PARAPLEGIA FROM RUPTURED LUMBAR DISCS IN ACHONDROPLASTIC DWARFS

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A herniated nucleus pulposus can be especially serious in the achondroplastic dwarf because of his abnormally narrow spinal canal. Two such dwarfs were operated on by us during the past year with relief of pain and paralysis caused by a ruptured nucleus pulposus between the 2nd and 3rd lumbar vertebrae. Although a number of cases of achondroplastic dwarfs with paralysis of the lower extremities are recorded in the literature, we could find no previous report of a ruptured disc being the cause of paralysis in these cases.

In appearance the achondroplastic dwarf differs from the normal because of the marked shortening of his extremities and the unusual shape of his large head. In utero there are some disturbances of cartilaginous centers1 especially of the epiphyses of the long bones, causing a retardation in long-bone growth later in life. By x-ray examination at birth, the spinal column of the achondroplastic is normal in length and curvature. However, the entire spinal canal is narrower than normal because of the shortness of the pedicles. As these dwarfs continue to subject their spinal column to the stresses and strains of everyday life, many will show changes in spinal curvature.
RUPTURED DISCS IN ACHONDROPLASTIC DWARFS

Fairbank pointed out that in achondroplastic dwarfs the spines are extremely lordotic, but often the dorsolumbar spines are surprisingly flat. Sometimes the apparent lordosis results from the unusual prominence of the buttock.

Donath and Vogl described more or less marked changes in the physiologic lordosis of these dwarfs at the dorsolumbar region. From the 12th dorsal to the 3rd lumbar segments the vertebral bodies show a definite wedging of varying degree. Thus the achondroplastic dwarf has an inherent weakness of the spinal column manifested by dorsolumbar kyphosis. Whereas some show flat backs, others reveal distinct gibbous formation at that level.

There are earlier cases in the literature of achondroplastic dwarfs with neurologic symptoms and signs suggestive of cord compression. However, Albrecht and Ranzi, in 1926, reported a case of compressive myelitis upon which they performed a laminectomy. Not only was the diagnosis confirmed but the achondroplastic dwarf regained many lost functions. Donath and Vogl felt that, in their case, arthritic changes above the level of the gibbus caused the formation of osteophytic spurs which encroached upon the spinal cord. Freund, then Kennedy et al., subsequently demonstrated similar findings in case studies. Vogl and Osborne, in 1949, reported another example and developed further the pathogenesis of varying degrees of transverse myelitis in achondroplastic dwarfs as being caused by exostoses encroaching upon a constricted spinal canal. They showed that the entire spinal canal is narrower than a normal adult canal. Since the achondroplastic dwarf’s spinal cord is of normal diameter, the cord just fits into the canal with no room to spare. Hypertrophic arthritic changes appear adjacent to and above the gibbous deformity because of the increased motion compensating for the lack of motion at the level of the gibbus.

REPORT OF CASES

Case 1. W. B., a 33-year-old white male auto worker, was well until mid December of 1950 when he noticed pain in both hips and his “feet gave way” while lifting materials and suddenly turning his body. He was taken to a hospital where he was put on bed rest. His hip pain diminished at rest but became very severe with radiation down both legs when he was on his feet. He also noticed numbness, tingling and weakness of his legs. He was admitted to Harper Hospital Jan. 8, 1951, on the orthopedic service.

Examination. The patient was an achondroplastic dwarf. He was in no acute distress. Positive findings were: an absent left knee jerk, an absent right ankle jerk and hypalgesia of the right little toe and lateral aspect of the right thigh. Both legs were weak.

Course. Because he was thought to have a mechanically unstable back, his spine was manipulated under sodium pentothal and a plaster body cast was applied. The patient did well until he was on his feet and then his pain and paresis recurred. He was referred to the neurosurgical service.

A lumbar puncture was done on Jan. 23, 1951, with injection of pantopaque. A few drops of yellowish CSF were obtained; total protein was 33 mg. per cent. The myelograms revealed a complete block at the level of the intervertebral disc between L2 and L3. The pantopaque did not flow up or down with the patient on the tilt table. This was thought to be consistent with an arachnoiditis.

Operation. A laminectomy was done with removal of the lower four lumbar spines and laminae. The spinal canal throughout was much shal-