HEPATIC ENCEPHALOMYELOPATHY IN 10 GOAT KIDS ASSOCIATED WITH CONGENITAL PORTOSYSTEMIC SHUNTING (CPSS)

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Ten goat kids (2 live and 8 dead) of various breeds, between 1.5 and 5 months of age, and with a body mass ranging from 3.67 to 18 kg were submitted for necropsy to the California Animal Health and Food Safety Laboratory System (CAHFS), or the Veterinary Medical Teaching Hospital (VMTH), School of Veterinary Medicine, University of California, Davis between 1999 and 2011. The history included two or more of the following clinical signs: ataxia, circling, blindness, seizures, teeth grinding, opisthotonus, paddling, general weakness, and ill thrift. Results of serum bile acids tests from 2 animals were 134 and 209 μmol/l (reference intervals: 0-50 μmol/l). Gross necropsy revealed that animals were in poor to fair body condition and had minimal fat reserve. Liver weights from 3 animals were 76 g (2% of the body weight), 280 g (1.8%), and 300 g (1.9%). Histologically, all animals had bilateral and symmetric spongy degeneration throughout the cerebrum, midbrain, cerebellum, brainstem, and spinal cord, more prominently at the white/grey matter junction. In three goats, proliferation of Alzheimer type II astrocytes were noted in the cerebral cortex and adjacent cerebral white matter. Histological lesions in the liver included atrophy of the hepatic parenchyma, small hepatocytes, increased numbers of arteriolar profiles, oval cell hyperplasia, and hypoplasia or absence of portal veins, and were consistent with congenital portosystemic shunting (cPSS). The clinical and pathological findings in all 10 goats were consistent with hepatic encephalopathy. Spongy degeneration of the CNS in these cases resulted from liver failure due to cPSS. cPSS should be considered in the differential diagnosis of young goats with a history of weakness, ill thrift, and neurological signs.