In May 2007, 111 adult mixed-breed cattle in southern Brazil were placed onto 110 acres of a new pasture that had previously been used for a soybean crop. Eight days after the introduction of the animals onto the pasture, clinical signs of illness were noted and deaths started to occur; 11 cows died in a period of 15 days after the onset of clinical signs.

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History

In May 2007, 111 adult mixed-breed cattle in southern Brazil were placed onto 110 acres of a new pasture that had previously been used for a soybean crop. Eight days after the introduction of the animals onto the pasture, clinical signs of illness were noted and deaths started to occur; 11 cows died in a period of 15 days after the onset of clinical signs.

Clinical and Gross Findings

The duration of clinical signs was approximately 3 to 7 days. Among the 11 affected cows, clinical signs included anorexia, staggering gait, incoordination, sternal recumbency, dark red urine, and death. Blood and urine samples were obtained from 1 affected cow before euthanasia (by means of an IV overdose injection of pentobarbital solution) and necropsy 3 days after onset of clinical signs. Gross changes detected during necropsy included extensive, well-demarcated, pale white areas that were distributed bilaterally throughout the skeletal muscles of the thigh regions (especially the semimembranosus, semitendinosus, quadriceps femoris, and biceps femoris muscles) and upper portions of the forelimbs and shoulders (supraspinatus and infraspinatus muscles; Figure 1). The urinary bladder contained dark red urine. There was serous atrophy of pericardial fat.

Formulate differential diagnoses from the history, clinical findings, and Figure 1—then turn the page—

Figure 1—Photographs of thigh muscles (A) and opened urinary bladder (B) of a cow that was euthanized because of anorexia, staggering gait, incoordination, sternal recumbency, and excretion of dark red urine of 7 days’ duration. In panel A, a cross section of the thigh muscles reveals extensive, well-demarcated, pale white areas (left) of myodegeneration and necrosis. In panel B, the bladder contains dark red urine.
Histopathologic Findings and Diagnostic Confirmation

Histologic examination revealed extensive areas of monophasic skeletal muscle changes characterized by hyaline myofiber degeneration and necrosis with mild inflammation (Figure 2). Affected myofibers had swollen and hypercoisinophilic sarcoplasm with loss of cross striations (hyaline degeneration) or segmentally fragmented sarcoplasm (necrosis) and occasional accumulations of neutrophils and scattered macrophages. No potential etiologic agents were observed in multiple sections of skeletal muscle. Serum biochemical analyses revealed high creatinine kinase activity (no values available), and urinalysis revealed the presence of myoglobin in the urine. No other laboratory abnormalities were detected. An in situ inspection of the pasture on which affected cattle had been grazing revealed heavy infestation by *Senna occidentalis* (coffee senna or coffee weed, formerly known as *Cassia occidentalis*; Figure 3), which is the most common cause of skeletal myonecrosis in cattle in southern Brazil. The referring veterinarian reported that most cows (including the necropsied cow) had been feeding on these plants.

**Morphologic Diagnosis and Case Summary**

Morphologic diagnosis: severe, multifocal, bilateral, monophasic skeletal myonecrosis of muscles of the thigh regions and upper portions of the forelimbs and shoulders.

Case summary: toxic skeletal myonecrosis and myoglobinuria in a cow.

**Comments**

In the case described in the present report, a presumptive diagnosis of toxic skeletal myonecrosis was made on the basis of the gross and histologic findings along with the laboratory test results, which indicated possible skeletal muscle damage and consequent myoglobinuria. Diagnostic confirmation was achieved by the detection of fresh *S. occidentalis* on the pasture and by the fact that specimens of the plant (especially leaves and stems) had been consumed by the affected cows. Ingestion of toxic plants is one of the most important causes of death in ruminants in southern Brazil, leading to important economic losses to the agriculture and food industry.\(^1\)\(^-\)\(^3\) *Senna occidentalis* is an annual shrub that sprouts during the spring and blossoms during the summer. The plant grows on pastures and roadsides throughout extensive tropical and subtropical areas, including southern Brazil and the southeastern United States.\(^4\) Clinical disease in domestic animals has been associated mainly with skeletal muscle necrosis and less often with cardiac myonecrosis and hepatocellular necrosis.\(^5\) Toxic effects may result either from direct ingestion of the plant, as described in the present report, or from ingestion of crop weeds (eg, soybean, corn, or sorghum) contaminated with the seeds of *S. occidentalis*.\(^1\)\(^-\)\(^4\)

Seeds are considered the most toxic.
part of the plant, but pods, leaves, and stems are also toxic.\textsuperscript{2} Multiple toxic compounds have been extracted from \textit{S occidentalis}, but the toxin responsible for the muscular changes, which develop as a consequence of uncoupling of oxidative phosphorylation, has yet to be identified.\textsuperscript{3,5} Naturally occurring poisoning in cattle, pigs, wild boars, and horses has been described, but \textit{S occidentalis}-associated toxicosis has been experimentally induced in other species, such as sheep, goats, poultry, and rabbits.\textsuperscript{2,5,7} Although no frost had occurred in the geographic area of the cattle farm before the outbreak described in the present report, it is common for the animals on pasture to ingest \textit{S occidentalis} after frosts, conditions that usually make it more palatable.\textsuperscript{5}

Similar to what occurred on the cattle farm in the present report, cases of \textit{S occidentalis} toxicosis in cattle are typically clustered in outbreaks with high mortality rates.\textsuperscript{3} Clinical signs are associated with the extensive pathological changes in skeletal muscles and include weakness, stumbling gait, and anorexia that typically progress to recumbency and myoglobinuria with increases in serum activities of muscle-derived enzymes.\textsuperscript{1,2,8} Recumbent bovids typically die a few days after the onset of clinical signs, presumably as a result of cardiac failure secondary to hyperkalemia rather than a consequence of degenerative myocardial changes, as previously thought.\textsuperscript{6} This hypothesis is supported by the fact that cardiac lesions are not typically detected in naturally occurring \textit{S occidentalis} poisoning in cattle.\textsuperscript{2} However, early degenerative myocardial changes (that would not be detected during routine histologic evaluation of cardiac tissue), cardiac mitochondrial changes (that would be evident only via electron microscopy), and renal changes related to myoglobinuria have all been implicated as possible factors that could impair cardiac function or cause metabolic abnormalities and consequently lead to death of affected individuals.\textsuperscript{6} The gross and histopathologic changes observed in the cow of the present report were typical of findings in cattle with \textit{S occidentalis} poisoning.\textsuperscript{1,2} Skeletal muscle changes consisting of myodegeneration and necrosis appear as pale white areas and are mainly localized to large muscle groups in the hind limbs.\textsuperscript{1,2,6} Similar to the cardiac changes, hepatocellular vacuolation with single-cell necrosis, renal tubular degeneration, and splenic lymphoid necrosis have all been described in association with \textit{S occidentalis} toxicosis in cattle but are not consistent findings in cases of naturally occurring poisonings.\textsuperscript{2} None of these changes were observed in the cow of the present report. Although the clinical and pathological findings of \textit{S occidentalis} poisoning are similar in cattle, pigs, and wild boars,\textsuperscript{2,7} \textit{S occidentalis} toxicosis has been associated with acute neurologic disease attributed to hepatocellular necrosis and hepatic encephalopathy in horses.\textsuperscript{9}

For the outbreak described in the present report, differential diagnoses were based on the presence of dark red urine in the necropsied cow and other affected animals. Therefore, diseases that induce hemoglobinuria, hematuria, and myoglobinuria in cattle were considered.\textsuperscript{8,10,11} On the basis of the geographic location of the cattle farm, the major differential diagnoses for hemoglobinuria in cattle included babesiosis caused by \textit{Babesia bovis} or \textit{Babesia bigemina}, bacillary hemoglobinuria caused by \textit{Clostridium baemolyticum}, leptospirosis, \textit{Brachyia radicans} toxicosis, and copper toxicosis. Differential diagnoses for hematuria included chronic \textit{Pteridium aquilinum} toxicosis (enzootic hematuria), malignant catarrhal fever, and pyleonephritis; differential diagnoses for myoglobinuria included \textit{S occidentalis} toxicosis, ionophore toxicosis, vitamin E and selenium deficiency, and prolonged recumbency. Following necropsy and detection of the gross changes in the skeletal muscles of the cow of this report, the list of differential diagnoses was narrowed to diseases that cause extensive, bilateral skeletal muscular damage with possible consequent myoglobinuria.

A presumptive diagnosis of \textit{S occidentalis} toxicosis may be made on the basis of the clinical and pathological findings in affected animals and by the detection of the plant in the pasture or as a contaminant of crop weeds. Diagnostic confirmation may be achieved at necropsy through observation of parts of the plant within the gastrointestinal tract of affected animals.\textsuperscript{8} The presence of \textit{S occidentalis} on the pasture and the fact that affected individuals were observed consuming the plant, along with findings from the necropsied cow of the present report, were sufficient for the diagnosis of toxic myonecrosis attributable to \textit{S occidentalis} poisoning in this cattle herd. There is no specific or effective treatment for \textit{S occidentalis} poisoning, and the main prophylactic plan should always target measures that would avoid contamination of pastures or crop weeds by \textit{S occidentalis}.\textsuperscript{8}

\section*{References}