Sodium carbonate intoxication on a chinchilla (*Chinchilla lanigera*) farm: a case report

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ABSTRACT: Massive deaths were reported on a chinchilla (*Chinchilla lanigera*) farm, which over 10 months led to the elimination of the herd. After three months of feeding, longitudinal precipitates inside the pellets were noted. The first symptoms were observed two weeks after the introduction of feed, and included lethargy, decreased mobility, and reduced appetite, as well as increased reactions to external stimuli. Over time, single deaths, hair loss and gnawing, white discolorations on teeth, and polyuria were reported. Haemorrhages of the reproductive tract and mass abortions were observed. Necropsy revealed the presence of transudate with pH = 10, severe hyperaemia of the intestinal mesentery, and extensive regressive lesions in the gastric and intestinal mucosa. Both grossly and microscopically, lesions were noted in the liver, kidneys, adrenal glands, and lungs. The urinary bladder was overfilled. No lesions were observed in the spleen or mesenteric lymph nodes. Histopathology of skin specimens revealed atrophy of the hair follicles. Diagnosis was sodium carbonate intoxication. Owing to the specific features of this species, it was impossible to introduce emetic-based treatment in order to eliminate the toxic agent from the body. No therapeutic measures were undertaken because of the late diagnosis of the toxic agent and late removal of feed, as well as extensive lesions on the gastrointestinal mucosa.

Keywords: sodium carbonate intoxication; chinchilla

The digestive tract in the chinchilla is particularly sensitive (Cousens 1963; Merry 1990; Hoefer 1994; Jenkins 1992). Numerous bacterial alimentary infections have been reported in these animals (Moore and Greenlee 1975), although intoxication cases have been described only rarely (Dall 1963). There is no data on clinical cases of sodium carbonate poisoning in animals. This paper presents a case report of chronic intoxication with sodium carbonate on a chinchilla farm. Sodium carbonate was added to feed instead of sodium bicarbonate, which is used in the nutrition of chinchillas at a dose of 1 mg/kg as a sodium source when balancing dietary electrolytes.

Sodium carbonate is an irritating and caustic substance which causes injuries when it comes into contact with the mucosa (Busch et al. 1983; Grant 1986; Johnson and Swanson 1987). Due to its hygroscopic properties, sodium carbonate is prone to lumping, and under humid conditions it is transformed into sodium hydroxide (also known as "lye soda"). These properties intensify its harmful effects on the digestive tract (Clarke et al. 1981; Thomas and Stone 1994). In addition, this compound dissociates and alkalises the environment. The irritation results in reduced blood vessel tension, while the caustic effect in the digestive tract starts at a dose of approximately 3 g. The LD_{50} in rats with oral administration is 4.090 mg/kg body mass (Lewis 2004).

Case description

This case took into account documentation collected by the breeder and documents recorded by the veterinary surgeon, which covered a period of 10 months (i.e., from the herd's introduction to the farm until its removal). Physical examinations of the herd were performed at least once weekly throughout the aforementioned period. Gross examinations of dead animals were conducted during these visits.

During necropsies of the chinchillas, specimens of the skin, stomach, small intestine (duodenum), large intestine (colon), liver, kidneys, adrenal glands, and lungs were collected from three animals at the end of the third, sixth, and eleventh weeks and after seven and 10 months. The samples were fixed in 10% buffered formalin and the paraffin sections were stained with haematoxylin and eosin (Bancroft and Gamble 2008). During the necropsies performed in the eleventh week, the reaction (pH) of fluid accumulated in the abdominal space and pleural space and pH of feed and its precipitates were determined using the potentiometric method. At that time, a bacteriological examination of the feed was also performed.

Clinical case

A chinchilla (*Chinchilla lanigera*) farm with an inventory of 1330 animals reported widespread deaths that resulted in the elimination of the whole herd within 10 months.

The breeding material used on this farm originated from an authorised breeding herd. The average body mass of adult animals was up to 600 g/animal. The husbandry and breeding conditions on the farm were exemplary and complied with all veterinary requirements. The building was new, well illuminated, equipped with a very good ventilation system (ground air conditioning), and had a separate storage room for feed and straw. The cages in the rooms were arranged in four-stage rows. The humidity was 40% and the temperature was 18 °C. The animals had constant access to water and were fed with feed from a single manufacturer.

After three months of feeding, yellowish and cream-yellow longitudinal precipitates of 1 mm in diameter and up to 3 mm long were noted inside the pellets; these precipitates were initially thought by the manufacturer to be fragments of non-pelleted cereals. An organoleptic evaluation revealed that these precipitates saponified on the tongue to cause numbness and were tart in taste. A glistening, yellow, and vitreous-transparent dust was visible in the bags of feed; it left sediment on the hand and was clearly visible against a dark background.

Clinical symptoms

The first clinical symptoms in the chinchillas were noted two weeks after the feed had been introduced, and included lethargy, reduced mobility, decreased appetite, and increased reactivity to external stimuli. After three weeks, sporadic and sudden deaths were reported, and then tendencies toward hair loss and the gnawing away of hair were observed. In addition, the chinchillas' coats became dull, loosened, and were prone to loss. After 1 month, alopecia areata progressing to alopecia universalis was reported in some animals (Figure 1). Lack of hair was also evident in locations that could not be self-mutilated.

During the sixth week of breeding, white discolorations appeared on the teeth. Two months later (two months and three weeks after the introduction of the feed), the aforementioned symptoms became more severe; hair loss in particular was more pronounced. Polyuria was also reported; large pools of urine and undigested light-brown stool with mucus were visible beneath the cages together with large amounts of crumbled feed, suggesting that the animals had discarded the feed instead of consuming it. Bleeding from the reproductive tract and massive abortions were also reported. Neonates were born small, and lived only up to one week because the mothers did not produce milk. Animal behaviour (the clamping of teeth on the bars of the cages) indicated that the animals were dying in pain. At that point, 405 deaths had been reported, and a change of feed was introduced. However, deaths continued; 95 additional deaths were recorded in chinchillas younger than six months of age. In the subsequent month, it was concluded that the animals were unsuitable for further breeding because of emaciation, limited mobility, and described changes in the hair coat, and 460 of them were euthanised. An additional

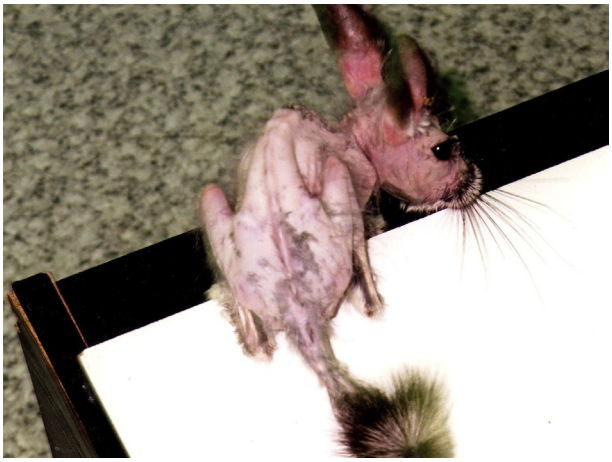

Figure 1. Alopecia universalis in a chinchilla. One month after introduction of the feed with sodium carbonate

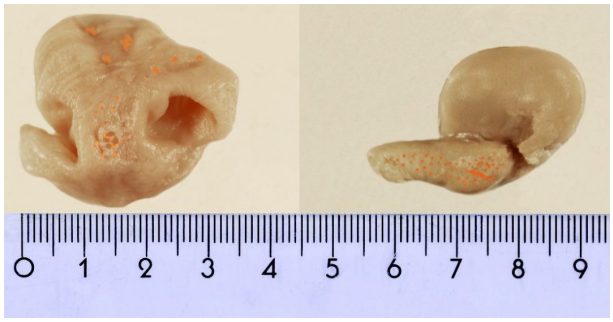

Figure 2. Haemorrhages and ulceration of the stomach mucosa (on the left) and under the adrenal gland capsule (on the right)

269 animals were euthanised 10 months after the introduction of the aforementioned batch of feed.

Morphological lesions

Transudate with pH = 10 was found in the abdominal space. Severe hyperaemia of the intestinal mesentery was noted.

Very deep defects in the gastric mucosa developed in several dozen cases (approximately 6% of the herd; Figure 2). Perforation of the gastric wall was sporadically observed. In addition, catarrhal and sometimes haemorrhagic effusions and numerous erosions were observed in this section of the digestive tract in the majority of dead animals. Grossly, the gastric mucosa was noticeably softened in the majority of cases. The small and large intestines were empty of contents. Occasionally, a small volume of liquid, with greenish content was observed in the large intestine. The small intestine mucosa was hypertrophic and showed signs of catarrhal inflammation. Histopathology revealed damaged tips and shortening of the intestinal villi in this section of the digestive tract.

The liver was enlarged, with compact texture and sub-capsular petechiae. Microscopic examination revealed parenchymatous degeneration, hyperaemia, and large areas of lipid infiltration and fatty degeneration in the liver, together with numerous small extravasations. Starting from the fourth month, necrotic foci were observed in animals that were found dead.

Enlarged and oedematous kidneys with parenchymatous degeneration (Figure 3), necrotic foci, and hyperaemia with extravasations were also reported. The adrenal glands were generally twice as large as the physiological norm (Figure 2) and microscopy revealed signs of hyperplasia and hypertrophy of the cortex with hyperaemia and extravasations (Figure 4) and vacuolar degeneration (Figure 5). The urinary bladder was overfilled. No lesions were noted in the spleen or mesenteric lymph nodes.

Transudate with pH of 8.5 was noted in the pleural cavity, and the lungs were oedematous and hyperaemic.

Histopathological examination of the skin specimens revealed normal layers with signs of hair follicle atrophy.

Chemical and microbiological examination of feed

Analysis of measured feed revealed that its pH ranged from 7.8 to 8.3, while the pH of precipi-

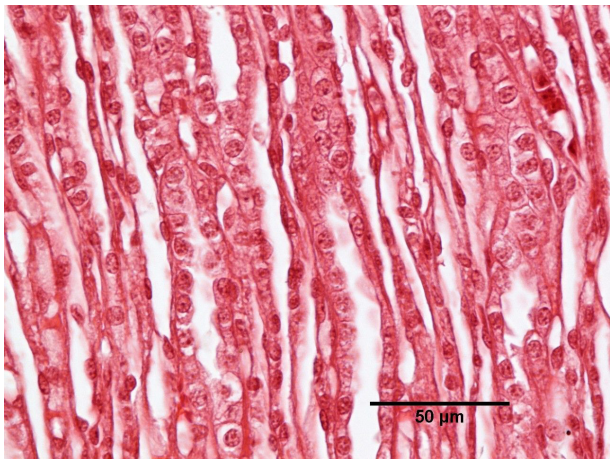

Figure 3. Chinchilla kidney with parenchymatous degeneration of the tubular cells; HE staining

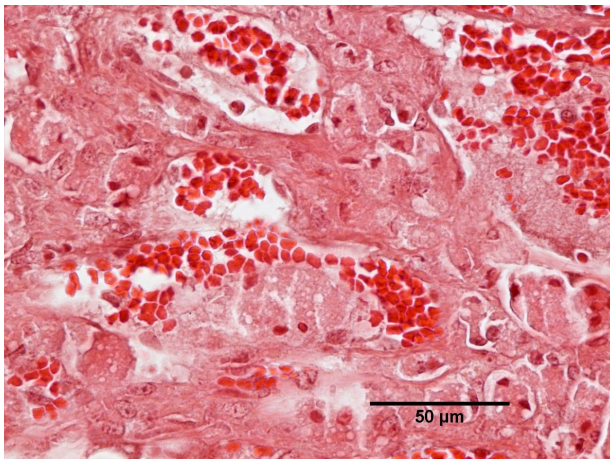

Figure 4. Chinchilla adrenal gland with hyperaemia, extravasations and necrotic foci; HE staining

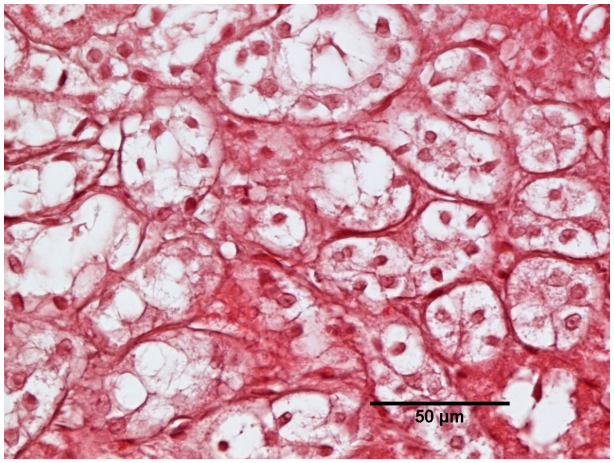

Figure 5. Necrotic foci and vacuolar degeneration of chinchilla kidney

tates extracted from the pellets was 10 to 10.5. The feed was also tested regarding the content of sodium in dry matter, which was 2.22% per DM. Microbiological examination did not reveal any pathogenic microorganisms.

DISCUSSION AND CONCLUSIONS

Even though chinchillas are thought to be relatively resistant to infectious diseases, bacterial infections have become increasingly important as intensive breeding and selection for production traits have resulted in an over-delicateness of many features in these animals (Crossley and Miguelez 2001; Baranowski and Wojtas 2011; Swiecicka et al. 2012). In the presented case, the evident lack of lesions in the spleen, large intestine, and lymph nodes excluded an infectious factor as a possible cause of morbidity and mortality. Furthermore, the microbiological investigations did not reveal any bacterial agent that could contribute to the sickness of the herd.

Chinchillas are extremely sensitive to the composition of feed mixture (Wolf et al. 2003). Symptoms and deaths are first seen in young nurtured animals and in pregnant and lactating females.

Because chinchillas are herbivorous, with a prolonged time span of digestion (Alworth and Harley 2012), feed remains in their digestive tract for three to six days. In addition, chinchillas swallow pellets immediately, practically without grinding; therefore, the aforementioned precipitates, encapsulated in the feed pellets as solid matter, did not exert a caustic or irritating effect on the oral mucosa. The lack of lesions in the oral cavity in the majority of animals and initially persisting appetite did not prompt them to refuse feed. The effects of this substance only became apparent in the stomach and small intestine. Sodium carbonate in a solid form, before it was dissolved in the gastric and intestinal juices, had an irritating and caustic effect on the mucosa resulting in the described lesions. The absorption of Na^+, OH^-, and HCO_3^- into the circulation was a toxodynamic factor with an effect on the whole body. The ions were generated in the gastric and intestinal juices from NaOH and Na_2CO_3 precipitates. These ions, present in large amounts, were distributed throughout the body, and disrupted the acid-base balance of the bodily fluids and depleted their buffer capacity, which led to the pH values of 8.5 to 10 (the physiological processes that normally take place in the gastric and intestinal fluids do not occur in this pH range).

Chinchillas have an excellently developed system of water absorption in the terminal section of the digestive tract, and they excrete a minor volume of urine (Alworth and Harley 2012). The excess of sodium resulted in some disturbances in the water-electrolyte balance, particularly an excessive excretion of urine and transudation of the fluids into the body cavities (pleural and peritoneal spaces). Sodium derivatives caused diarrhoea by irritating the mucosa of the digestive tract. The prolonged accumulation of NaOH and/or Na_2CO_3 particles resulted in a caustic effect and lysis of the tissues, and consequently in the perforation of the gastric wall and death. The excess of Na^+ also affected the blood (over-coagulability) and blood vessels (hyperaemia). Parenchymatous degeneration developed in the kidneys and liver. Hyperaemia of the mucosa, petechiae, disruption of tissue integrity, inflammation, and necrosis were the direct effects of the contact action of sodium solid derivatives observed in the digestive tract. These lesions caused chronic stress, which was confirmed by hyperplasia and hypertrophy of the adrenal medulla and hair coat mutilation (Rees 1963; Tisljar et al. 2002).

Because of the lack of acute symptoms of intoxication in the chinchillas and the specific features of this species, as well as the number of animals in the herd, it was impossible to implement any specific emesis-based treatment in order to eliminate the toxic agent from the body. The potential efficacy of administration of organic acids (citric, acetic, and ascorbic) was questionable because the diagnosis of the toxic agent and removal of feed happened

after a significant period of time, and injuries to the mucosa of the gastrointestinal tract were extensive. The presence of sodium carbonate in feed was most probably due to the human factor. As a feed additive destined for feed formula of other animal species it could have been added to the chinchilla feed by mistake during the production process in the company. The Polish Chinchilla Breeders' Association received similar reports regarding disturbances to chinchilla health from other chinchilla farms in the country at that time.

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