

CASE 4

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This 38 year-old man was found to have a pulmonary mass which was diagnosed by biopsy as adenocarcinoma. He was treated with Cytoxan and steroids. Several months later he experienced sudden onset of low back pain, severe paraplegia, sensory loss in both lower extremities and loss of bladder and rectal control. He gave the additional history that at age 16 he had had a mediastinal mass diagnosed, which "disappeared" after radiation therapy. Examination showed a severe spastic paraplegia, a sensory level beginning at T-11, and becoming dense below L-1, and absent sphincter control. Spinal fluid contained 29 lymphocytes but normal protein. A myelogram was normal. About 3 months later he developed moderate weakness of the left upper extremity. Examination showed persistence of paraplegia and lower extremity sensory loss. There was generalized muscle wasting. His left upper extremity was weak with increased tone, hyperreflexia, clumsiness of the left hand, but no sensory loss. His subsequent course was gradually downhill. He died 8 months after onset of his neurological disease.

Necropsy showed a 10 x 7 x 4 cm anterior mediastinal malignant tumor which had glandular features. Pleural and lung tumor deposits showed both glandular and undifferentiated malignant changes. A few small hepatic metastases were found. The central nervous system showed major spinal cord lesions with extensive perivascular lymphocytic infiltration, even extending into adjacent meninges. There was neuronal and myelin destruction with considerable gitter cell reaction.

Diagnoses:

1. Necrotizing myelitis due to remote effects of malignancy.
2. Anterior mediastinal malignant tumor, possibly of teratomatous origin.

Submitted are 1 gross Kodachrome, 1 slide stained with H and E, 1 slide stained for myelin, and 1 unstained slide.

Points for discussion:

1. Evidence for relationship between malignancy and remote effects on CNS, e.g. myelopathy, neuropathy, myopathy, encephalopathy.
2. Incidence of malignancy and such remote effects.
3. Mechanisms by which such CNS changes may occur. What evidence for such mechanisms operating? How to demonstrate such mechanisms?