



## Communication

[Comunicação]

### Poisoning by *Enterolobium contortisiliquum* pods in calves showing photosensitization and digestive lesions

[Lesões digestivas e fotossensibilização em bezerros intoxicados pelas favas de *Enterolobium contortisiliquum*]

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A number of *Enterolobium* species including *E. contortisiliquum* (Grecco *et al.*, 2002; Mendonça *et al.*, 2009), *E. gummiferum* and *E. timbouva* (Tokarnia *et al.*, 2012) are toxic to cattle causing digestive signs, abortions and hepatogenous photosensitization. Spontaneous intoxication by *E. contortisiliquum* also occurs in goats and is associated with digestive disorders or abortions, but without photosensitization (Benicio *et al.*, 2007).

*Enterolobium* spp. are well-developed trees, and intoxication occurs by ingestion of the pods that fall spontaneously to the ground or fall along with twigs that are torn by the wind, are pruned, or are reached by the animals in the lower parts of the tree (Grecco *et al.*, 2002; Mendonça *et al.*, 2009). Digestive disorders with or without photosensitization were experimentally reproduced by feeding cattle (Mendonça *et al.*, 2009, Leal *et al.*, 2017), sheep (Leal *et al.*, 2017; Bacha *et al.*, 2017; Pupin *et al.*, 2017) and goats (Benicio *et al.*, 2007) with the pods of *E. contortisiliquum*. The reported digestive clinical signs of *E. contortisiliquum* poisoning are usually described as non-specific and include diarrhea, anorexia, ruminal atony, apathy, and dehydration (Grecco *et al.*, 2002; Mendonça *et al.*, 2009).

Recently, based on experimental studies done in sheep fed with the pods of *E. contortisiliquum* at our laboratory (Bacha *et al.*, 2017; Pupin *et al.*, 2017), it was determined that such digestive signs results from ruminal acidosis due to the high

content of non-fiber carbohydrates in the pods. Bromatological analysis of pods from *E. contortisiliquum* (Bacha *et al.*, 2017) revealed 1:2.2 ratio between neutral detergent fiber corrected for protein and non-fiber carbohydrates, which is much higher than the 1:1 recommended ratio (Poore *et al.*, 1993) to prevent depression in fiber digestion and preserve normal ruminal function. These results support the hypothesis that fermentable carbohydrates (starches and sugars) in the non-fermentable carbohydrate fraction of *E. contortisiliquum* pods may induce acute lactic ruminal acidosis.

Additionally, the gross and histopathological ruminal changes were characterized. The characterization of the digestive lesions of *E. contortisiliquum* poisoning as resultant from ruminal acidosis was yet unreported until the experiments performed by Bacha *et al.* (2017) and Pupin *et al.* (2017). The aim of this paper is to describe an outbreak of photosensitization and digestive lesions suggestive of ruminal acidosis in calves associated to ingestion of *E. contortisiliquum* pods in order to further confirm previous experimental studies.

The outbreak occurred in a farm in a small municipality (21° 41' 56" S 57° 52' 58" W) of the midwestern Brazilian, state of Mato Grosso do Sul, in October, 2018. There were 238 female and male calves aged 12-15 months, in poor body condition, raised in *Brachiaria brizantha*

pasture which contain lots of *E. contortisiliquum* trees in fruiting stage and others invasive plants. Besides the forage, the animals received only mineral supplement. Two days after the calves were introduced to this pasture, the clinical signs started and thirty-four of those got sick (morbidity rate of 12.7%). The first manifestation observed was subcutaneous edema of the ventral jaw and neck that progressed to erythema and skin necrosis. Focally extensive red dermal areas (Figure 1A) was observed in portions where necrotic skin sloughed (Figure 1B).

These were interpreted as lesions of photosensitization (photodermatitis) and were

typically located in the ventral aspect of the neck, dorsum, bilaterally in the flank and the posterior and medial aspect of both hindlimbs. The lesions varied in intensity among affected calves, and some calves had injuries in almost the whole skin surface. Some calves developed greenish malodorous diarrhea and drooling. Within 3-23 days from the onset of the clinical signs, fourteen calves died (mortality rate of 5.2% and a case fatality rate of 42.1%). Two out of six adult dairy cows that were kept in the same pasture showed signs of photosensitization and recovered without any treatment. Even after the onset of the clinical signs, calves were not removed from the pasture.



Figure 1. Poisoning by *E. contortisiliquum* pods in calves showing photosensitization and digestive lesions. A. There are focally extensive red areas of sloughed skin. B. Portions of necrotic skin are sloughing.

The authors made two on-site visits to the farm to collect data on the outbreak. Two calves were euthanized *in extremis* after being ill for five and 25 days, respectively. Necropsy findings were similar in both necropsied calves. They were in poor body condition and markedly dehydrated. There was paleness of conjunctiva and oral mucosa. Extensive alopecic, red and crusty areas of the skin were seen especially in the ventral aspect of the body. The rumen was moderately filled with dry pasture and there were numerous well-demarcated ulcers of 2-4cm at the dorsal sac and ruminal pillars, with raised borders, and covered by yellow fibrillar material (Figure 2). The abomasal mucosa of one of the calves was similarly ulcerated (Figure 3), and *E. contortisiliquum* seeds was seen admixed with abomasal contents. Fragments of the abomasum, encephalon, forestomachs, Gasserian ganglia, heart, kidney, liver, large intestine, lung, pituitary,

rete mirabilis, skin, small intestine, and spleen were collected in buffered 10% formalin and routinely processed for histopathology.

Histologically, the ruminal ulcers consisted of a deep base of granulation tissue covered by neutrophils, fibrin and cell debris (Figure 4). The abomasal lesions were similar but moderate. In the skin, there was coagulative necrosis of the epidermis with detachment and loss of most of the keratinocytes; numerous viable and degenerate neutrophils and fibrin covered the portions of skin deprived of the epidermis, forming a serocellular crust. Multifocal intraepithelial pustules occurred at sites where the integrity of the epidermis was conserved. There was no apparent edema of the underlying dermis but rather fibrovascular proliferation (granulation tissue). All the other organs were macroscopically and microscopically unaffected.

*Poisoning by Enterolobium contortisiliquum...*



Figure 2. Poisoning by *E. contortisiliquum* pods in calves showing photosensitization and digestive lesions. Two well-demarcated ulcers of 2-4cm in diameter located in the mucosa of the dorsal sac of the rumen and ruminal pillars. The ulcers have raised borders and are covered by yellow exudate.



Figure 3. Poisoning by *E. contortisiliquum* pods in calves showing photosensitization and digestive lesions. The abomasal mucosa shows two deep ulcers with raising borders.

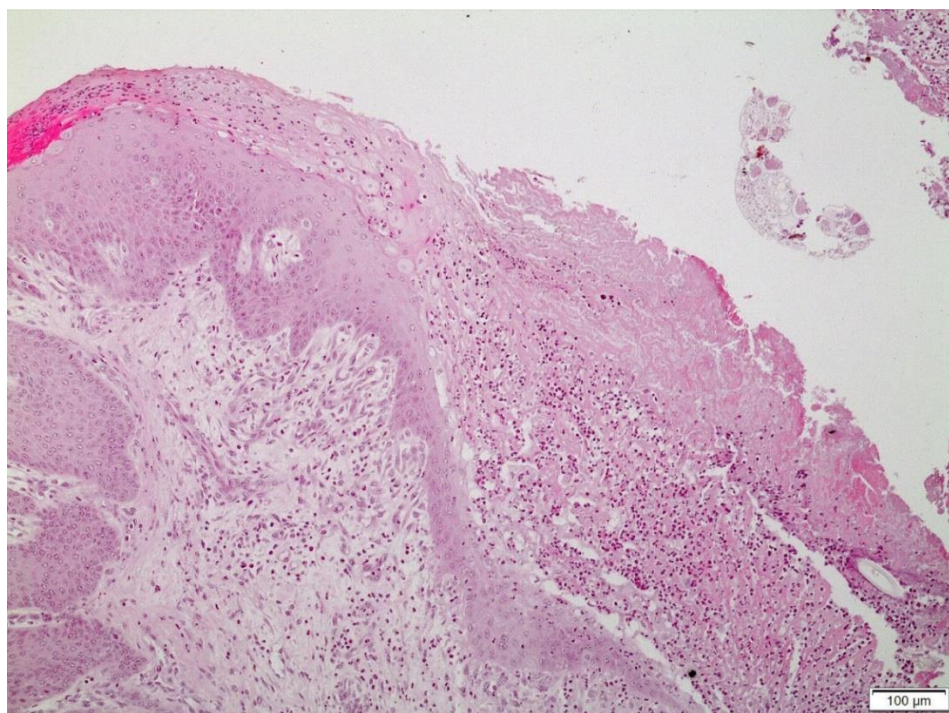


Figure 4. Rumen from a calf poisoned by the *E. contortisiliquum* pods. The epithelial had segmental loss due to coagulative necrosis. The epithelial gap is covered by serocellular exudate that rests on a base of granulation tissue (scarring). Neutrophils infiltrate the upper portion of the granulation tissue. HE.

In the current outbreak, *E. contortisiliquum* toxicosis occurred in 34 calves that fed on the pods of this plant. Some cattle died in the acute phase of the disease (but were not necropsied) and others developed subacute to chronic ruminal lesions. The following information was used to reach the diagnosis of *E. contortisiliquum* toxicosis in the current outbreak: (i) presence of *E. contortisiliquum* trees with large amounts of their pods fallen to the ground with evidence of being consumed by the affected cattle, (ii) cattle with clinical signs and lesions of photosensitization and digestive disorders, known to be associated with the poisoning by *E. contortisiliquum* in ruminants (Leal *et al.*, 2017; Pupin *et al.*, 2017), and (iii) presence of *E. contortisiliquum* seeds in the abomasum of one necropsied calf.

The findings in this outbreak could suggest for the first time an association between the occurrence of rumenitis due to acidosis and spontaneous intoxication by *E. contortisiliquum* in cattle. Spontaneous cases of ruminal acidosis occur in ruminants fed with large amounts of non-fiber carbohydrates (corn, wheat and barley) without prior adaptation (Nagaraja and Titgemeyer,

2007). As was already experimentally demonstrated (Bacha *et al.*, 2017; Pupin *et al.*, 2017), the pods of *E. contortisiliquum* have a high content of non-fiber carbohydrates which triggers ruminal acidosis. Drops in ruminal pH are associated with the accumulation of lactic acid, which potentially leads to ruminal acidosis (Hobson and Stewart, 1997). No attempt was made to determine the ruminal pH in the cases of this outbreak. However, ruminal pH is a diagnostic aid only in the acute phases of the disease since the pH rises in the subacute and chronic stages (Uzal *et al.*, 2016). In the same way, the ruminal content that is copious and porridge-like in the acute phases of ruminal acidosis may assume a healthy appearance over time and present itself even parched with the progression of the condition, although a poor nutritional state persists (Uzal *et al.*, 2016).

Acute ruminal acidosis is the first clinical manifestation of naive sheep fed with pods of *E. contortisiliquum* (Bacha *et al.*, 2017; Pupin *et al.*, 2017) and also in cattle (Leal *et al.*, 2017). Sheep may die at this stage or recover, but photosensitization wasn't seen; however, in cattle, those which recovered had photosensitization

(Leal *et al.*, 2017), as observed in the calves of the current outbreak, since the plant is highly hepatotoxic (Leal *et al.*, 2017). Hepatic lesions were not seen in the two necropsied calves, which could be due to the reversibility of hepatic lesions, similarly to those observed in a previous study (Leal *et al.*, 2017). As the necropsied calves were in a subacute or chronic phase of the disease, it is most likely that the liver lesions had already regenerated, without scarring; the same does not apply to the ruminal ulcers. In animals that survive the acute phase of ruminal acidosis, hydropic degeneration and necrosis of the ruminal epithelial supervenes. The unprotected ruminal mucosa is then subject to invasion by bacteria or fungi. Animals that also survive this stage will develop ulcers (Uzal *et al.*, 2016), similar to those found in the two necropsied cattle in this study.

Intoxications by other plants that cause similar clinical manifestations as *Stryphnodendron fissuratum* (Lazaro *et al.*, 2018) and *Brachiaria* spp. (Riet-Correa *et al.*, 2011) were ruled out in the differentials due to the absence of S.

*fissuratum* in the property, and due to the absence of hepatic lesions characteristic of *Brachiaria* spp. poisoning, which includes clusters of foamy macrophages, cholestasis and birefringent crystals in the lumen of bile ducts (Riet-Correa *et al.*, 2011).

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*Keywords: cattle diseases, plant poisoning, ruminal acidosis, photosensitization*

#### RESUMO

*Este trabalho descreve um surto de rumenite e abomasite decorrente de sobrecarga de carboidratos em um rebanho de 238 bezerros, com idades entre 12 e 15 meses, causada pela ingestão dos frutos de Enterolobium contortisiliquum. As taxas de morbidade, mortalidade e letalidade foram de, respectivamente, 12,7%, 5,2% e 42,1%. Clinicamente, os bovinos apresentaram fotossensibilização, salivação e diarreia. Os achados de necropsia foram semelhantes nos dois bezerros necropsiados e consistiram de fotodermatite e rumenite ulcerativa multifocal, subaguda a crônica, e abomasite. A relevância deste relato é que, pela primeira vez, foi possível associar a ocorrência da rumenite devido à sobrecarga de carboidratos com a intoxicação espontânea por E. contortisiliquum em bovinos, confirmando achados anteriormente descritos em experimentos realizados com ovinos.*

*Palavras-chave: doenças de bovinos, intoxicação por plantas, acidose ruminal, fotossensibilização*

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