CASE REPORTS

SPINAL-CORD COMPRESSION SECONDARY TO GAUCHER’S DISEASE

CASE REPORT

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Fig. 1. Normal anteroposterior and lateral views of thoracolumbar spine, April 1961.

Gaucher’s disease is a lesion of the reticuloendothelial system. Normal cells are replaced by histiocytes containing the cerebrosid keratin.2,7 When Gaucher first described this disease in 1882 the marked prevalence of involvement of bones was not appreciated. In 1909 Risel6 first noted the characteristic microscopic lesions of bone in Gaucher’s disease. af Klercker4 and Junghagen3 did the first complete roentgen-ray studies in this disease. Since then the true incidence of the skeletal lesions has been recognized.10 Indeed, Snapper has stated that the marrow of the bone is always involved.8,9 Certainly after splenic involvement, osteoarticular pathology causes the most important signs and symptoms.2 Though the usual site of pathology of the bone is the femur, the vertebral bodies are involved frequently.1,5 Keratin-laden histiocytes invade and destroy normal trabecular structure with resultant vertebral collapse and gibbus formation. It would be expected that spinal-cord compression may occur following such collapse. About 300 cases of Gaucher’s disease have now been described in the world literature but there does not appear to be

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any report of spinal-cord compression following vertebral collapse. Such a case was seen recently at the Neurological Institute of New York and forms the basis of this report.

**CASE REPORT**

*History.* The patient was a 41-year-old Jewish female in whom a diagnosis of Gaucher's disease was made in 1947 and a splenectomy was carried out. In 1951 pain in the back and hip developed, radiating into the right thigh. On neurological examination there was some weakness of muscles of the right thigh, and the right knee and ankle jerks were depressed slightly. Roentgenograms of the spine and right leg were normal. Symptoms disappeared in 2 months. This pain recurred in December 1953 and roentgenograms then showed irregular translucencies and increased densities of the right pubic bone, hip and sacrum. There was minimal weakness of flexion of the right hip; the remainder of the neurological findings were normal. Again symptoms subsided spontaneously. In 1955 pain recurred. On examination there was limitation of motion of the right hip. There were no

*Fig. 8.* Complete myelographic block at L1 caused by extradural mass. Patient is in the 30° head-down position.