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Outbreaks of copper deficiency in ruminants in the semiarid region of Paraíba, Brazil

Surtos de deficiência de cobre em ruminantes na região semiárida da Paraíba, Brasil

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Abstract

Five outbreaks of copper deficiency in goats and one in cattle are reported in the semiarid region of the state of Paraíba, Northeastern Brazil. In four outbreaks of delayed enzootic ataxia, the goat kids showed weakness that progressed to paralysis of the four limbs and recumbency. Head tremors were also present. In another outbreak of congenital enzootic ataxia, the kids were born with paralysis due to congenital copper deficiency. The serum copper concentrations were below the reference values. Histologically, myelin degeneration was observed mainly in the spinal cord. In one outbreak of copper deficiency in cattle, adult cows exhibited chronic diarrhea lasting 6-8 months. The cows were supplemented with parenteral copper glycinate and recovered in 20-30 days after treatment. It is concluded that due to the occurrence of copper deficiency in goats and cattle, the supplementation of this mineral is necessary in grazing ruminants in the semiarid region of Paraíba.
Key words: Hypocuprosis, enzootic ataxia, swayback, diarrhea

Resumo

Cinco surtos de deficiência de cobre em caprinos e um em bovinos são descritos na região semiárida da Paraíba. Em quatro surtos de ataxia enzoótica os cabritos mostraram, após o nascimento, fraqueza evoluindo para paralisia dos quatro membros e decúbito permanente. Tremores de cabeça também ocorreram. Em outro surto, os cabritos nasceram com paralisia devida à deficiência de cobre. Os valores séricos de cobre estavam abaixo dos valores normais. Na histologia, degeneração da mielina foi observada, principalmente na medula. Em um surto de deficiência de cobre em bovinos, vacas adultas apresentaram diarreia crônica durante 6-8 meses. As vacas foram suplementadas parenteralmente com glicinato de cobre e se recuperaram em 20-30 dias após o tratamento. Conclui-se que, em consequência da ocorrência de carência de cobre em caprinos e bovinos, a suplementação deste mineral é necessária em ruminantes em pastejo no semiárido da Paraíba.
Palavras-chave: Hipocuprose, ataxia enzoótica, carência congênita de cobre, diarreia

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Copper (Cu) deficiency is one of the most common mineral deficiencies in ruminants raised on tropical pastures (OWEN et al., 1965; MORAES et al., 1999). In kids, Cu deficiency due to low Cu ingestion by the goats during pregnancy is manifested in two ways: delayed enzootic ataxia, which appears 3-26 weeks after birth and is characterized by progressive ataxia and weakness due to myelin degeneration; and congenital enzootic ataxia, in which the kids are born with clinical signs similar to those of delayed enzootic ataxia or, more rarely, with severe clinical signs of brain lesions due to myelin degeneration leading to porencephaly or hydranencephaly of the cerebral white matter (SUMMERS; CUMMINGS; DE LAHUNTA, 1995; UNDERWOOD; SUTTLE, 2010).

In cattle, Cu deficiency is responsible for weight loss, diarrhea, failure of hair pigmentation, anestrus, abortion, uterine infections, and sudden death (SUTTLE, 2010; RIET-CORREA et al., 1993).

In northeastern Brazil, low Cu concentrations were found in liver tissue from cattle in the state of Piauí with a chronic disease characterized by wheezing (TOKARNIA et al., 1968). Outbreaks of enzootic ataxia were diagnosed in sheep and goats in the state of Pernambuco (SANTOS et al., 2006) and in sheep in the states of Piauí (TOKARNIA et al., 1966) and Rio Grande do Norte (SOUSA et al., 2009). In the semiarid region of Pernambuco, marginal levels of Cu and Zn were found in the serum and liver of goats and sheep (MARQUES et al., 2011).

This paper reports the epidemiology, clinical signs and histologic findings in outbreaks of enzootic ataxia and congenital disease (swayback) due to Cu deficiency in goats. An outbreak of diarrhea associated with Cu deficiency in adult cows is also reported.

Data on the occurrence of the disease were collected by interviews with the farmers or during visits to farms where the disease was occurring. In all outbreaks the kids with signs of paralysis were sent to the Veterinary Hospital for examination. After clinical examination one kid of each outbreak was euthanized and necropsied. During necropsies samples of tissues of the abdominal and thoracic cavities, thyroid, skeletal muscles and the whole central nervous system (CNS) were fixed in 10% buffered formalin. After fixation the CNS transverse sections taken from the cervical, thoracic and lumbar spinal cord, medulla oblongata, pons, rostral colliculi, thalamus, internal capsule, cortex, cerebellar peduncles and cerebellum were examined histologically. Longitudinal sections of the spinal cord were also studied. All tissues were embedded in paraffin, sectioned at 4–6 µm, and stained with hematoxylin and eosin. Selected sections of the CNS were also stained with luxol fast blue for myelin. In two cases, serum and liver samples were sent to the Federal Rural University of Pernambuco for cooper analysis by atomic absorption spectrometry coupled to mass (ICP – Plasma) (MARQUES et al., 2011). An outbreak of Cu deficiency was diagnosed in the municipality of Patos during a visit to investigate chronic diarrhea in adult cows. Direct smears with *acid-fast* staining *were performed in samples from four affected cows.* To determine if the diarrhea was caused by Cu deficiency the cows with diarrhea were treated parenteral with one dose of 120 mg Cu glycinate (Glypondin ®).

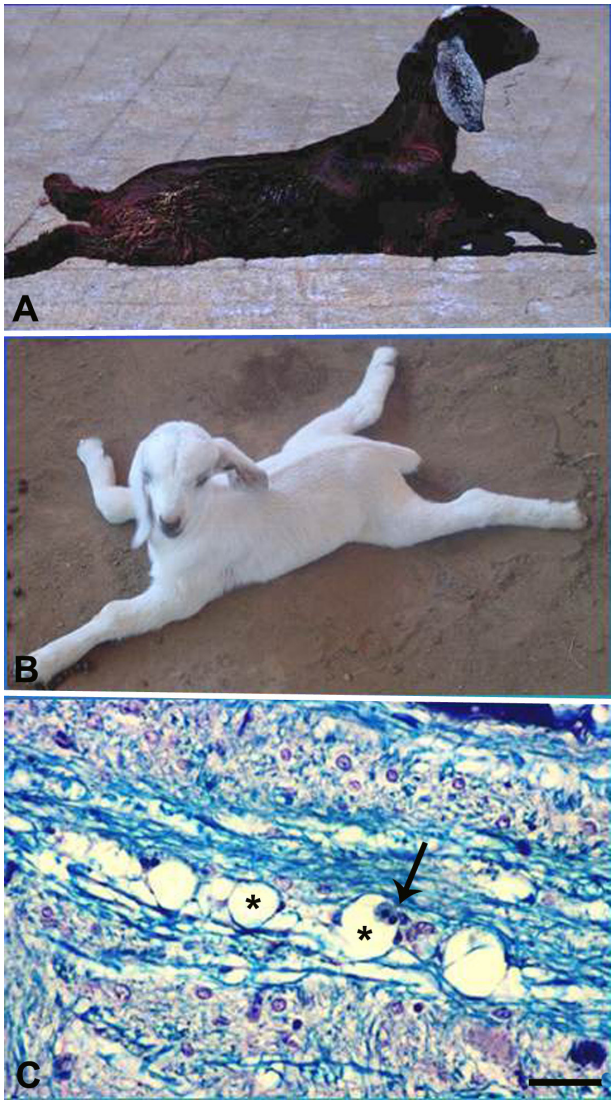
One outbreak of enzootic ataxia occurred in the municipality of Lagoa Seca in September 1998 affecting a 5-month-old crossbred kid. The farmer reported that other kids died previously, but did not inform the number. The second outbreak occurred in the municipality of Mogeiro, in September-October 1999, affecting 20 1- to 5-month-old crossbred kids. Another outbreak occurred in a herd in the municipality of Patos affecting 15 crossbred kids of approximately 30 days old, between May and June 2000. Those 3 outbreaks occurred in herds reared in native pastures (named *caatinga*). The fourth outbreak occurred from December 2005 to May 2006 in the municipality of São Sebastião do Umbuzeiro affecting 4 out of 33 Alpine goats that

in December, at the start of the outbreak, were 1- to 7-month-old. The herd was grazing in a pasture of *Cenchrus ciliaris*. The farmer informed that one of the goats affected in May 2006 was a 12-month-old goat that showed clinical signs since more than one month before. In the 4 farms all affected kids died or were euthanized and necropsied.

One case of congenital enzootic ataxia was observed in the municipality of Patos in a herd of goats of the Savana breed. The farmer informed that the goats had been purchased recently and could not say whether similar cases had happened in the herd earlier. All herds with delayed or congenital enzootic ataxia were not receiving adequate mineral supplements.

The goats initially showed weakness or spasticity and ataxia of the hindlimbs, followed by paralysis of the four limbs, causing recumbency (Figure 1A). Head tremors were also present. In one case, the animal had bruxism, decrease of pupillary reflex, absence of anal reflex, swallowing and purulent nasal discharge. One 12-month-old goat chronically affected showed bilateral atrophy of the hind limb muscles. In one outbreak of enzootic ataxia, serum Cu concentrations in two kids were 1.61 and 1.29 µmol/L, which were below the normal range of values (9.24-23.6 µmol/L) for the species (SMITH; SHERMAN, 2007).

Figure 1. A and B) Kids with enzootic ataxia (A) and congenital cooper deficiency (B) showing flaccid paralysis of the four limbs. C). Histology of the spinal cord showing Wallerian-like degeneration of the white matter characterized by the presence of vacuoles ordered in chains (*), some of them containing macrophages (arrow). Luxol fast blue, bar=50µm.



Source: Elaboration of the authors.

The kid affected by congenital deficiency exhibited, immediately after birth, marked incoordination and difficulty of standing for long periods and later became recumbent with flaccid paralysis of the four limbs (Figure 1B). On radiographs, the kid was found to have generalized bone rarefaction in the vertebral column, limbs and joints. The metaphyseal cortex of the long bones showed greater radiolucency and was thinner than normal, showing osteoporosis in the distal femur and humerus. The serum Cu concentration (6.43 mol/L) was below the normal range for the species.

No significant lesions were found on necropsies of the five affected kids. Histological findings were similar in all goats. Some neurons in the spinal cord, and with lower frequency in the brainstem, were eosinophilic, with diffuse chromatolysis and sometimes with a marginalized nucleus. In the spinal cord, mainly in the ventral funiculi, the white matter showed Wallerian-like degeneration that was characterized by the presence of vacuoles, sometimes ordered in chains, and occasionally containing macrophages or axonal residues (Figure 1C). Those lesions are characteristic of enzootic ataxia due to Cu deficiency (SUMMERS; CUMMINGS; DE LAHUNTA, 1995).

In 2003 in the municipality of Patos, in a herd of 25 Holstein crossbred cattle, four adult cows showed chronic diarrhea lasting 6-8 months. The cows were grazing in a *Cynodon dactylon* (Tifton grass) pasture. Fecal smears were negative for acid-fast bacteria. After treatment with parenteral Cu glycinate, three cows recovered in 20 to 30 days and one died one week after treatment. In this case, the diagnosis was made on the basis of the response to Cu supplementation. After the recovery of the affected cows the farmer started to use mineral supplementation containing Cu and no more cases of diarrhea were observed in adult cattle.

The results of this study indicate that Cu deficiency occurs in ruminants in the state of Paraíba. The observation of outbreaks of neurological disease in young goats, with clinical and pathological characteristics of Cu deficiency in this species and low levels of Cu, confirmed the occurrence of enzootic ataxia, a disease already reported in other states of the Brazilian semiarid region, including Piauí (TOKARNIA et al., 1966), Rio Grande do Norte (SOUSA et al., 2009), and Pernambuco (SANTOS et al., 2006). In these cases, the clinical signs are due to Cu deficiency in late pregnancy, which leads to a failure in the second stage of myelination of the spinal cord in neonates a few weeks after birth, mainly between 2 and 4 months old (SUTTLE, 2010), but the disease can occur up to 26 weeks of age (SUMMERS; CUMMINGS; DE LAHUNTA, 1995). The observation of the disease in a 12-month-old goat in one outbreak in the municipality of São Sebastião do Umbuzeiro was probably due to the fact that this was a surviving goat in an outbreak that started 5 months before.

Cooper deficiency can be primary due to low concentration of Cu in the pastures or secondary due to ingestion of high concentrations of molybdenum (Mo), iron (Fe) or sulfur (UNDERWOOD; SUTTLE, 2010). In goats and sheep in the states of Pernambuco (MARQUES et al., 2011) and Paraíba (SILVA, 2014) Cu concentrations in serum and liver were low or marginal and concentrations of Fe and Mo were within normal or marginal ranges suggesting that, in these states, Cu deficiency is primary.

The congenital form of Cu deficiency, observed in one outbreak, had not been previously diagnosed in goats or sheep in Brazil. This congenital form is due to extreme Cu deficiency of the fetus in the last 2 months of pregnancy, which results in the occurrence of the disease in neonates (SUTTLE; FIELD, 1968). The marked incoordination and difficulty of standing for long periods is characteristic of congenital Cu deficiency in kids, which can also exhibit flaccid or spastic paralysis in all four limbs, resulting in a total inability to walk and in death latter (UNDERWOOD; SUTTLE, 2010). Radiographic findings of irregular radiolucency

patterns of the long bones may result from the failure of endochondral ossification, which causes retarded growth, decreased weight gains and gait alterations. Changes related to bone rarefaction caused by generalized osteoporosis due to depression of osteoblastic activity are also observed in cattle and sheep with severe Cu deficiency (SUTTLE, 2010).

Chronic diarrhea was the only clinical form of Cu deficiency observed in cattle in this survey. This form of the disease has not previously been reported in Brazil. According to Ward (1978), animals with severe Cu deficiency usually have a rapid and effective response to supplementation, as was observed in the cows with diarrhea after the treatment with Cu glycinate. This diagnosis draws attention to the need to include Cu deficiency in the differential diagnosis of chronic diarrhea in cattle in the Brazilian semiarid region. It has to be differentiated mainly from paratuberculosis, which is a common disease in the region (MEDEIROS et al., 2012).

During the period of this study, no outbreaks of disease caused by Cu deficiency were observed in sheep, which are reared in the same areas and similar conditions as goats, suggesting greater susceptibility of goats to the disease, as observed by Santos et al. (2006). It is known that levels of Cu stored in the liver of goats are 10 times lower than in other ruminants (MESCHY, 2000). Thus, goats are more susceptible to Cu deficiency, mainly pregnant females with twin pregnancies, which are more prone to produce kids that will be affected by swayback or enzootic ataxia (MESCHY, 2000).

The occurrence of outbreaks of different diseases caused by Cu deficiency in the semiarid region of Paraiba indicates the need for Cu supplementation in grazing livestock and mainly in pregnant goats.

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