

Case 6

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Clinical Abstract:

This 37 year old man developed supraclavicular and hilar adenopathy four years before his death. A lymph node biopsy was interpreted as nodular sclerosing Hodgkin's disease. Chemotherapy resulted in complete remission for two years at which time he again had a good response to chemotherapy. He was continued on maintenance chemotherapy every other month. 3.5 months prior to his death, he noted fatigue, weight loss and low grade fever. He was noted to have pulmonary nodules and enlarged mediastinal nodes which again responded to chemotherapy. He then developed headache with temperature of 99.2 and papilledema. Lumbar puncture showed opening pressure of 250, clear CSF, 5 red cells, 85 white cells (70% monos), negative cytology, protein 294 mg%, glucose less than 10 mg%. Gram stain, India ink and cryptococcal antigen tests were negative. All cultures of blood, spinal fluid and bone marrow remained negative. The patient received intravenous Ampicillin, Amphotericin and oral 5-fluorocytosine. His headaches improved but the CSF findings were unchanged. Ampicillin and Amphotericin were stopped after two weeks and one week, respectively. Chemotherapy was continued and intrathecal methotrexate was begun. The latter did not cause any improvement of the CSF abnormalities or the patient's symptoms. One week prior to his death, an Ommaya reservoir was placed in the frontal lobe and the brain and meninges were biopsied. The biopsy showed no pathological changes. While in the recovery room, the patient developed seizures and decerebrate posturing necessitating reopening the operative site. No evidence of increased pressure or hemorrhage was noted. The patient continued to deteriorate and died.

Necropsy Findings: Hodgkin's disease, nodular sclerosing type involving hilar lymph nodes and lung, pulmonary congestion and edema.

The brain was swollen. The leptomeninges at the base of the brain were thickened, firm and fibrous, particularly about the optic chiasm and in the interpeduncular cistern. There was extensive softening of the basal ganglia, internal capsule and brain stem, bilaterally.

MATERIAL SUBMITTED: Optic chiasm or cerebral cortex (H & E and unstained).

Point for Discussion:

What is the cause of the lesions in the brain?