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Amaranthus spp. poisoning in cattle grazing a pasture co-infested with cyanogenic *Sorghum sudanense*[1]

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ABSTRACT.- Alberti TS, Marcolongo-Pereira C, Moraes MS, Almeida RR, Zoia WA, Pereira MM, Raffi MB, Sallis ESV. ***Amaranthus* spp. poisoning in cattle grazing a pasture co-infested with cyanogenic *Sorghum sudanense*.** *Pesquisa Veterinária Brasileira 46:e07889, 2026*. Laboratório Regional de Diagnóstico, Faculdade de Veterinária, Universidade Federal de Pelotas, Capão do Leão, Cx. Postal 354, Pelotas, RS 96010-900, Brazil. E-mail: clairton.pereira@ifes.edu.br

An outbreak of acute mortality is described in a herd of 356 Red Angus cattle grazing a drought-affected pasture in southern Brazil co-infested with regrowing *Sorghum sudanense* and fruiting *Amaranthus* spp. Seventeen cattle (morbidity 4.77%) developed apathy, anorexia, tremors, exercise intolerance, and dark feces suggestive of melena two days after introduction to the paddock; clinical signs lasted approximately 24 hours before death, with a case fatality rate of 100%. Four affected cattle were necropsied. Main gross findings included moderate, multifocal subcutaneous petechiae, unclotted blood in the great vessels, severe, diffuse perirenal edema, and diffusely pale and soft kidneys. Histologically, all examined cattle had severe, diffuse acute tubular necrosis accompanied by intratubular casts and hyaline droplets, along with mild, multifocal centrilobular hepatic necrosis. These findings were consistent with *Amaranthus* spp.-induced nephrotoxicity. A qualitative picrate paper test on macerated *S. sudanense* samples was positive, indicating cyanogenic potential and raising the possibility of a concurrent contribution of cyanide poisoning, which could not be confirmed. This outbreak illustrates the diagnostic challenge posed by the co-occurrence of a nephrotoxic and a cyanogenic toxic plant in the same pasture. Also, it reinforces the importance of comprehensive toxicological investigation in such scenarios.

INDEX TERMS: Toxic plants, nephrotoxicity, acute tubular necrosis, hydrocyanic acid, ruminant.

RESUMO.- [**Intoxicação por *Amaranthus* spp. em bovinos mantidos em pastagens coinfestadas com *Sorghum sudanense* cianogênico.**] Descreve-se um surto de mortalidade aguda em um rebanho de 356 bovinos Red Angus mantidos em uma pastagem afetada pela estiagem, no sul do Brasil, coinfestada por *Sorghum sudanense* em rebrote e *Amaranthus* spp. em frutificação. Dois dias após a introdução no piquete, 17 bovinos (morbidade de 4,77%) desenvolveram apatia, anorexia, tremores, intolerância ao exercício e fezes escuras sugestivas de melena. Os sinais clínicos persistiram por aproximadamente 24 horas antes da morte, com letalidade de 100%. Quatro bovinos afetados foram necropsiados. Os principais achados macroscópicos incluíram petéquias subcutâneas multifocais moderadas, sangue não coagulado nos grandes vasos, edema perirrenal difuso acentuado e rins difusamente pálidos e amolecidos. Histologicamente, todos os bovinos examinados apresentaram necrose tubular aguda difusa acentuada, acompanhada de cilindros intratubulares e gotículas hialinas, além de necrose hepática centrolobular multifocal discreta. Esses achados foram compatíveis com nefrotoxicidade induzida por *Amaranthus* spp. O teste qualitativo em papel de picrato, realizado em amostras maceradas de *S. sudanense*, foi positivo, indicando potencial cianogênico e levantando a possibilidade de uma contribuição concomitante de intoxicação por cianeto, que não pôde ser confirmada. Este surto ilustra o desafio diagnóstico imposto pela coexistência de uma planta nefrotóxica e uma planta cianogênica na mesma pastagem, e reforça a importância da investigação toxicológica abrangente nesses cenários.

TERMOS DE INDEXAÇÃO: Plantas tóxicas, nefrotoxicidade, necrose tubular aguda, ácido cianídrico, ruminante.

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INTRODUCTION

Ingestion of toxic plants is an important cause of illness and death in cattle raised under extensive production systems in Brazil (Tokarnia et al. 2012, Riet-Correa et al. 2023). In these systems, pastures simultaneously harbor multiple toxic plant species (Welch et al. 2016). Nevertheless, when cattle graze paddocks containing more than one toxic species, the clinicopathological consequences of concurrent exposure remain poorly characterized, which can hinder recognition of overlapping toxic syndromes and complicate diagnostic and prognostic interpretation.

Sorghum sudanense (Sudan grass) is widely cultivated as a summer forage crop (Sousa et al. 2025). However, under specific environmental and phenological conditions, this species may develop substantial cyanogenic potential, particularly during early regrowth and periods of water stress (Giantin et al. 2024, Sousa et al. 2025). After ruminal hydrolysis, cyanogenic glycosides release hydrogen cyanide (HCN), which inhibits cytochrome c oxidase, causes histotoxic hypoxia, and may lead to peracute or acute death in affected cattle (Muwel et al. 2018, Giantin et al. 2024, Sousa et al. 2025). In contrast, *Amaranthus* spp. are common weeds in cultivated fields and disturbed pastureland. Under specific environmental and phenological conditions, particularly when plants are at the seed-setting or fruiting stage, ingestion may cause nephrotoxicity in cattle (Machado & Riet-Correa 2023, García et al. 2024, Machado et al. 2026). Although the toxic principle remains unidentified, *Amaranthus*-associated poisoning is characterized primarily by acute tubular injury and necrosis, predominantly in the proximal convoluted tubules, and in some cases, extending to segments of the nephron loop and distal nephron. This renal injury may progress to azotemia, uremia, edema, and death in severely affected cattle (Machado et al. 2026).

Although poisoning by *S. sudanense* and *Amaranthus* spp. has been well documented separately, outbreaks occurring in pastures co-infested with both plants are rarely described. Herein, we describe an outbreak of acute cattle mortality attributed to *Amaranthus* spp. poisoning in a pasture co-infested with cyanogenic *S. sudanense*, in which a concurrent contribution of cyanide could not be excluded. This report emphasizes the epidemiological setting, clinicopathological findings, and diagnostic challenges posed by the co-occurrence of nephrotoxic and cyanogenic toxic plants.

MATERIALS AND METHODS

Ethical approval. As the outbreak occurred spontaneously under natural grazing conditions and all necropsies and tissue collections were performed exclusively for routine diagnostic purposes, without euthanasia or additional animal handling for research, approval from the Institutional Animal Ethics Committee was not required.

A site visit was conducted at a property in Arroio Grande, Rio Grande do Sul, southern Brazil, to investigate acute cattle deaths. Epidemiological data, including herd management, pasture history, feeding practices, environmental conditions preceding the outbreak, and clinical course of affected animals, were obtained through interviews with fdoughtarm staff and direct observation. A pasture survey revealed that the affected cattle had been grazing a paddock with regrowing *Sorghum sudanense* heavily infested by fruiting *Amaranthus* spp. Both plants were identified based on macroscopic botanical characteristics. *Amaranthus* was identified only at the genus level, as species-level identification was not performed.

Based on the presence of both plants in the paddock, poisoning by *Amaranthus* spp., with a possible concurrent contribution of cyanide from *S. sudanense*, was suspected in the present case.

Four affected cattle underwent complete *post mortem* examination. Tissue samples from the thoracic, abdominal, and brain organs were collected, fixed in 10% neutral-buffered formalin, routinely processed, embedded in paraffin, sectioned at 3 μm, and stained with hematoxylin and eosin (HE) for histopathological evaluation.

Fresh macerated *S. sudanense* samples were tested for hydrocyanic acid (HCN) using the picrate paper test, a qualitative screening method. Filter paper strips impregnated with sodium carbonate and picric acid were suspended within sealed containers with the macerated plant material; a color change from yellow to orange-brown was interpreted as a positive result for HCN production. However, this method does not allow the quantification of HCN concentrations.

RESULTS

In February 2026, a herd of 356 Red Angus cattle from a farm in Arroio Grande, Rio Grande do Sul, southern Brazil, was introduced into a paddock containing regrowing *Sorghum sudanense*, which the farm owner reported having previously cultivated in the area (Fig. 1). The pasture had previously been grazed on three occasions without reported clinical disease. It was heavily infested with fruiting *Amaranthus* spp. (Fig. 1), as were other paddocks on the property. The outbreak occurred after a drought exceeding 20 days.

Two days after introduction into the paddock, 17 cattle (morbidity 4.77%, 17/356) developed apathy, anorexia, tremors, exercise intolerance, and dark feces suggestive of melena. Clinical signs lasted approximately 24 hours before death, with a case fatality rate of 100% (17/17). Upon identification of the initial case, the remaining 339 cattle were promptly moved from the paddock.

The main gross findings included moderate multifocal subcutaneous petechiae, unclotted blood in the great vessels, severe diffuse perirenal edema, and diffusely pale and soft kidneys (Fig. 2). Mild subcapsular renal petechiae and multifocal cortical hemorrhages were also observed in one animal (Fig. 3).



Fig. 1. *Amaranthus* spp. poisoning in cattle grazing a pasture co-infested with cyanogenic *Sorghum sudanense*. Paddock showing regrowth of *S. sudanense* heavily infested with fruiting *Amaranthus* spp.

The abomasal mucosa exhibited multifocal petechiae. The abomasal lumen contained a moderate amount of hemorrhagic content. The intestinal lumen contained dark digested content, consistent with melena. No abomasal ulcers were observed.

Histologically, all cattle examined had severe diffuse acute tubular necrosis (Fig. 4), accompanied by intratubular casts and hyaline droplets (Fig. 5). Mild multifocal centrilobular hepatic necrosis was also observed. The picrate paper test performed on fresh macerated *S. sudanense* samples yielded a positive result within 5 min, as evidenced by a color change from yellow to orange-brown, indicating the presence of hydrocyanic acid (Fig. 6–7).

DISCUSSION

Poisoning by *Amaranthus* spp. was diagnosed in this outbreak based on the epidemiological setting, clinical course, and characteristic renal lesions. Severe diffuse acute tubular necrosis, accompanied by intratubular casts and hyaline droplets in all necropsied cattle, is characteristic of *Amaranthus* spp.-associated nephrotoxicity and has been consistently reported in natural and experimental poisoning in cattle (Peixoto et al. 2003, Stigger et al. 2013, Melo et al. 2014, García et al. 2024, Machado et al. 2026). Likewise, perirenal edema, pale and soft kidneys, and renal petechiae observed grossly are compatible with the acute renal injury that characterizes this intoxication (Stigger et al. 2013, García et al. 2024, Machado et al. 2026). The relatively low morbidity (4.77%) observed in this outbreak likely reflects variation in plant intake among animals, as *Amaranthus* spp. poisoning generally depends on substantial consumption of toxic plant material.

Mild multifocal centrilobular hepatic necrosis was also observed. Centrilobular (zone 3) hepatocytes are particularly susceptible to hypoxic injury because they are located farthest from the incoming oxygenated blood, making centrilobular necrosis a common and nonspecific response to reduced tissue oxygenation in domestic animals (Cullen & Van Wettere 2025). In the present outbreak, this lesion may have resulted from terminal circulatory compromise associated with severe systemic illness, while histotoxic hypoxia caused by cyanide exposure represents another possible mechanism. Centrilobular necrosis may also develop during the agonal period as a consequence of impaired hepatic perfusion (Cullen & Van Wettere 2025). Thus, although this hepatic change supports hypoxic injury, it is nonspecific and cannot be attributed to a single mechanism.



Fig. 2. *Amaranthus* spp. poisoning in cattle grazing a pasture co-infested with cyanogenic *Sorghum sudanense*. Kidneys diffusely pale and soft.

The presence of regrowing *S. sudanense* represented an important differential diagnostic consideration in the affected paddock. The picrate paper test on plant samples was positive, confirming the cyanogenic potential of the forage. Drought and early regrowth are recognized factors that increase the cyanogenic potential of sorghum species (Muwel et al. 2018, Giantin et al. 2024, Sousa et al. 2025), and both conditions were present during the outbreak. Nevertheless, the available evidence was insufficient to establish concurrent cyanide poisoning. The picrate paper test is a qualitative screening method and demonstrates cyanogenic potential rather than the amount of HCN to which the cattle were actually exposed. Moreover, the test was performed on plant material rather than ruminal contents. Quantitative determination of cyanide in forage and ruminal contents, preferably using a validated analytical method such as gas chromatography-mass spectrometry, would therefore have been necessary to establish toxic exposure firmly.



Fig. 3. *Amaranthus* spp. poisoning in cattle grazing a pasture co-infested with cyanogenic *Sorghum sudanense*. Subcapsular renal petechiae observed in one animal.

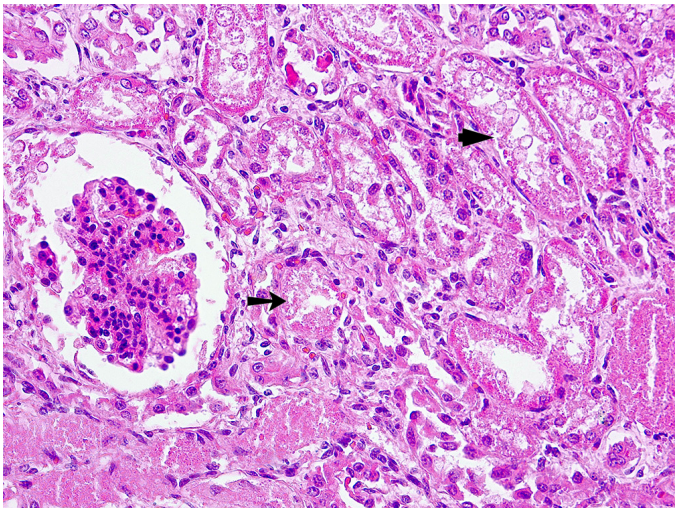


Fig. 4. *Amaranthus* spp. poisoning in cattle grazing a pasture co-infested with cyanogenic *Sorghum sudanense*. Juxtamedullary nephrons showing severe, diffuse acute tubular necrosis of the proximal convoluted tubules (black arrow) and distal convoluted tubules (arrowhead). HE, obj. 400x.

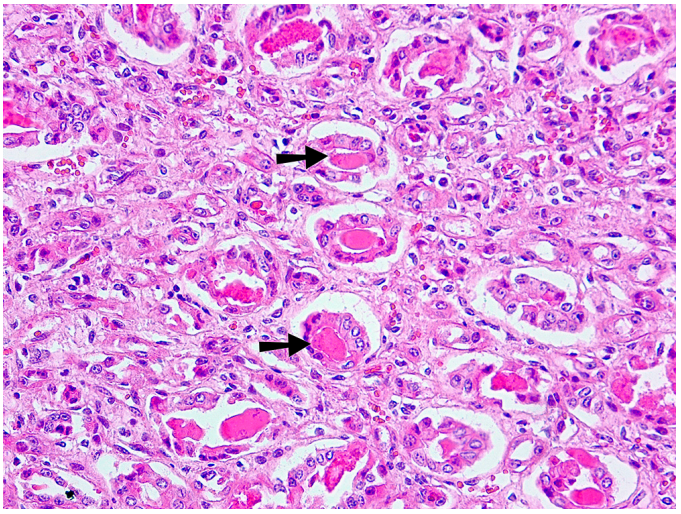


Fig. 5. *Amaranthus* spp. poisoning in cattle grazing a pasture co-infested with cyanogenic *Sorghum sudanense*. Medullary region showing collecting ducts with intratubular hyaline casts and epithelial desquamation (black arrows). HE, obj. 400x.



Fig. 6–7. *Amaranthus* spp. poisoning in cattle grazing a pasture co-infested with cyanogenic *Sorghum sudanense*. Picrate paper test performed on macerated *S. sudanense* samples. (**6**) Paper strip before exposure (yellow). (**7**) Positive result after 5 minutes, evidenced by a color change to orange-brown, indicating the presence of hydrocyanic acid.

The clinical course also provided limited support for cyanide intoxication. Cyanide poisoning is typically hyperacute, with death occurring within minutes to a few hours after ingestion (Tokarnia et al. 2012, Riet-Correa & Mendonça 2023), whereas affected cattle in this outbreak survived for approximately 24 hours. In addition, gross lesions associated with cyanide poisoning are generally nonspecific and inconsistent (Kennedy et al. 2021), and the classically described bright-red coloration of venous blood was not documented. Consequently, although the presence of cyanogenic *S. sudanense* indicates that exposure to cyanide was possible and may have contributed to the clinical outcome, its role in the outbreak could not be confirmed.

This outbreak illustrates the diagnostic complexity that can arise when plants with different toxic mechanisms occur simultaneously in the same pasture. In this case, the epidemiological, gross, and histological findings strongly supported *Amaranthus* spp. poisoning as the primary diagnosis, particularly because of the characteristic and severe renal lesions. By contrast, the contribution of cyanogenic *S. sudanense* remains uncertain because evidence of plant cyanogenic potential was not accompanied by quantitative confirmation of toxic exposure. The coexistence of both plants nevertheless highlights the possibility that more than one toxic mechanism may contribute to acute cattle mortality under field conditions. These findings reinforce the importance of integrating epidemiological, clinical, pathological, and toxicological data, particularly when multiple toxic plants are present, and obtaining appropriate samples early in the investigation for quantitative toxin determination in both forage and biological specimens.

CONCLUSION

Poisoning by *Amaranthus* spp. was identified as the primary cause of this outbreak of acute mortality in cattle based on the epidemiological, clinical, and pathological findings. The concurrent presence of cyanogenic *Sorghum sudanense* raises the possibility that cyanide exposure may have contributed to the clinical outcome, although this could not be confirmed in this case. This outbreak highlights the diagnostic challenges of simultaneous toxic plant exposure with different mechanisms of action. Also, it underscores the importance of integrating epidemiological, pathological, and toxicological data when investigating acute livestock mortalities.

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Credit author statement.- Tainá dos Santos Alberti: Conceptualization, Investigation, Formal analysis, Methodology, Writing – original draft. Clairton Marcolongo-Pereira and Eliza Simone V. Sallis: Conceptualization, Funding acquisition, Resources, Supervision, Writing – original draft, Writing – review & editing. Marta Santos de Moraes, Roseli Rodrigues de Almeida, Wesley Aquino Zoia and Matheus Mendes Pereira: Investigation, Formal analysis, Methodology. Margarida Buss Raffi: Resources, Writing – review & editing.

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