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Reference No.:

Clinical Abstract:

A 49 year old man with a history of binge drinking drank steadily for three days. On the third day he vomited coffee ground material several times and began to complain of headache, blurred vision, generalized weakness, and muscle pain, following which he became progressively lethargic. At a local hospital the vital signs were normal and a neurologic examination showed no focal signs. Following a brief period of agitation, he suddenly collapsed and became apneic and hypotensive. He was rapidly intubated and respiratory support was established. Serum electrolytes were sodium 146 mEq/L, potassium 3.8 mEq/L, chloride 84 mEq/L, and CO_2 8 mEq/L. Arterial blood pH was 6.84, pO_2 175, and pCO_2 22. The patient was treated with bicarbonate, dexamethasone, dopamine, furosemide, oxygen, and physostigmine, and then transferred to the Medical Center Hospital of Vermont

Upon arrival the patient was deeply comatose and hyporeflexic. Repeat serum electrolytes gave a potassium of 2.9 mEq/L and a CO_2 of 3 mEq/L. CSF examination disclosed an opening pressure of 220 mm H_2O , a normal protein and no cells. Therapy was continued with bicarbonate, hemodialysis, and dexamethasone. The patient remained deeply comatose and support was discontinued on the sixth hospital day.

The general autopsy showed superficial ulceration of the trachea secondary to intubation, and mild to moderate centrilobular fatty change of the liver. There was no evidence of pancreatitis or of renal tubular necrosis. The brain was edematous and weighed 1540 grams following fixation. The ventricular spaces were nearly obliterated, and uncus and cerebellar herniation was present. Bilateral hemorrhagic necrosis of the putamen was evident. The white matter of the frontal, temporal and occipital lobes appeared greyish and necrotic with a thin rim of normal appearing white matter at the grey-white matter junction.

Material submitted: One 2X2 Kodachrome of cerebral hemispheres
at the coronal level of the mammillary bodies
One LFB/H+E slide from frontal lobe

Points for discussion: 1. Diagnosis
2. Pathogenesis