Ciência Rural, Santa Maria, v.48:09, e20180074, 2018 http://dx.doi.org/10.1590/0103-8478cr20180074
ISSNe 1678-4596
PATHOLOGY

Acute rumenitis due to lipid overload in a bovine

Humberto Binsfeld Bonadiman[1] Claiton Ismael Schwertz[1*] Susy Hermes de Sousa[1] Ronaldo Michel Bianchi[1] Rafaela Albuquerque Caprioli[1] André Gustavo Cabrera Dalto[2] Saulo Petinatti Pavarini[1] David Driemeier[1]

[1]Setor de Patologia Veterinária, Faculdade de Veterinária (FAVET), Universidade Federal do Rio Grande do Sul (UFRGS), 91540-000, Porto Alegre, RS, Brasil. E-mail: claiton.schwertz@outlook.com. *Corresponding author.
[2]Setor de Grandes Ruminantes, Faculdade de Veterinária (FAVET), Universidade Federal do Rio Grande do Sul (UFRGS), Porto Alegre, RS, Brasil.

ABSTRACT: *The objective of this paper is to report the epidemiological, clinical, and pathological aspects of a case of rumenitis due to the ingestion of soybean oil in a bovine. The ox had access to barrels that stored soybean oil and ingested an indeterminate amount of the product. After consuming it, the animal presented hiporexia; liquid, brownish, and greasy feces; severe dehydration (12%); apathy; sternal recumbency; and death with a clinical evolution of 4 days. At necropsy, the rumen was filled with voluminous food and moderate amount of white-gray liquid with a greasy appearance. Upon microscopic examination, hydropic degeneration of the epithelium and areas of mucosal necrosis were observed in the rumen and reticulum. From these findings, we concluded that the bovine developed a state of acidosis and acute rumenitis due to excessive intake of lipids.*
Key words: *diseases of ruminants; nutrition; ruminal acidosis; soybean oil.*

Ruminite aguda por sobrecarga de lipídeos em um bovino

RESUMO: *O objetivo deste trabalho é relatar os aspectos epidemiológicos, clínicos e patológicos de um caso de ruminite por ingestão de óleo de soja em um bovino. O animal teve acesso acidental a tonéis que armazenavam óleo de soja e ingeriu uma quantidade indeterminada do produto. Após o consumo, o bovino apresentou hiporexia, fezes líquidas, acastanhadas e de aspecto gorduroso, desidratação severa (12%), apatia, decúbito esternal e morte, com evolução clínica de quatro dias. Na necropsia o rúmen estava repleto por alimentos volumosos e moderada quantidade de líquido branco-acinzentado e de aspecto gorduroso. Microscopicamente, no rúmen e retículo havia degeneração hidrópica do epitélio e áreas de necrose da mucosa. A partir destes achados, concluímos que o bovino desenvolveu um quadro de acidose e ruminite aguda por consumo excessivo de lipídeos.*
Palavras-chave: *doenças de ruminantes; nutrição; acidose ruminal; óleo de soja.*

The rumen consists of a complex ecosystem wherein the nutrients consumed are digested by fermentative processes mediated by microorganisms. The main products formed by this fermentation are volatile fatty acids and the remains of microorganisms, which are used as a protein source. In this environment, the unsaturated fatty acids are transformed into saturated fatty acids and these are transformed by two main routes: lipolysis and hydrogenation (CASTILLO et al., 2014).

Variations in diet lead to changes in the metabolism and composition of rumen microbiota, and can cause degenerative and inflammatory lesions in the mucosal epithelium, as is classically observed in ruminal acidosis due to fermentable carbohydrates overload, or ruminal lactic acidosis (PACHECO & CRUZ, 2015; UZAL et al., 2016). Therefore, the objective of this paper was to report a case of acute ruminitis due to lipid overload in a bovine, presenting the epidemiological, clinical, and pathological findings.

A 2-year-old male Jersey bovine had a history of incidental access to barrels containing soybean oil, from which it abruptly ingested an indeterminate amount. After consuming the product, the animal presented diarrhea, characterized by liquid, brownish, and greasy feces; oligodipsia; hyporexia; mild dehydration; and ruminal atony. Treatment with fluid therapy and restitution of the ruminal flora was administered; however, there was no clinical improvement, and over a period of 4 days the animal experienced severe apathy, dehydration (12%), sternal recumbency, and finally death.

After death, the bovine was submitted for necropsy. The clinical history was obtained from the

Received 02.05.18 Approved 08.02.18 Returned by the author 08.20.18
CR-2018-0074.R1

owner and fragments of several organs were collected, fixed in a 10% buffered formalin solution, routinely processed, and stained with hematoxylin and eosin. At necropsy, marked enophthalmia (dehydration) and liquefied feces adhering to the hair of the perineal region were observed. The rumen was filled with voluminous food, as well as white-gray liquid with a greasy appearance (Figure 1A). Multifocal areas of red discoloration were observed in the ruminal mucosa, and were better noted when the keratinized layer was removed. The contents of the abomasum and small and large intestines were yellowish to light brown in color, and were greasy and liquefied.

Upon microscopic examination, the rumen and reticulum exhibited moderate and multifocal areas of hydropic degeneration of the epithelium, characterized by the formation of intracytoplasmic vacuoles (Figure 1B), as well as extensive areas of necrosis of the mucosa epithelium, characterized by nuclei in karyorrhexis and karyolysis, as well as hypereosinophilia and loss of cytoplasmic limits of keratinocytes. Associated with the foci of necrosis, there was marked inflammatory infiltrate of neutrophils and a small number of lymphocytes and plasma cells, which formed intraepithelial pustules and were also observed to cluster between the necrotic epithelium and the lamina propria. There was a large amount of coccoid bacteria amidst necrotic areas and deposited in the keratinized layer, which was fragmented (Figures 1C and 1D). In the other organs, no significant lesions were observed.

Although the negative effects of excessive lipids on the diet of cattle have been reported (GALBRAITH et al., 1971; DOREAU & FERLAY, 1995; MESSANA et al., 2013), there are no previous reports of deaths from excessive intake of soybean oil. Based on the biochemistry of the rumen and the pathophysiology of lactic acidosis in ruminants, it is assumed that, in this case, an excessive production of fatty acids such as propionate and acetate by lipolytic microorganisms occurred (FUENTES et al., 2009). Thus, a condition similar to that in ruminal lactic acidosis due to excessive carbohydrate consumption may have occurred. The higher production of fatty acids leads to diminution of the pH, which promotes proliferation of streptococci, particularly *Streptococcus bovis*, which produce more lactic acid and create an adverse environment for gram-negative bacteria and protozoa. With a pH<5.0, *Lactobacillus* sp. proliferation occurs, which induces further lactate formation (RADOSTITS et al., 2006). Higher concentration of lactate increases the osmotic pressure, which favors the passage of intravascular and interstitial fluid into the rumen. The excessive acidity of the content with increased osmolarity causes lesions in the ruminal mucosa. The high electrolyte imbalance and D-lactate absorption in the blood lead to metabolic acidosis, severe dehydration, and hemoconcentration, which can lead to death of the animal (RADOSTITS et al., 2006).

The clinical signs presented in the current case, such as diarrhea, ruminal atony, and dehydration, were compatible with ruminal acidosis and rumenitis (AFONSO & MENDONÇA, 2007; GONZÁLEZ et al., 2014). In addition to the clinical signs, the ruminal lesions were also similar to those described in cases of lactic acidosis (QUEVEDO et al., 2015). The differential diagnosis was based on the animal's history, which demonstrated that the ox did not receive concentrate and did not have incidental access to this type of feed. Additionally, upon necroscopic examination, it was evident that the content was composed mainly of voluminous food rather than grains.

Ruminal acidosis is most often associated with the consumption of concentrate, particularly corn and wheat. However, there have been cases associated with the consumption of alternative products rich in starch, sucrose, lactose, or glucose, such as barley, oats, bakery products, sorghum, potatoes, fruit, molasses, brewery waste, and, in some cases, new and succulent pastures, which are rich in fermentable carbohydrates (AFONSO & MENDONÇA, 2007; BRUST et al., 2015).

The histological findings of the rumen were similar to those observed in *Baccharis* spp. and *Baccharidastrum triplinervium* poisonings. The differential diagnosis was made by the absence of these plants on the farm and due to the fact that the bovine was raised at that location. Typically, *Baccharis* spp. poisoning occurs when cattle from areas free of the plant are transferred to infested pastures (RISSI et al., 2005), and the *Baccharidastrum triplinervium* poisoning occurs when the animals are transferred to highly infested areas with a lack of forage (LANGOHR et al., 2005).

Another differential diagnosis includes acute sodium fluorosilicate poisoning, in which the ruminal lesions resemble those described in this case. In this poisoning, animals must be exposed to contaminated areas or have access to large amounts of commercial formulations of the product (LUCIOLI et al., 2007; PANZIERA et al., 2018). Moreover, in *Baccharis* spp. and sodium fluorosilicate poisonings, lymphoid necrosis in the lymph nodes and spleen is frequently observed (RISSI et al., 2005; LUCIOLI

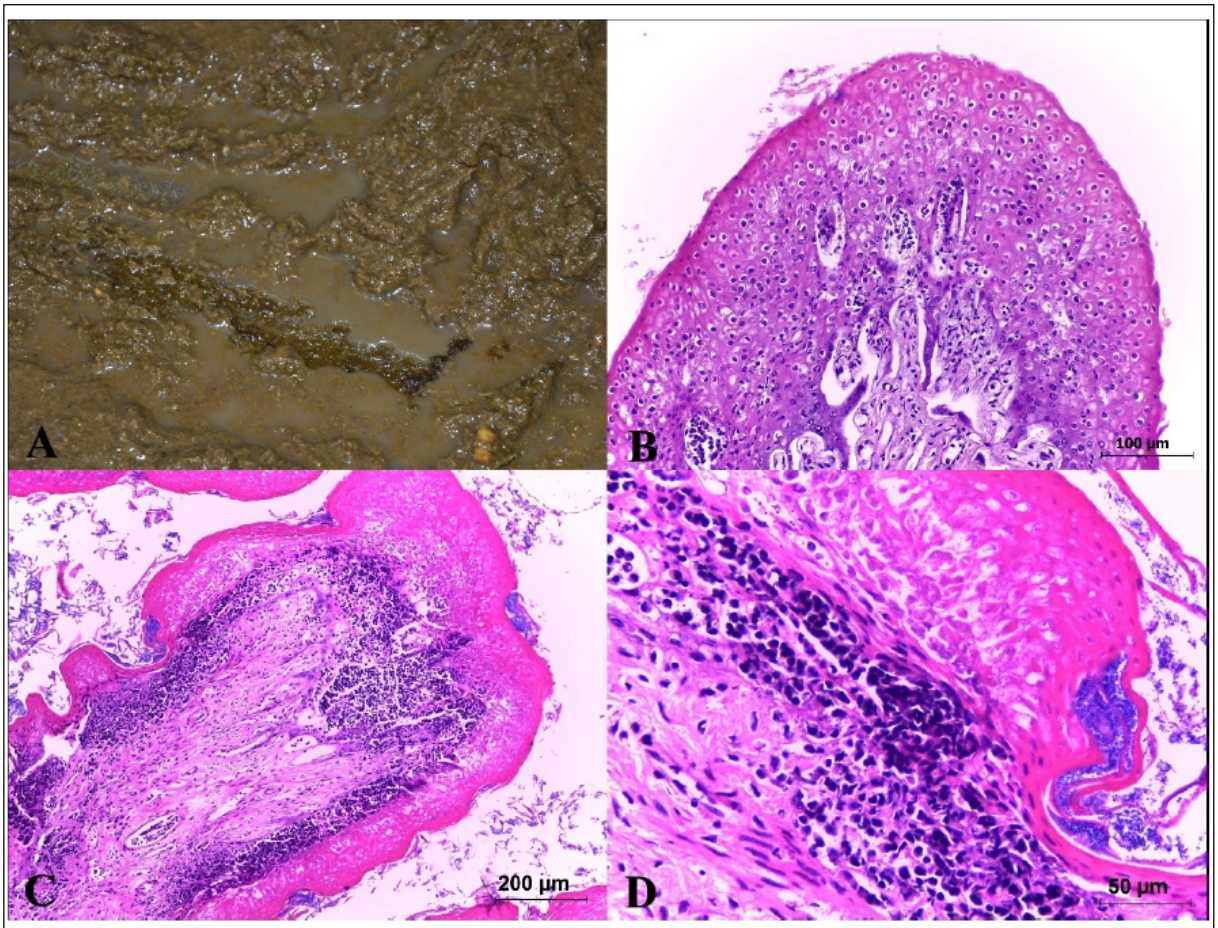

Figure 1 - A: Acute rumenitis due to lipid overload in a bovine. (A) Ruminal content composed of voluminous and moderate amount of white-gray liquid with a greasy appearance. B: Rumen. Hydropic degeneration of keratinocytes at the top of the ruminal papilla and a mild multifocal infiltrate of neutrophils forming intraepithelial pustules (hematoxylin and eosin [H&E] 200x). **C:** Rumen. Marked necrosis of the epithelium associated with marked inflammatory infiltrate that extends to the lamina propria, composed predominantly of neutrophils. Bacterial myriads are also observed in the superficial mucosa (H&E 100x). **D:** Rumen, greater increase of figure C. Necrosis of the epithelium; inflammatory infiltrate of neutrophils, lymphocytes, and plasma cells; fragmentation of the keratinized layer; and coccoid bacterial myriads are observed (H&E 400x).

et al., 2007, PANZIERA et al., 2018), which was not observed in the case described here.

Through the association of the history of soybean oil intake, clinical information, and macroscopic and microscopic changes, we concluded that the bovine developed a state of ruminal acidosis and acute ruminitis due to excessive intake of lipids.

DECLARATION OF CONFLICTING OF INTERESTS

The authors declare no conflict of interest. The founding sponsors had no role in the design of the study; in the collection, analyses, or interpretation of data; in the writing of the manuscript, and in the decision to publish the results.

ACKNOWLEDGEMENTS

The authors are grateful to Conselho Nacional de Desenvolvimento Científico e Tecnológico (CNPq) and Coordenação de Aperfeiçoamento de Pessoal de Nível Superior (CAPES).

AUTHORS' CONTRIBUTIONS

All authors contributed equally for the conception and writing of the manuscript. All authors critically revised the manuscript and approved of the final version.

REFERENCES

AFONSO, J.A.B.; MENDONÇA, C.L. Acidose láctica ruminal. In: RIET-CORREA, F. et al. **Doenças de**

Ruminantes e Equídeos. 3.ed, v.2. São Paulo: Livraria Varela, 2007. p. 313-318.

BRUST, L.A.C. et al. Cattle diseases associated with consumption of beer residues. **Pesquisa Veterinária Brasileira**, v.35, n.12, p. 956-964. 2015. Available from: <http://www.scielo.br/pdf/pvb/v35n12/1678-5150-pvb-35-12-00956.pdf>. Accessed: Sept. 15, 2017.

CASTILLO-GONZALEZ, A.R. et al. Rumen microorganisms and fermentation. **Archivos de Medicina Veterinaria**, Valdivia, v. 46, n. 3, p. 349-361, 2014. Available from: <http://www.scielo.cl/pdf/amv/v46n3/art03.pdf>. Accessed: Aug. 21, 2017. doi: 10.4067/S0301-732X2014000300003.

DOREAU, M.; FERLAY, A. Effect of dietary lipids on nitrogen metabolism in the rumen: a review. **Livestock Production Science**, v. 43, i. 2, 1995. Available from: <https://doi.org/10.1016/0301-6226(95)00041-I>. Accessed: Aug. 21, 2017. doi: 10.1016/0301-6226(95)00041-I.

FUENTES, M.C. et al. Effect of pH and level of concentrate in the diet on the production of biohydrogenation intermediates in a dual-flow continuous culture. **Journal of Dairy Science**, v. 92 , i. 9, 2009. Available from: <http://dx.doi.org/10.3168/jds.2008-1722>. Accessed: Aug. 21, 2017. doi: 10.3168/jds.2008-1722.

GALBRAITH,H. et al. Antibacterial activity of long chain fatty acids and the reversal with calcium, magnesium, ergocalciferol and cholesterol. **Journal of Applied Bacteriology**, v. 34, i. 4, 1971. Available from: <http://onlinelibrary.wiley.com/doi/10.1111/j.1365-2672.1971.tb01019.x/pdf>. Accessed: Aug. 21, 2017. doi: 10.1111/j.1365-2672.1971.tb01019.x.

GONZÁLEZ, F.H.D. et al. **Transtornos metabólicos nos animais domésticos**. 2 ed. Porto Alegre: Editora da UFRGS, 2014. p. 79-84.

LANGOHR, I.M. et al. Poisoning in cattle by *Baccharidastrum triplinervium* (*Asteraceae*). **Pesquisa Veterinária Brasileira**, v.25, n.4, p.235-238, 2005. Available from: <http://www.scielo.br/scielo.php?script=sci_arttext&pid=S0100-736X2005000400009>. Accessed: Nov. 29, 2017. doi: 10.1590/S0100-736X2005000400009.

LUCIOLI, J. et al. Acute sodium fluorsilicate poisoning in cattle in the state of Santa Catarina. **Pesquisa Veterinária Brasileira**, v.27, n.1, p.49-52. 2007. Available from: <http://www.scielo.br/pdf/pvb/v27n1/09.pdf>. Accessed: Nov. 29, 2017. doi: 10.1590/S0100-736X2007000100009.

MESSANA, J.D. et al. Rumen fermentation and rumen microbes in Nellore steers receiving diets with different lipid contentes. **Revista Brasileira de Zootecnia**, v. 42, n. 3, 2013. Available from: <http://www.scielo.br/pdf/rbz/v42n3/08.pdf>. Accessed: Sept. 10, 2017. doi: 10.1590/S1516-35982013000300008.

PACHECO, R.D.L.; CRUZ, G.D. Acidosis in cattle. In: Puniya A., Singh R., Kamra D. (eds). **Rumen Microbiology**: From Evolution to Revolution. Springer, New Delhi, 2015. Cap. 21, p. 315-327.

PANZIERA, W. et al. Acute sodium fluorosilicate poisoning in cattle. **Acta Scientiae Veterinariae**, v. 46 (Suppl 1): 280, 2018. Available from: <http://www.ufrgs.br/actavet/46-suple-1/CR_280.pdf>. Accessed: May 20, 2018.

QUEVEDO, L.S. et al. Outbreak of ruminal acidosis in semi-extensive breeding beef cattle production - Case report. **Revista Brasileira de Medicina Veterinária**, v. 37, n. 3, p. 269-274, 2015. Available from: <http://www.rbmv.com.br/pdf_artigos/14-09-2015_11-22RBMV%200156a.pdf>. Accessed: Aug. 19, 2017.

RADOSTITS, O.M. et al. **Veterinary Medicine: A textbook of diseases of cattle, sheep, goats, pigs and horses.** 10th ed. Saunders, 2006. 2065p.

RISSI, D.R. et al. Spontaneous *Baccharis coridifolia* poisoning in cattle. **Pesquisa Veterinária Brasileira**, v.25, n.2, p.111-114, 2005. Available from: <http://www.scielo.br/scielo.php?script=sci_arttext&pid=S0100736X2005000200008&lng=en&nrm=iso>. Accessed: Jul. 29, 2017. doi: 10.1590/S0100-736X2005000200008.

UZAL, F.A. et al. Alimentary system. In: MAXIE, M.G. **Jubb, Kennedy, and Palmer's pathology of domestic animals**. 6.ed. St. Louis: Elsevier, 2016. V.2, Cap.6, p.01-257.