.4±0.6 N.S	4.7±0.3 N.S	3.9±0.2 N.S	78.3±0.08 **	1.2±0.03 **	95.6±0.2 *
		8.8-14.2	4.3-5.8	2.7-4.6	56.5-82.4	0.8-1.4	86.5-105.3
Clinically healthy	5	10.5±0.8	53,±0.2	3.2±0.1	32.5±0.9	0.56±.03	80.4±0.8
		9.0-13.5	4.0-6.3	2.5-4.2	18.6-37.0	0.31-.62	72.6-82.3

N.S. Non Significant
** Significant at P < 0.01
* Significant at P < 0.05