Asian Journal of Research in Animal and Veterinary Sciences

1(2): 145-150, 2018; Article no.AJRAVS.41386

Histopathological Finding in Urea Toxicity in Cattle

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Authors' contributions

This work was carried out in collaboration between all authors. All authors read and approved the final manuscript.

Article Information

DOI: 10.9734/AJRAVS/2018/41386
Editor(s):
(1) Dr. Jean-Marie Exbrayat, Professor, Reproduction and Comparative Development, University of Lyon, General Biology, Catholic University of Lyon, Ecole Pratique des Hautes Etudes, France.
Reviewers:
(1) Musa Zakariah, University of Maiduguri, Nigeria.
(2) Sunday Akau Hena, University of Jos, Nigeria.
Complete Peer review History: http://www.sciencedomain.org/review-history/25482

Case Report

Received 11th April 2018
Accepted 22nd June 2018
Published 10th July 2018

ABSTRACT

Aim: the aim of this paper was to investigate the histopathological changes in the liver, kidney and lung during urea toxicity in cattle.
Place and Duration of Study: In September 2015, Alslait South Dairy Farm, Khartoum, Sudan reported sudden death of some cattle occurred after fed wet concentrated feed prepared from molasses and urea.
Methodology: Clinical history and clinical signs were recorded. Among 12 cattle of affected shed, 9 (male=2, female =7) cattle showed clinical sign and within 2-3 hours of onset of clinical sign 7cattle were dead. Postmortem examination was done and liver, kidney, lung and feed samples were collected and sent for laboratory analysis.
Result: Results revealed that the male were less prone to be affected (16.7%) with no case fatality

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(0%) in comparison to highly affected female (50.7%) with very high case fatality (77.7%). On postmortem examination, congested liver and kidney, gastroenteritis with hemorrhagic intestine, edema of lung were observed. Histopathological results revealed necrosis of hepatic cells and renal proximal tubules with dissociation of hepatic cord and infiltration of inflammatory cells in the kidney, congestion of the pulmonary alveolar capillaries, bronchial haemorrhage and emphysema and interstitial pneumonia.
Toxicological testing was done on the supplied feed samples and non-protein nitrogen was calculated as 28.18%.
Conclusion: in Sudan, urea is used in urea molasses straw preparation as an effective and inexpensive source of non-protein nitrogen (NPN) in feed supplements in the ruminant. Cautions might be taken as urea poisoning may occur in ruminants when incorrect dose or feeds are inappropriately mixed with urea. The onset of the clinical picture may start in a matter of minutes to hours after consumption of urea and in most cases it is acute and can cause heavy mortality.

Keywords: Urea toxicity; cattle; histopathological finding.

1. INTRODUCTION

Urea poisoning is one of the more commonly suspected toxicities of ruminants especially cattle [1]. Dietary urea has been used for decades as an effective and inexpensive source of non-protein nitrogen (NPN) in feed supplements in the ruminant. The nitrogen from urea is released in the rumen as ammonia and can be used by rumen micro-flora to synthesize protein [2, 3].This protein then becomes available to the animal through the normal processes of digestion and absorption. However, if more urea is consumed than the rumen organisms can metabolize, the ammonia is absorbed from the rumen into the blood. The ammonia is then converted back to urea in the liver and is then excreted by the kidneys. This pathway can easily be overwhelmed, when excess ammonia and urea circulate in the blood, causing poisoning [4]. Poisoning may occur periodically when ruminants gain access to large quantities or are fed large amounts of urea; when they are not adapted to it or when feeds are improperly mixed or high urea concentration is present in low energy, low protein, and high roughage diets [3. Poisoning can occur rapidly from a few minutes to four hours after consumption. Suspect urea poisoning cattle are found dead [4].

In Sudan, urea is also used in urea molasses straw preparation in the diary production program. The objective of this paper was to investigate the histopathological changes in the liver, kidney and lung during urea toxicity in cattle.

2. PRESENTATION OF CASE

Alslait South Dairy Farm, Khartoum reported the sudden death of some cattle which occurred after wet urea concentrate feeding. Veterinarian in the local veterinary unit did postmortem examination for the dead animals and liver, kidney, lung and as well as feed samples were sent to Central Veterinary Research lab (CVRL), Khartoum.

2.1 Case History

The cattle were regularly fed with fodder along with concentrated feeds. On the day of case fatality, cattle were offered wet concentrated feed prepared from molasses and urea. After feeding, the cattle were found with labored breathing, respiratory distress, and salivation cyanosed mucus membrane (buccal cavity and tongue) mydriasis, salivation, convulsion and bloat. Among 12 cattle of the affected shed, 9 (male=2, female=7) cattle showed clinical sign and within 2-3 hours of onset of clinical sign, 7cattle were dead.

2.2 Post-Mortem Inspection

Postmortem examinations of dead cattle were performed and liver, kidney, lung and feed samples were collected and sent for laboratory analysis.

Laboratory tests: The various organs from dead animals and feed samples were collected by the veterinarian. Samples were packed properly within sterile zipper clip bag and submitted to pathology and toxicology laboratories, Central Veterinary Research lab (CVRL), Khartoum.

Non-protein nitrogen was determined in the feed according to the method described by Sandhu [(5]. Tissues for histopathological investigation were fixed in 10 % formaldehyde embedded in paraffin, cut in 5 µm thick sections and stained

with haematoxylin and eosin stain according to Bancroft and Gamble [6].

2.3 Finding

The male were less prone to be affected (16.7%) with no case fatality (0%) in comparison to highly affected female (50.7%) with very high case fatality (77.7%).

2.4 Postmortem Finding

On postmortem examination congested liver and kidney, frothy bloat in the rumen, gastroenteritis with hemorrhagic intestine and edema of the lung were observed.

2.5 Histopathological Results

Histopathological results revealed necrosis of hepatic cells with dissociation of hepatic cord, sinusoidal dilation and infiltration of inflammatory cells (Fig. 1).

Kidney showed necrosis of the cells of the renal proximal tubules, haemorrhage and infiltration of inflammatory cells (Fig. 2).

Lung showed congestion of the pulmonary alveolar capillaries, bronchial hemorrhage, pulmonary edema, infiltration of inflammatory cells and emphysema and interstitial pneumonia (Fig. 3).

2.6 Toxicological Finding

Toxicological test was done on the supplied feed samples, non-protein nitrogen were calculated as 28.18%.

3. DISCUSSION

It has been known for quite a long time that urea can be recycled and used as a source of nitrogen for the rumen microorganisms [7]. When high urea concentration is consumed by animals, the urea molecule is broken down into two ammonia units. Rumen and blood ammonia levels increase dramatically within 20-30 minutes of consumption [8]. The amount of urea included in concentrate mixtures for cattle or sheep should not exceed 3 percent and usually the addition of 1 to 1.5 percent will prove adequate [8] and it was approved that urea stops bacterial growth and fermentation in concentrations over 10% [8,9].

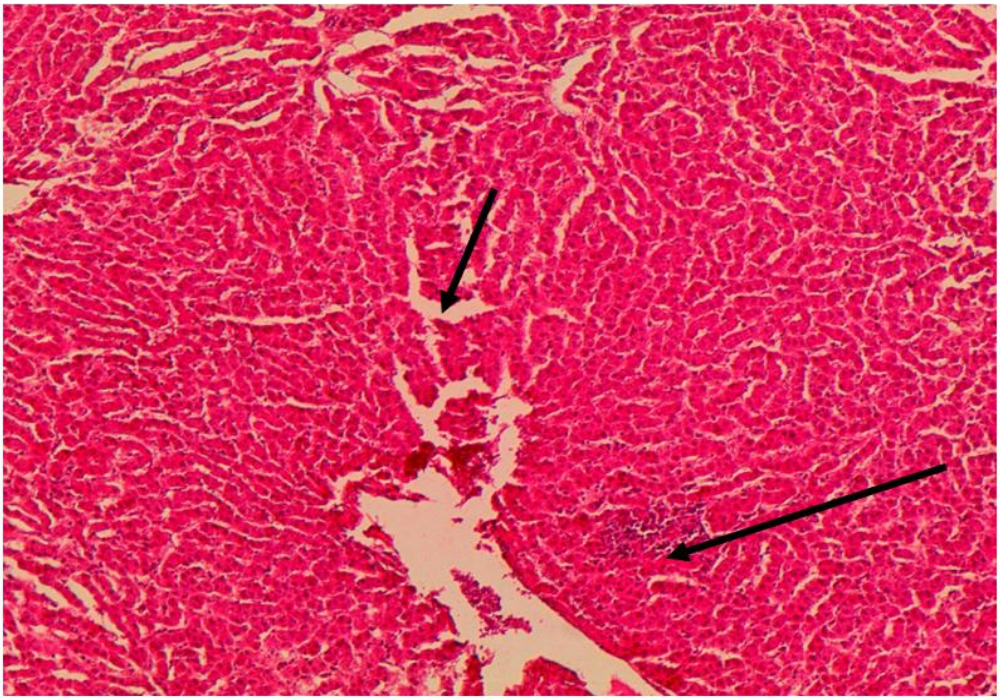

Fig. 1. Liver section showed sinusoidal dilation (Short arrow) and infiltration of inflammatory cells (long arrow). H & E X 10

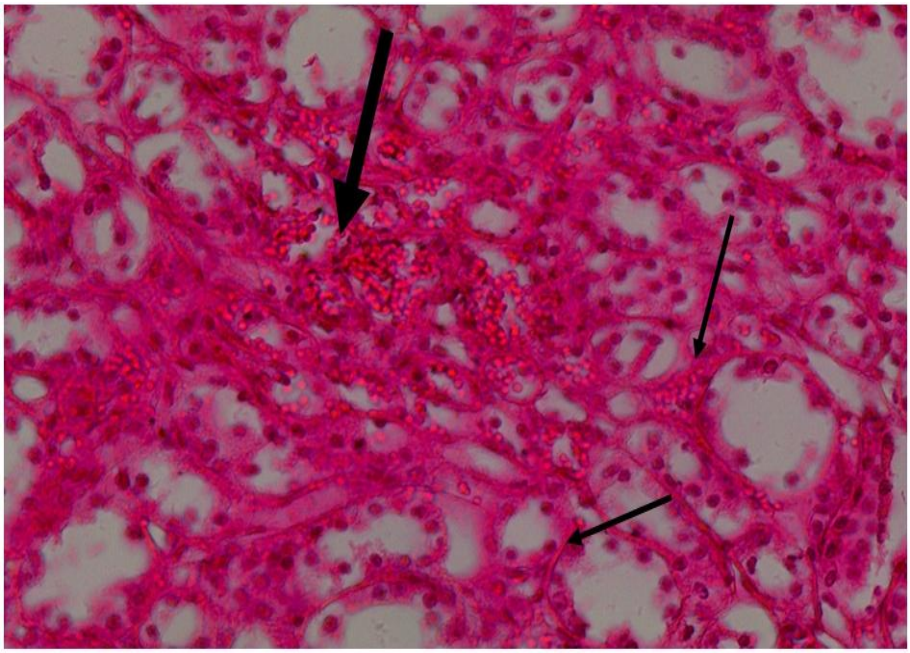

Fig. 2. Kidney section showed severe haemorrhage (thick arrow), necrosis and dilation of medullary renal tubules (thin arrows) H & E X 40

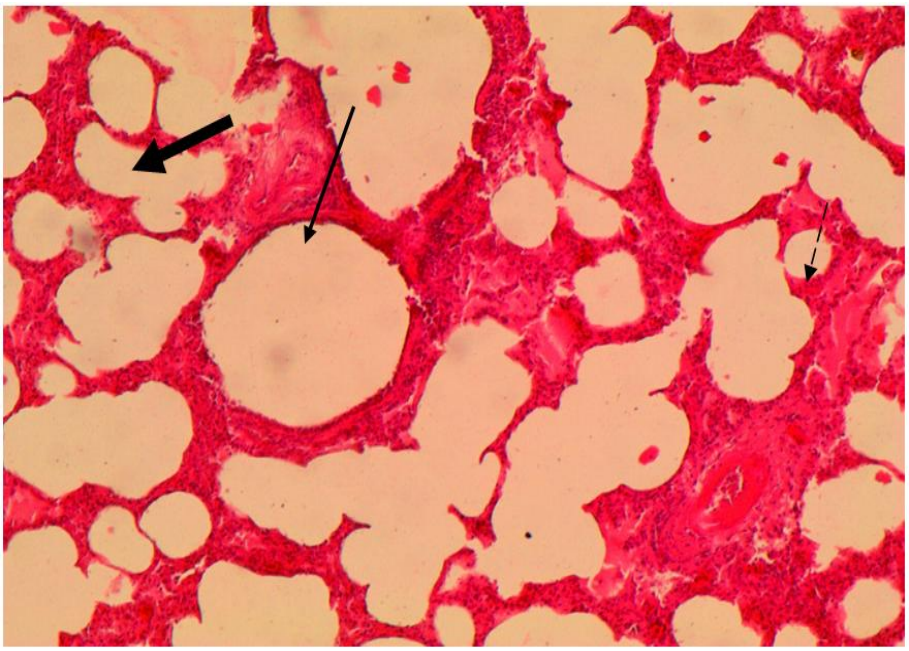

Fig. 3. Lung section showed emphysema (thick arrow), thickening of alveolar septa (thin arrow), edema and infiltration of inflammatory cells (dashes arrow) H & E stain X 40

Blood ammonia concentrations generally cause the toxicity problems, clinical signs of ammonia toxicity in animals are restlessness, dullness, weakness, loss of elasticity of the skin due to dehydration, high temperature, muscle tremors profuse salivation, rumen a tony, bloat, dyspnea, incoordination, vocalization, lung edema, tonic-clonic convulsion, and finally death by heart failure [8,9,10,11,12], which coincides with our findings. The male were less prone to be affected in comparison to highly affected female, this result was similar to that reported by Shaikat, et al. [13].

The presence of congestion of liver with some damage matched with findings of Horner [14], Sharma, et al. [3] who reported liver congestion and pericarditis. It is generally agreed that urea toxicity is equivalent to ammonia poisoning [15]. Ammonia poisoning prevents the release of carbon dioxide from the red blood cells while nitrites prevent the red blood cells from carrying oxygen to body tissue [3,14]. Toxicity problems are usually associated with the ingestion of excess levels of urea. The utilization of ammonia depends upon the growth rate of ruminal microbes and is usually limited by the availability of readily fermentable carbohydrates (i.e. grains).

In acute ammonia toxicity Rodrigue,s et al. [16] reported, the kidney showed hyper anemia, enlarged sinusoids within an apparently decreased amount of hematopoietic tissue, edema on tubular cells and tubular necrosis, and an enlarged Bowman's capsule. The liver presented dilatation of hepatic sinusoids, fatty deposition in hepatocytes and Mallory bodies.

In this study histopathological results showed congestion of the pulmonary alveolar capillaries, bronchial haemorrhage, pulmonary edema and emphysema and interstitial pneumonia similar to that results reported by Rodrigues et al. [16]. Latha, and Rajyasree [17] reported similar changes in birds exposed to urea for 30 days and showed degenerative and inflammatory changes in the liver, kidney and lungs.

4. CONCLUSION

A non-protein nitrogenous source such as urea has been used as feed additive for a long time in cattle feeding. The conventional and proper dose maintaining in the mixing of urea with a feed can be a handy and economical source of protein from non-protein nitrogenous substances for animals. The deliberate use of urea in cattle feed can be fatal and cause severe farm animal loss. So, farmers should have proper knowledge about the dose and method of urea supplementation in cattle feed and should be cautious enough in this situation.

CONSENT

It is not applicable.

ETHICAL APPROVAL

It is not applicable.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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Peer-review history:
The peer review history for this paper can be accessed here:
http://www.sciencedomain.org/review-history/25482