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Clinical Abstract:

This 68 year old man presented with complaints of bilateral fluctuating deafness and left-sided head pain. Some ten months later, he was admitted with an acute illness involving abnormal limb movements which may possibly have been seizures, and an acute decrease in level of consciousness. This was associated with fever and neck stiffness, and the spinal fluid showed a pleocytosis with lymphocyte predominance. The protein was also elevated but no organisms were cultured. Investigations failed to reveal an infective agent, and a parameningeal infection was ruled out. There was no response to a wide range of antibiotics and the patient only improved when hydrocortisone treatment was given. On this latter treatment there was improvement in his level of consciousness, resolution of temperature, and the patient recovered with persisting deafness and ataxia. Attempts to reduce the steroid therapy resulted in recurrence of his acute symptoms, and he was maintained on long-term steroid therapy. Examination six months after the acute illness showed persistent sensory neural deafness and evidence of steroid therapy with capillary fragility and proximal weakness and wasting. A repeat lumbar puncture at this time showed 2 polymorphs and 1 lymphocyte with an elevated protein at 78 mg%. Temporal artery biopsy was normal and muscle biopsy was inconclusive. Fourteen months after his acute illness, he developed a perforated duodenal ulcer which was surgically treated. He remained in a long-term care facility until August 1980, when he developed shortness of breath, was noted to be febrile and generally lethargic. He responded to an increased dose of Prednisone and diuretic therapy. Chest x-rays revealed opacifications in both lower lobes thought to be due to atelectasis. He was discharged but readmitted the following day because of weakness and inability to take food by mouth. He deteriorated and died a week after his admission.

Necropsy Findings:

The cause of death was massive pulmonary embolus. The prostatic veins were thrombosed. There was coronary atherosclerosis and minor myocardial scarring. The falx cerebri and the dura below the frontal poles and the left temporal and parietal lobes was slightly thickened and adherent to the brain. Fibrous thickening was also noted in the tentorium and roof of the anterior 4th ventricle.

MATERIAL SUBMITTED: H & E section from falx cerebri