Case report: outbreak of sodium monensin intoxication in feedlot cattle from Mexico

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SUMMARY

In July 2010, 89 out of 800 calves (8–18 months old) from a feedlot located in the state of Tamaulipas, Mexico, exhibited severe oedema in inclined areas followed by sudden death. Hydrothorax, hydropericardium, pulmonary oedema, and enlargement of the heart and dilated ventricles with fatty degeneration were seen at necropsy. The liver showed severe diffuse congestion coupled to several necrotic foci and gallbladder distension. Histologically, severe diffuse fatty degeneration with vacuolisation and dissociation of the hepatic cords, multifocal necrosis and tissue infiltration with neutrophil and eosinophil granulocytes, cholestasis and fibrosis were observed in the liver parenchyma. Because of recycling of the concentrate containing sodium monensin from the past day to the next one, monensin intoxication was suspected. After complete concentrate removal from diet, no other case has occurred in this feedlot. These results showed that the improper use of adequate amount of monensin as a food additive can lead to a severe intoxication leading to acute cardiac failure in calves.

Keywords: Sodium monensin, improper use, outbreak intoxication, feedlot cattle, Mexico, cardiac failure.

RÉSUMÉ

En juillet 2010, 89 veaux âgés de 8 à 18 mois issus d’un même lot de 800 têtes implanté dans l’état de Tamaulipas, Mexico, ont présenté de sévères oedèmes des régions déclives puis sont morts brutalement. A l’autopsie, il a été noté la présence d’un hydrothorax et d’un œdème pulmonaire, d’un hydropéricarde et d’une hypertrophie du cœur dont les ventricules dilatés présentaient une dégénérescence graisseuse, ainsi qu’une sévère congestion diffuse du foie couplée à la présence de plusieurs foyers de nécrose et une distension de la vésicule biliaire. L’histologie a permis de mettre en évidence dans le parenchyme hépatique des lésions étendues et diffuses de dégénérescence graisseuse avec vacuolisation cellulaire et désorganisation des cordes hépatiques, des foyers de nécrose multifocaux, des infiltrations tissulaires par des neutrophiles et éosinophiles, une choléstase et des zones de fibrose. En raison du recyclage d’un jour à l’autre du concentré contenant le monensin, une intoxication par cet anticoccidien a été suspectée. Après le retrait complet de la ration, plus aucun cas ne s’est déclaré dans ce lot de veaux. Ces résultats montrent que l’utilisation inappropriée du monensin distribué comme additif alimentaire même à des doses non toxiques peut conduire à une intoxication sévère se traduisant par une défaillance cardiaque chez les veaux.

Mots clés : Monensin sodique, emploi inapproprié, intoxication, lot de veaux, Mexique, défaillance cardiaque.

Introduction

Sodium monensin, the carboxylic ionophore antibiotic produced by Streptomyces cinnamonensis, is widely used as food additive in order to improve the performance of both dairy and beef cattle, but decreasing the food intake [1, 10, 14,]. However, since the introduction as growth promoter in the 1970’s decade, accidental intoxication due to overdose, misuse and mixing errors in food preparation has been reported in bovines and other domestic species [1, 4, 5, 8, 10, 14].

The use of this ionophore has a wide margin of safety in cattle, because the adverse effects of sodium monensin have been reported with doses above 20 mg/kg of body weight [1, 14], which is higher in comparison with the LD₅₀ of 3 mg/kg and 12 mg/kg for horses and sheep, respectively [14]. Consistent lesions associated with sodium monensin intoxication in cattle are necrosis of both skeletal and cardiac muscle, with the consequent acute cardiac failure or chronic cardiovascular insufficiency [1, 6, 14, 18].

In Mexico, despite the fact that the beef production exceeded three million tons in 2009 [17], there are no reports concerning the amounts of sodium monensin used in feedlots or intoxication with this compound. Thus, this communication describes the findings related with an outbreak of suspected sodium monensin intoxication in feedlot cattle located in the Northeast of Mexico.

Case history and clinical findings

The reported outbreak occurred in feedlot cattle located in municipality of Hidalgo, in the state of Tamaulipas, Mexico. The feedlot consisted of 800 mixed-breed males, 8 to 18 months old, which were fed with a ration containing ground sorghum grain 70%, Buffel grass hay 17%, soybean meal 5.5%, sugarcane molasses 5% and vitamin-mineral premix (Ecox 200, Eco Animal Health, Mexico) 2.5%; fresh water was supplied ad libitum. Using the aforementioned vitamin-mineral premix, the animals were supplied with 386 mg/head/day of sodium monensin, this amount being considered within the
ranges of the LD50 for bovines, in agreement with several reports [1, 4].

In July 2010, 89 animals showed one or more of the following clinical signs during a 2 week long period: depression, weakness, progressive wasting, diarrhoea, severe oedema of the intermandibular, neck, brisket and ventral regions, followed by prostration and sudden death of all the 89 affected animals (mortality rate in herd: 11.13%). In order to establish a pathological diagnosis, tissues samples, i.e. liver, kidney, spleen, rumen, omasum, abomasum, heart and brain of the spontaneously died calves, were submitted to the Pathology Teaching Laboratory of the College of Veterinary Medicine at the Universidad Autónoma de Tamaulipas; in addition, four animals were euthanized and necropsies were carried out in the aforementioned pathology laboratory.

Pathological findings

During necropsy, it was observed that the subcutaneous tissue showed severe and diffuse oedema and focal haemorrhages. Hydrothorax, hydropericardium, pulmonary oedema, and enlargement of the heart and dilated ventricles with fatty degeneration (figure 1) were also noted. The liver was the most affected organ with several macroscopic alterations; i.e., changes in the texture from smooth to rugged, severe multifocal colour changes and severe diffuse congestion, a moderate thickness of the hepatic capsular surface and focal adherences, changes in the surface consistency from friable to hard, several necrotic foci, and also severe distension of the gall bladder (figure 2).

Histopathological findings were determined by staining the hepatic tissue with haematoxylin and eosin and examined under light microscopy after routine processing. There were severe alterations at hepatic parenchymal level, which included severe diffuse fatty degeneration with vacuolization, severe dissociation of the hepatic cords, moderate and focal cholestasis, severe and diffuse neutrophil and eosinophil infiltrations, multifocal necrosis and fibrosis (figure 3). The same lesions were also identified in the spontaneously died calves.

Diagnosis and treatment

Clinical signs and pathological findings suggested the presence of a toxic process and during the follow-up the diet was found to be the only common source for the outbreak. Despite the fact that the cattle was fed correctly concerning the daily intake of sodium monensin, the afternoon residual food was mixed with that for the next day, therefore progressively increasing the amount of sodium monensin in the concentrate; similar errors have been previously reported and considered as circumstantial evidence of sodium monensin intoxication.
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[8, 15]. Even though the exact intake of sodium monensin could not be determined, at this time any test for the diet was withdrawn because the source of the intoxication was determined as an error in the feeding process and the diet containing the suspected toxic concentration of sodium monensin was no longer available for analysis.

As consequences, the suspected diet was completely removed and the food recycling was suspended and the animals were fed only with Buffel grass hay. In addition, the most affected animals received a symptomatic and supportive treatment, consisting of oral administration of sodium bicarbonate (1 g/L), intravenous saline solution (3L per animal and per day) and, intramuscular injections of genabilic acid (Menbutone, 5 mg/kg) and sorbitol (Hepatonic, Virbac, Mexico, 25 mg/kg) each 48 hours until recovery. Finally, after the implementation of these measures, no other cases of sodium monensin intoxication were recorded in this feedlot.

**Discussion**

Sodium monensin is used in poultry, swine and cattle production in order to prevent coccidiosis and improve the performance of livestock; however, improper use of this compound could result in intoxication, either by ignorance or by accident [8, 12]. The clinical signs and lesions here reported are in agreement with those previously described for sodium monensin intoxication in cattle [7, 8, 12] and in lambs [4]. However, gross and histological lesions related to necrosis of skeletal and cardiac muscles were not evident in the four necropsied animals, probably because all cases were acute [16]; this condition has been reported for bovines [7, 18], and also in lambs [4, 11] and horses [3]. Even though laboratory assays are required in order to determine the sodium monensin intoxication, the hydrothorax, hydropericardium, subcutaneous and pulmonary oedema and liver congestion found during necropsy in the present study are lesions associated with cardiac failure [7, 9], which is considered as the main affected organ leading to the consequent clinical signs [4-6]. Furthermore, the outbreak follow-up determined that the source of this outbreak was the improper use of the sodium monensin into the diet, which is agreement with other reports [13, 15]. Concerning the mortality, the rate obtained here was higher than previously reported by SCHWEITZER et al. [15], but considerably lower when compared to the reported mortality for feedlot cattle by BASARABA et al. [1] or the mortality rates recorded in non-ruminant species [2, 10], including pre-ruminant bovines [6].

In Mexico, the use of sodium monensin as food additive is well documented by several authors. However there are no previous reports concerning intoxication of intensively farmed species with this ionophore. Thus, and to the best of our knowledge, this is the first report related with the toxic effects of sodium monensin in the country, in agreement with a literature

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**FIGURE 3:** Severe and diffuse histopathological lesions consistent with dissociation of hepatic cords and cholestasis (asterisks) (A.) and also severe and diffuse fatty degeneration (arrows) and diffuse neutrophil and eosinophil infiltrations (arrowheads) (B.) in a slaughtered calf suspected for monensin intoxication, Haematoxylin-eosin, 100 X.
search performed in the Network of Scientific Journals of Latin America and the Caribbean, Spain and Portugal (RE- DALYC), the United States National Library of Medicine (MEDLINE/PubMed), and the Web of Science (Thomson Scientific) databases. Finally, this report emphasizes the importance of the veterinarian role, not only as the animal science professionals, but also as advisers in the proper use of ionophores and intoxication surveillance.

Acknowledgment

The study was supported by the Universidad Autónoma de Tamaulipas under the project “Panorama del estado de la salud animal en México y sus diversas implicaciones”. The publication of this paper was supported in full by the Fondo Mixto de Fomento a la Investigación Científica y Tecnológica CONACYT - Gobierno del Estado de Tamaulipas (México). We thank Dr. Emiliano Salatino for their critically reading of the manuscript.

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