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Spontaneous poisoning of cattle by onion (Allium cepa) in Brazil¹

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ABSTRACT.- Hugen G.F.G.P., Ogliari D., Wicpolt N.S., Melchioretto E., Traverso S.D., Wiser C., Hemckmeier D. & Gava A. 2024. **Spontaneous poisoning by onion (***Allium cepa***) in cattle in Brazil**. *Pesquisa Veterinária Brasileira 44:e07204, 2024*. Graduate Program in Animal Science, Centro de Ciências Agroveterinárias, Universidade do Estado de Santa Catarina, Av. Luiz de Camões 2090, Conta Dinheiro, Lages, SC 88520-000, Brazil. E-mail: aldo.gava@udesc.br

The state of Santa Catarina is the largest national producer of onions. The consumption of onion (*Allium cepa*) by domestic animals can cause hemolytic anemia and hyperhemoglobinemia of Heinz bodies in erythrocytes. The epidemiological data and the pathological clinical picture were obtained during a visit to the property where the disease occurred. A herd of 54 beef cattle from a property in the municipality of Bom Retiro/SC, was placed in an onion crop, which had not been completely harvested because it was outside the desired standard for trade. The animals remained in this field for eight days. At the end of this period, all of them presented apathy, anorexia, brown urine and mucous membranes and an onion odor on expiration. The macroscopic changes observed were pale mucous membranes, blood with decreased viscosity and increased clotting time, liver with an evident lobular pattern and remains of onion in the rumen. The carcass gave off a strong onion odor. By histological analysis, there was central lobular coagulation necrosis in bridging in the liver. In the kidneys, there were intracytoplasmic hyaline droplets in tubular epithelial cells and a deposit of eosinophilic material in the lumen of the tubules. For the diagnosis of onion poisoning, epidemiological data associated with clinical signs, such as pale mucous membranes with evidence of hemoglobin pigmentation (brown mucosa) and hemoglobinuria, should be taken into account. This article seems to be the first report of onion poisoning in cattle in Brazil.

INDEX TERMS: Hemolytic anemia, hemoglobinuria, hemoglobin nephrosis, centrilobular necrosis, poisoning, onion, *Allium cepa*, cattle, Brazil.

RESUMO.- [Intoxicação espontânea por cebola (*Allium cepa*) em bovinos.] O estado de Santa Catarina é o maior produtor nacional de cebola. O consumo de cebola (*Allium cepa*) por animais domésticos pode causar anemia hemolítica, hiperhemoglobinemia de corpúsculos de Heinz nos eritrócitos. Os dados epidemiológicos e o quadro clínico-patológico foram obtidos em visita à propriedade onde a enfermidade ocorreu. Um rebanho de 54 bovinos de corte de uma propriedade localizada no município de Bom Retiro/SC, foi colocado em uma lavoura de cebola, a qual não havia sido completamente colhida por estar fora do padrão desejado para o comércio. Os animais permaneceram por um período de oito dias nesta lavoura e, ao final deste período, todos apresentaram apatia, anorexia, urina e mucosas de coloração marrom e odor de cebola na expiração. As alterações macroscópicas observadas foram mucosas pálidas, sangue com viscosidade diminuída e aumento do tempo de coagulação, fígado com padrão lobular evidente e restos de cebola no rúmen. A carcaca exalava forte odor de cebola. Pela análise histológica havia, no fígado, necrose de coagulação centrolobular em ponte e nos rins havia gotas hialinas intracitoplasmáticas em células do epitélio tubular e depósito de material eosinofílico na luz de túbulos. Para o diagnóstico da intoxicação por cebola devese levar em conta principalmente os dados epidemiológicos associados aos sinais clínicos, como palidez de mucosas com evidências de pigmentação por hemoglobina (mucosa marrom)

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e hemoglobinúria. Este artigo parece ser o primeiro relato de intoxicação por cebola em bovinos no Brasil.

TERMOS DE INDEXAÇÃO: Anemia hemolítica, hemoglobinúria, nefrose hemoglobínica, necrose centrolobular, intoxicação, cebola, *Allium cepa*, Brasil.

INTRODUCTION

Brazil is one of the world's largest producers of onions (*Allium cepa*), with 1,500,000 tons produced each year, making it the third most important vegetable economically. This vegetable has the highest gross production value. The state of Santa Catarina is the largest national onion producer, generating approximately R\$400 million per year (IBGE 2019, Epagri-Ciram 2020).

Production in Santa Catarina accounts for 33% of the national production volume, with a total area of 20,000 ha and a volume of 500 thousand tons. The municipalities with the highest production are Ituporanga, Alfredo Wagner, Aurora, Imbuia, and Bom Retiro. Most volumes are produced on family farms, with around 8,000 families directly involved in onion cultivation (Epagri-Ciram 2020).

Due to the large production volumes, low economic value, and low quality, onions are supplied as cattle feed. According to producers, they are well accepted as feed by this species. However, the consumption of onion by domestic animals can lead to hemolytic anemia, hyperhemoglobinemia, and Heinz bodies in erythrocytes, resulting in organ damage and/or death when consumed in large quantities. Cases of such poisoning have been reported worldwide (Rae 1999, Selim et al. 1999, Fighera et al. 2002). Cases of spontaneous poisoning by onion in cattle (Koger 1956, Lazarus & Rajamani 1968, Hutchison 1977, Verhoeff et al. 1985, Carbery 1999, Rae 1999), equines (Thorp & Harshfield 1939, Pierce et al. 1972), sheep (Aslani et al. 2005), buffaloes (Borelli et al. 2009), cats (Kaplan 1995), and dogs (Farkas & Farkas 1974, Spice 1976) as well as experimental poisoning in cattle (Lincoln et al. 1992), equines (Pierce et al. 1972), sheep (James & Binns 1966, Kirk & Bulgin 1979, Fredrickson et al. 1995, Selim et al. 1999), dogs (Gruhzit 1931, Harvey & Rackear 1985), and cats (Robertson et al. 1998, Fighera et al. 2002) have been reported. Poisoning by wild onion (Allium validum) has also been reported by Van Kampen et al. (1970) in sheep.

The epidemiology of this poisoning differs with the species. In sheep, cattle, buffaloes and equines, poisoning occurs when they are housed in areas where onions are cultivated or with access to onion storage areas or receive inadequate quantities (Lazarus & Rajamani 1968, Pierce et al. 1972, Hutchison 1977, Rae 1999, Aslani et al. 2005, Borelli et al. 2009). Small animals, such as dogs and cats, have been poisoned by eating homemade foods containing onion, regardless of whether it was raw, cooked, or dehydrated (Farkas & Farkas 1974, Kaplan 1995, Robertson et al. 1998).

Toxicity to plants of the genus *Allium* is associated with its disulfide compounds propyl n-disulfide, S-methylcysteine sulfoxide (SMCO), and S-propyl-L-cysteine sulfoxides, which are derived from amino acids. These compounds can lead to hemolytic anemia due to cyanosis, formation of Heinz bodies, and even death (Williams et al. 1941, Hutchison 1977, Block 1991, Rae 1999, Borelli et al. 2009, Aslani et al. 2005). The clinical symptoms observed in cattle poisoned by onion include lack of appetite, increased heart and respiratory rates, pale mucous membranes, jaundice, dark urine, and odor of onion in breath and feces (Hutchison 1977, Verhoeff et al. 1985, Rae 1999). Necropsy findings include jaundice, dark brown kidneys and urine, tissues with an onion odor, and onion fragments within the rumen. Histological symptoms include centrilobular necrosis and vacuolation of hepatocytes in the mid-zonal areas of the liver and hemoglobinuric nephrosis in the kidneys (Hutchison 1977, Rae 1999, Borelli et al. 2009).

The objective of the present study was to describe cases of spontaneous onion poisoning in cattle in Brazil.

MATERIALS AND METHODS

Animal Ethics. The execution of the search followed a procedure analyzed and approved by the Ethics Committee on the Use of Animals of the "Universidade do Estado de Santa Catarina" (CEUA-UDESC) under protocol no. 7666200720.

The epidemiological data and the clinical-pathological profile of onion poisoning were obtained during a visit to a site of the disease and recorded at the CAV-UDESC Animal Pathology Sector. Necropsy of a diseased cow was conducted, and tissue samples were collected from the lungs, heart, liver, kidneys, spleen, rumen, reticulum, omasum, abomasum, large and small intestines, brain, bladder, and bone marrow. The samples were fixed in 10% buffered formalin, and 3-µm-thick histological sections were prepared, stained with hematoxylin and eosin (HE) stains, and observed under an optical microscope.

RESULTS

A herd comprising 54 beef cattle, aged 1-8 years and including both sexes, in the municipality of Bom Retiro/SC, was placed in an onion field after harvesting. There was a surplus of crops as they were not within the desired pattern for trade (Fig.1). The cattle remained on this farm for eight days; at the end of this period, all animals presented with varying degrees of apathy, anorexia, brown urine, mucous membranes (Fig.2), polydipsia, and polyuria. These signs were more evident in adult animals. Three cattle that presented with a serious clinical condition were subjected to fluid therapy. During this treatment, one cow became agitated and died. The others were removed from contact with onions and recovered within a few days.

Macroscopic analysis of the samples from the dead cow revealed pale mucous membranes, blood with reduced viscosity and increased clotting time, and liver with an evident lobular pattern and yellowish-brown color (Fig.3). The kidneys were dark brown, with a slight difference between cortical and medullary tissues (Fig.4). The bladder was distended with dark urine (Fig.5). A large number of onion skins and leaves were observed in the rumen. The entire corpse had a strong onion odor, especially after opening the gastrointestinal tract.

The main histological observations included degeneration and necrosis of renal tubular epithelium cells accompanied by cytoplasmic hyaline drops and deposits of eosinophilic material similar to hemoglobin in the lumen of the tubules. Centrilobular coagulation bridging necrosis was observed in the liver (Fig.6), and there were hyaline, intracytoplasmic drops in cells of the tubular epithelium and deposition of eosinophilic material in the lumen of the tubules of the kidneys (Fig.7).

DISCUSSION

The diagnosis of onion poisoning was confirmed based on the clinical-pathological profile and the fact that onions were consumed in large quantities during grazing. The outbreak monitored in the present study reveals that onions are palatable but toxic to cattle.

The hemolytic anemia observed in the present study was related to the cattle's spontaneous ingestion of a large amount of onion. In the municipality of Bom Retiro, where the outbreak was observed, and in other municipalities, such as Ituporanga, Alfredo Wagner, and Aurora, onion cultivation is done on a large scale. When onions become unsuitable for commercial sale, producers leave the product on the farm. In the present study, the cattle were sent to the farm with a large quantity of onion available, and owing to the scarcity of pasture on the property, they ingested the plants. According to Rae (1999), onions are easily consumed by cattle, who often prefer them to high-quality forage or grains. Onion consumption can cause hemolytic anemia and methemoglobinemia (Fighera et al. 2002), resulting in hemolysis with ischemia-induced damage and hyperhemoglobinemia, characterized by hemoglobinuric nephrosis and centrilobular hepatic necrosis (Borelli et al. 2009). The cattle poisoned by onion in the present study had pale mucous membranes, hemoglobinuria and exercise intolerance. One animal undergoing fluid therapy had severe agitation and died shortly afterward. This reinforces that animals that are clinically ill due to ingesting the plant should not be moved.

Onion-induced hemolytic anemia appears earlier in dogs, cats, and horses than in ruminants (Pierce et al. 1972, Fredrickson et al. 1995). The delay in the onset of the ruminant poisoning may be associated with the function of the forestomach (Selim et al. 1999). Koger (1956) observed that younger cattle appear more resistant to onion poisoning. This is in line with the observation of our study that only adult animals fell seriously ill. Borelli et al. (2009) revealed that



Fig.1. Onion farm where cattle poisoning occurred. Municipality of Bom Retiro/SC.



Fig.2. Bovine. Spontaneous poisoning by *Allium cepa*. Pale brown conjunctiva.



Fig.3. Bovine. Spontaneous poisoning by *Allium cepa*. Liver, yellowishbrown.



Fig.4. Bovine. Spontaneous poisoning by *Allium cepa*. Kidney, dark brown with multiple dark spots.



Fig.5. Bovine. Spontaneous poisoning by *Allium cepa*. Bladder, containing dark-colored urine.

adult buffaloes died after consuming onions, but lactating calves did not fall ill.

The clinical signs, epidemiology, and macroscopic and histological observation in the present report were similar to those described in previous studies on onion poisoning in cattle and buffaloes (Koger 1956, Hutchison 1977, Verhoeff et al. 1985, Rae 1999, Borelli et al. 2009).

The epidemiological data associated with clinical signs must be considered to diagnose onion poisoning, such as pallor of mucous membranes with evidence of hemoglobin pigmentation (brown mucosa) and hemoglobinuria. Macroscopic observations, such as increased clotting time, the slightly brown coloration of mucous membranes and serous membranes, and histological examination of the kidneys and liver, define the diagnosis.

The differential diagnosis of onion poisoning in cattle includes poisoning by any plant that causes hemolytic anemia, such as *Brachiaria radicans* (Gava et al. 2010), *Ditaxis desertorum* (Tokarnia et al. 1997) *Indigofera suffruticosa* (Salvador et al. 2010), and *Brassica oleracea* (Selim et al. 1999), bacillary hemoglobinuria, babesiosis and postpartum hemoglobinuria (Tokarnia et al. 2012). In Santa Catarina, the main differential diagnoses are *Brachiaria radicans* poisoning, babesiosis, and bacillary hemoglobinuria; these conditions were ruled out in this study based on epidemiology and microscopy.

The treatment indicated for animals with onion poisoning involves restricting access to the plant. Fluid therapy and blood transfusion are indicated for severely affected animals (Lincoln et al. 1992, Tokarnia et al. 2012). Treatment with oral antibiotics may be beneficial in reducing rumen anaerobic bacteria that promote the formation of some oxidative substances (Selim et al. 1999). According to Lincoln et al. (1992), a viable alternative to ingesting onions by ruminants may be to mix them with the feed and not exceed 25%.

CONCLUSION

In onion-producing regions, this vegetable is given to cattle because of its good palatability; however, when ingested in large quantities, it causes hemolytic anemia and death in cattle.

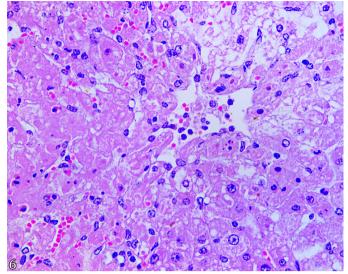


Fig.6. Bovine. Spontaneous poisoning by Allium cepa. Liver, centrilobular necrosis. HE, obj.40x.

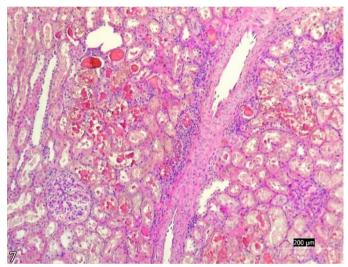


Fig.7. Bovine. Spontaneous poisoning by *Allium cepa*. Kidney, hyaline, intracytoplasmic droplets in the tubular epithelium. HE, obj.10x, bar = 200μm.

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Conflict of interest statement.- The authors declare that there is no conflict of interest.

REFERENCES

- Aslani M.R., Mohri M. & Movassaghi A.R. 2005. Heinz body anaemia associated with onion (*Allium cepa*) toxicosis in a flock of sheep. Comp. Clin. Pathol. 14:118-120. https://dx.doi.org/10.1007/s00580-005-0563-5
- Block E. 1991. The organosulfur chemistry of the onion. Phosphorus, Sulfur, and Silicon Relat. Elem. 58(1/4):3-15. <https://dx.doi. org/10.1080/10426509108040623>

- Borelli V., Lucioli J., Furlan F.H., Hoepers P.G., Roveda J.F., Traverso S.D. & Gava A. 2009. Fatal onion (*Allium cepa*) toxicosis in water buffalo (*Bubalus bubalis*). J. Vet. Diagn. Invest. 21(3):402-405. https://dx.doi.org/10.1177/104063870902100321 < https://dx.doi. org/10.1177/104063870902100321 < https://dx.doi.
- Carbery J.T. 1999. A case of onion poisoning in a cow. N. Z. Vet. J. 47(5):184. https://dx.doi.org/10.1080/00480169.1999.36140 https://dx.doi.org/10.1080/00480169 https://dx.doi.org/10.1080 https://dx.doi.org/10.1080 https://dx.doi.org
- Epagri-Ciram 2020. Núcleo de Estudos em Cebolicultura. Centro de Informações de Recursos Ambientais e de Hidrometeorologia de Santa Catarina, Florianopolis. Available at http://ciram.epagri.sc.gov.br/CebolaNet/ Accessed on Feb. 25, 2020.
- Farkas M.C. & Farkas J.N. 1974. Hemolytic anemia due to ingestion of onions in a dog. J. Am. Anim. Hosp. Assoc. 10:65.
- Fighera R.A., Souza T.M., Langohr I. & Barros C.S.L. 2002. Intoxicação experimental por cebola, *Allium cepa* (Liliaceae), em gatos. Pesq. Vet. Bras. 22(2):79-84. https://dx.doi.org/10.1590/S0100-736X200200020008
- Fredrickson E.L., Estell R.E., Havstad K.M., Shupe W.L. & Murray L.W. 1995. Potential toxicity and feed value of onions for sheep. Livest. Prod. Sci. 42(1):45-54. https://dx.doi.org/10.1016/0301-6226(94)00066-G
- Gava A., Deus M.R.S., Branco J.V., Mondadori A.J. & Barth A. 2010. Intoxicação espontânea e experimental por *Brachiaria radicans* (tanner-grass) em bovinos. Pesq. Vet. Bras. 30(3):255-259. https://dx.doi.org/10.1590/S0100-736X2010000300012
- Gruhzit O.M.I. 1931. Anemia of dogs produced by feeding of the whole onions and of onion fractions. Am. J. Med. Sci. 181:812-815.
- Harvey J.W. & Rackear D. 1985. Experimental onion-induced hemolytic anemia in dogs. Vet. Pathol. 22(4):387-392. https://dx.doi.org/10.1177/030098588502200414 < https://dx.doi.
- Hutchison T.W. 1977. Onions as a cause of Heinz body anaemia and death in cattle. Can. Vet. J. 18(12):358-360. <PMid:597815>
- IBGE 2019. Produção da Pecuária Municipal (PPM) 2018. Instituto Brasileiro de Geografia e Estatística, Brasília. Available at <</p>
 https://www.ibge.gov. br/estatisticas/economicas/agricultura-e-pecuaria/9107-producao-dapecuaria-municipal.html> Accessed on Feb. 3, 2020.
- James L.F. & Binns W. 1966. Effects of feeding wild onions (*Allium validum*) to bred ewes. J. Am. Vet. Med. Assoc. 149(5):512-514. <PMid:6008240>
- Kaplan A.J. 1995. Onion powder in baby food may induce anemia in cats. J. Am. Vet. Med. Assoc. 207(11):1405. <PMid:7493865>
- Kirk J.H. & Bulgin M.S. 1979. Effects of feeding cull domestic onions (*Allium cepa*) to sheep. Am. J. Vet. Res. 40(3):397-399. <PMid:475092>
- Koger L.M. 1956. Onion poisoning in cattle. J. Am. Vet. Med. Assoc.129(2):75. <PMid:13331835>

- Lazarus A.E. & Rajamani S. 1968. Poisoning due to onion spoilage in cattle. Indian Vet. J. 45(10):877-880. <PMid:5751821>
- Lincoln S.D., Howell M.E., Combs J.J. & Hinman D.D. 1992. Hematologic effects and feeding performance in cattle fed cull domestic onions (*Allium cepa*). J. Am. Vet. Med. Assoc. 200(8):1090-1094. https://dx.doi.org/10.2460/javma.1992.200.08.1090 https://dx.doi.org/10.2460/javma.1992 https://dx.doi.org/10.2460/javma.1992 https://dx.doi.org/10.2460/javma.1992 https://dx.doi.org/10.2460/javma.1992 https://dx.doi.org/10.2460/javma.1992 https://dx.doi.079 https://dx.doi.079 https://dx.doi.079 https://dx.doi.079
- Pierce K.R., Joyce J.R., England R.B. & Jones L.P. 1972. Acute hemolytic anemia caused by wild onion poisoning in horses. J. Am. Vet. Med. Assoc. 160(3):323-327. <PMid:5061884>
- Rae H.A. 1999. Onion toxicosis in a herd of beef cows. Can. Vet. J. 40(1):55-57. <PMid:9919370>
- Robertson J.E., Christopher M.M. & Rogers Q.R. 1998. Heinz body formation in cats fed baby food containing onion powder. J. Am. Vet. Med. Assoc. 212(8):1260-1266. <PMid:9569166>
- Salvador I.S., Medeiros R.M.T., Pessoa C.R.M, Dantas A.F.M., Sucupira Júnior G. & Riet-Correa F. 2010. Intoxicação por *Indigofera suffruticosa* (Leg. Papilionoideae) em bovinos. Pesq. Vet. Bras. 30(11):953-957. https://dx.doi.org/10.1590/S0100-736X2010001100009
- Selim H.M., Yamato O., Tajima M. & Maede Y. 1999. Rumen bacteria are involved in the onset of onion-induced hemolytic anemia in sheep. J. Vet. Med. Sci. 61(4):369-374. https://dx.doi.org/10.1292/jvms.61.369
- Spice R.N. 1976. Hemolytic anemia associated with ingestion of onions in a dog. Can. Vet. J. 17(7):181-183. <PMid:949673>
- Thorp F. & Harshfield G.S. 1939. Onion poisoning in horses. J. Am. Vet. Med. Assoc. 94:52-53.
- Tokarnia C.H., Britto M.F., Barbosa J.D., Peixoto P.V. & Döbereiner J. 2012. Plantas Tóxicas do Brasil para Animais de Produção. 2. ed. Helianthus, Rio de Janeiro. 566p.
- Tokarnia C.H., Chagas B.R., Chagas A.D. & Silva H.K. 1997. Anemia hemolítica causada por *Ditaxis desertorum* (Euphorbiaceae) em bovinos. Pesq. Vet. Bras. 17(3/4):112-116. https://dx.doi.org/10.1590/S0100-736X1997000300004
- Van Kampen K.R., James L.F. & Johnson A.E. 1970. Hemolytic anemia in sheep fed wild onion (*Allium validum*). J. Am. Vet. Med. Assoc. 156(3):328-332. <PMid:5413557>
- Verhoeff J., Hajer R. & Van Den Ingh T.S. 1985. Onion poisoning of young cattle. Vet. Rec. 117(19):497-498. https://dx.doi.org/10.1136/vr.117.19.497 <PMid:4082398>
- Williams H.H., Erickson B.N., Beach E.F. & Macy I.G. 1941. Biochemical studies of the blood of dogs with N-propyl disulfide anemia. J. Lab. Clin. Med. 26(6):996-1008. https://dx.doi.org/10.5555/uri:pii:S0022214341900768