Charcot Joint of the Lumbar Spine

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Historically, the relationship between spinal cord lesions and certain joint diseases is well recognized, having been pointed out by Mitchell in 1831. Later, neurogenic arthropathies were observed in hemiplegia by Scott and Allison in 1843, in syringomyelia by Blasius in 1848, in acute myelitis by Magnier in 1859, and in relation to lesions of peripheral nerves by Packard in 1861 (described by Delano). Following Charcot's description in 1868 of joint changes in tabetics, neurogenic arthropathies became known as "Charcot joints." Jordan in 1936 first described the condition in association with diabetes mellitus.

The first reported case of spinal neuroarthropathy is attributed to the German physician Kronig in 1884 (described by Holland). Zucker and Marder in 1952 described the first reported case of Charcot spine occurring with diabetes mellitus.

Etiology

Eloesser in experiments with cats in 1917 contributed important information on the etiology of Charcot joints. In one group of animals, he divided a number of posterior roots supplying a limb and found that some of the animals developed Charcot joints in the affected limb. In a second group of cats, he injured joints and then divided posterior roots to the involved extremity. He found that the cutting of the posterior roots had no effect on the existent joint deformity. In a third group of animals, he divided posterior roots and then injured the joint; within 3 weeks every cat thus treated developed a Charcot joint. He concluded "this, then, was not a slow aggravation of a pre-existing deforming arthritis; it was a sudden response of an anesthetic joint to the acute trauma of operation, a rapid reaction to bone injury by the production of a typical Charcot joint."

Foster and Bassett stated that the following conditions may contribute to the formation of Charcot joint: 1) major trauma or repeated small traumas associated with continued movement of a diseased limb; 2) intact motor power to the affected joint; 3) impairment of pain impulses; 4) impairment of proprioceptive impulses which normally inhibit hypermobility of joints; 5) chronicity of the underlying nervous disorder; and 6) metabolic disturbances.

In several large series of Charcot joints, the condition occurred most commonly in tabes dorsalis, with syringomyelia being the second most common cause. Diabetes mellitus and other neurologic disorders are infrequent etiologic conditions.

Pathology

The pathological changes typical of Charcot joints have been described by several authors. The process is believed to be essentially the same in all involved joints. Early in the process, joint cartilage is lost in places and may become covered by a vascular connective tissue. In the deeper layers of the joint, there is an abnormal proliferation of cartilage cells. Destruction of cartilage is accompanied by destruction of underlying bone, which becomes irregular in structure. The spaces between the lamellae are filled with connective tissue. Resumption of enchondral ossification occurs, and is often so great and so diverse that by simultaneous absorption and proliferation an enormous incongruity of the joint surfaces may result. Either the atrophic or hypertrophic changes may predominate.

In the vertebral column, there is initially a narrowing of the intervertebral disc space and a relaxation of ligaments. Degenerative changes lead to erosions and osteoporosis of the vertebrae. Proliferative changes result in sclerosis of the vertebral bodies, as well as bony growth and osteophytes. Tiny
fractures occur, and detached bony fragments are found. Late in the process bony proliferation springs from the articular margins and tends to strengthen the disorganized joint by producing some degree of ankylosis. Compression of the vertebral body develops at times with displacement posteriorly and laterally. Usually, the process is localized in one to three vertebrae, often with a sharp kyphosis and lateral rotation.

The gross changes described above are evident radiographically, although early signs of fragmentation and fine debris formation are usually less evident in radiographs of the spine than overlying soft tissues and gas shadows.7,21

**Clinical Data**

Most patients are in the fifth and sixth decades of life. The time interval in tabes dorsalis from the syphilitic infection and the development of the arthropathy varies from 5 to 40 years. Among tabetics, approximately 4% to 10% develop Charcot joints, most often in the lower extremities. The knee is the joint most often involved, with hip and feet (tarsal joints and phalanges) being next in frequency. Spine involvement comprises from 6% to 21% of Charcot joints depending on the joints quoted.5,6,20 Ankle, shoulder, hands, elbows, and other joints are less often involved.

Several authors have described the general features of Charcot joint of the spine.7,10,11,13,14,21,22 The site of predilection is the lumbar spine, but cases have been reported involving the thoracic spine,13,14 the cervical spine5,7,10 and the sacrum.13 The great majority of cases occur in tabetic patients. Only a minority of patients have associated neurogenic arthropathies of other joints.

Usually the signs and symptoms characteristic of tabes dorsalis are well developed. A kyphosis or scoliosis of the lumbar or thoracolumbar spine is often present. The irregularity of the spine is sometimes palpable. There is no local tenderness and the spinal movements are excessively free, but the affected segment of the spine may remain rigid due to ankylosis.

The differential diagnosis7,13,14,21 includes osteoarthritis, suppurative osteomyelitis, spinal tuberculosis, thyphoid and Brucella spondylitis, Paget's disease and other bone maladies, and old traumatic conditions.

The symptoms and signs of nerve root compression occur in a minority of cases. Involvement of posterior spinal roots with resultant sensory disturbances is mentioned by several authors, including Campbell and Doyle.9 Pressure upon nerve roots due to collapsing spine or hypertrophic bone formation may lead to considerable discomfort. These root pains are usually gradual and insidious in onset and of moderate severity. Occasionally, however, they attain considerable intensity and may simulate gastric crises of peptic ulcer pain.

Allergan in 1960 claimed to be the first author to report the occurrence of Charcot spine as a cause of motor symptoms and signs due to pressure upon anterior nerve roots. He reported two patients with tabes dorsalis, both of whom developed bilateral foot drop followed by marked weakness in both legs. In both cases radiographs showed marked destructive changes affecting the lumbosacral vertebrae of a type characteristic of tabetic arthropathy. Storey70 reported a further case of paralysis of the legs from lumbosacral root involvement.

**Case Reports**

We have observed five cases of Charcot joint of the lumbar spine.

*Case 1.* A 61-year-old white man was hospitalized in 1961 with the diagnosis of atonic neurogenic bladder and urinary tract infection. The patient gave a history of tabes dorsalis diagnosed in 1941 and treated with several courses of penicillin. A full neurologic examination was not done; however, the patient was noted to have a “tabetic gait,” a positive Romberg sign, and absent deep tendon reflexes in the legs. The blood serology (VDRL and Kolmer) tests were positive. A lumbar puncture was not done. Charcot joints of L4-5 and L5-S1 were noted on pyelography, and films available from 1956 and 1961 showed progressive bony changes over the 5-year period (Fig. 1 left). The spine lesion was not treated.

*Case 2.* A 58-year-old white man was hospitalized in 1963 for evaluation of weight