Secondary hepatogenous copper toxicosis associated with spontaneous poisoning by *Senecio brasiliensis* in sheep

M.R.S. Ilha¹, A.P. Loretti¹,², S.S. Barros³, C.S.L. Barros⁴

*Senecio brasiliensis* poisoning is responsible for 50% of plant poisoning of cattle in Rio Grande do Sul, Brazil. Losses in the Brazilian cattle industry due to this plant toxicosis are estimated at $7.5 million annually. Sheep are resistant to pyrrolizidine alkaloid (PA) poisoning and are used to control *S. brasiliensis* growth in contaminated paddocks. In a previous study, secondary hepatogenous copper toxicosis was observed in sheep experimentally dosed with *S. brasiliensis* from this geographic area (Barros CSL et al. Pesq Vet Bras. 8:110-116, 1989).

This study is part of an investigation of an outbreak of *S. brasiliensis* hepatotoxicosis in sheep in southern Brazil. Fifty four percent of the flock (51/94 sheep) that had access to *S. brasiliensis* died, most of them succumbing after an acute fatal hemolytic crisis. We describe here the clinical and pathological findings of six sheep naturally poisoned by *S. brasiliensis* that died of secondary hepatogenous copper toxicosis.

The flock had been grazing for 7 months in paddocks of native pasture heavily infested with *S. brasiliensis*. Clinical signs included photodermatitis, progressive emaciation, apathy, weakness, lethargy, aimless walking, unsteady gait, jaundice and hemoglobinuria. Necropsy findings included severe jaundice, brown urine, and dark, mottled, swollen kidneys. These sheep had small, firm livers with small randomly, scattered regenerative nodules. Histopathological findings included accumulation of hemoglobin and hemosiderin in renal tubular epithelial cells and hemoglobin casts. Megalocytosis, biliary ductal proliferation and moderate periportal fibrosis were also observed. Ceroid and copper-laden macrophages were scattered throughout the hepatic parenchyma or formed small aggregates in the portal triads. Single cell necrosis and scattered foci of hepatocellular necrosis were present. Spongy degeneration of the cerebral white matter was observed. Electron microscopy revealed intracellular edema and mitochondrial degeneration in renal proximal convoluted tubular epithelial cells. Ultrastructural changes in the liver included accumulation of numerous lipid droplets in the cytoplasm of the hepatocytes and lysosomes containing electron-dense structures that corresponded in most of the cases to ceroid-lipofuscin. Chemical analysis by atomic absorption spectrophotometry revealed high concentrations of copper in liver (369 - 1248 ppm dry matter) and kidney (152 - 687 ppm). In this study, quantitative analysis of copper and molybdenum in native pasture and in *S. brasiliensis* specimens were not done. It is reported that the Cu:Mo ratio in the native pasture from this area can vary from 11 to 49 and that Cu:Mo ratio above 10 in the pasture is considered potentially toxic for sheep that have ingested PA-containing plants.

*S. brasiliensis* spontaneous poisoning can happen when sheep are kept in heavily infested paddocks for a prolonged period of time as in this case; these sheep also can develop secondary hepatogenous copper toxicosis under this circumstance. A similar situation has been reported in Australia due to the PA-containing plants *Heliotropium* spp and *Echium plantagineum*.

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¹Department of Pathobiology, Ontario Veterinary College, University of Guelph, Guelph, ON, Canada.
²Faculty Veterinary Medicine, Federal University Rio Grande do Sul (UFRGS), Porto Alegre, RS, Brazil
³Faculty of Veterinary Medicine, Federal University of Pelotas (UFPel), Pelotas, RS, Brazil
⁴Department of Pathology, Federal University of Santa Maria (UFSM), Santa Maria, RS, Brazil

*To be considered for graduate student award*