Intoxicação espontânea por *Senecio brasiliensis* (Asteraceae) em ovinos no Rio Grande do Sul

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An outbreak of spontaneous *Senecio brasiliensis* poisoning in grazing sheep in the county of Mata, Rio Grande do Sul, southern Brazil, is described. The disease occurred on one farm in middle January 1997. Fifty-one (54.2%) out of 94 sheep were affected, and 50 animals (53.2%) died. This flock of sheep had been grazing for approximately 7 months (from June 1996 to January 1997) in paddocks heavily infested with *S. brasiliensis*. Clinical signs included photodermatitis, progressive emaciation, apathy, weakness, neurological signs such as drowsiness, aimless walking and unsteady gait, jaundice and hemoglobinuria. There was amelioration of the skin lesions in those sheep that developed hepaticogenous photosensitization. Main necropsy findings in 9 sheep included small, firm, tan or greenish liver with few to numerous small, yellowish, well-circumscribed nodules measuring up to 3 mm in diameter and randomly scattered throughout the hepatic parenchyma. There was also marked distension of the gallbladder which contained large amounts of inspissated, dark green bile and straw-colored cavitary effusions (hydropericardium and ascitis). Five sheep developed lethal acute hemolytic crisis, secondary to massive release into the blood stream of copper accumulated in the liver (hepatogenous chronic copper poisoning). Apart from the aforementioned liver lesions, other gross findings in those animals included severe and diffuse jaundice, dark brown urine (hemoglobinuria) and swollen, friable, finely stippled or diffusely dark kidneys. The main histopathological findings included hepatomegaly, biliary ductal proliferation ( bile duct hyperplasia) and moderate periportal fibrosis. The portal triads were infiltrated with variable numbers of mononuclear cells. There was heavy accumulation of brownish pigment in macrophages identified as ceroid or copper with PAS and rhodamine stainings, respectively. Those ceroid and copper-laden macrophages were scattered on the remnant hepatic parenchyma or formed small aggregates in the portal triads. Main histopathological findings in the kidneys of 5 sheep, that developed fatal hepatogenous chronic copper poisoning, included tubular nephrosis, accumulation of hemoglobin and hemosiderin in epithelial tubular cells and hemoglobin casts (hemoglobinuric nephrosis). Morphological evidence of hepatic encephalopathy included spongy degeneration (*status spongiosus*) of the...