COMPRESSION OF SPINAL CORD BY OSTEITIS DEFORMANS (PAGET’S DISEASE), GIANT-CELL TUMOR AND POLYOSTOTIC FIBROUS DYSPLASIA (ALBRIGHT’S SYNDROME) OF VERTEBRAE

A REPORT OF FOUR CASES

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(Received for publication March 17, 1951)

Spinal cord compression by a lesion of the vertebra is most often the result of traumatic injury, metastatic carcinoma, infiltrating sarcoma, tuberculous spondylitis, pyogenic osteomyelitis, osteoarthritis, infiltrating leukemia or multiple myeloma. Primary osteogenic sarcoma and benign neoplasms of the spine rarely cause compression of the spinal cord. Although osteitis deformans (Paget’s disease) is not an uncommon disease and frequently involves the spine, it rarely causes compression of the spinal cord. Polyostotic fibrous dysplasia belongs to the rare category of bone disease. The vertebrae are scarcely ever affected, and involvement of the spinal cord has never before been recorded in this condition to our knowledge.

OSTEITIS DEFORMANS

Among 274,397 admissions to the Mount Sinai Hospital in a period of 17 years from October 1933 to October 1950, 131 were for osteitis deformans, an incidence of approximately 1 to every 2,000 admissions. Of these patients, only 1 had paraplegia as a result of spinal cord compression by the hypertrophic vertebral. Following is a brief report of this case:


Fifteen months before admission the patient began to have difficulty in walking, associated with numbness of both lower extremities. For the last 3 weeks he had been unable to walk unless assisted and could not stand without support.

Examination. There was marked weakness in both lower limbs, especially the left. The ankle jerk was absent on the left and markedly exaggerated on the right. Other deep tendon reflexes of the lower limbs were all exaggerated but equal on both sides. Signs of Babinski, Chaddock, Oppenheim and Rossolimo were elicited on both sides. Hypalgesia was demonstrated from below D4. Vibration sense was absent from below the D4 spinous process. Deep and position sensation were lost from below the ankles. Abdominal reflexes were all present and equal on both sides. There was a kyphosis of the upper dorsal spine.

Laboratory findings: Hb. 15 gm., WBC 6,550 with 61 per cent neutrocytes. E.S.R. 15 mm./hr., fasting sugar 140 mg. per cent, urea nitrogen 17 mg. per cent,
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serum albumin 3.4, globulin 3.0, acid phosphatase 8 KAU, alkaline phosphatase 40 KAU, cholesterol 480 mg. per cent, phosphorus 3.1 mg. per cent, and calcium 10.5 mg. per cent.

Roentgenograms of spine and pelvis revealed loss of normal homogeneous cortical density of the bones of the pelvis and almost all of the vertebral bodies. The cortex was thickened and the trabeculae were markedly coarsened by a wavy thick bony meshwork with radiolucent interstices. Small cyst-like areas were scattered between the heavy trabeculae. There was marked flattening of the D3 vertebra with a similar coarsened trabecular pattern and biconcavity of its upper and lower surfaces (Fig. 1). The skull showed several small areas of decreased density as well as a generalized sclerosis. Diagnosis: Generalized osteitis deformans (Paget's disease).

Course. On the 11th hospital day, myelography was done. Before the instillation of pantopaque, manometric test disclosed a complete block of the CSF flow. The fluid was xanthochromic. The myelogram revealed a complete block to the cephalad flow of the pantopaque at the level of the D3 vertebra (Fig. 2). There was an irregular rounded capping to the proximal end of the column, suggesting an extradural lesion.

Operation. On the next day laminectomy was

Fig. 1. Case 1. Paget's disease. Posteroanterior view of dorsal spine showing coarsened trabecular pattern of the upper segments.

Fig. 2. Case 1. Paget's disease. Myelogram showing obstruction at level of D3.