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Case Report Open Access

Charcot Spine after Remote Cervical Spine Injury

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Abstract

Charcot spine, also known as neuropathic spinal arthropathy and Charcot spinal arthropathy, is a rare and potentially devastating consequence of spinal cord injury. Treatment of this condition ranges from observation, immobilization with bracing or jacket, or surgical stabilization. A 52-year-old male, with a history of a C6-7 spinal cord injury over 30 years ago, presented to our hospital with complaints of worsening of his chronic low back pain over the prior 6 months. His pain was exacerbated with movement, and he also found it more difficult to sit up straight in his wheelchair. MR imaging of the spine demonstrated the large fluid collection with transection of the thecal sac and spinal cord at T10-11, along with the prior known spinal cord injury at C6-7. Given the patient's clinical presentation and signs of progressive degenerative changes with worsening kyphosis, the patient underwent a T7-L3 posterior instrumented fusion for stabilization. Through the same incision, an extracavitary approach was utilized for placement of an expandable titanium case for anterior column support. After the operation, he experienced a significant reduction in pain and noted improved posture in his wheelchair.

Keywords: Charcot spine; Spinal neuropathic arthropathy; Spinal cord injury; Paraplegia

Introduction

Charcot spine, also known as neuropathic spinal arthropathy and Charcot spinal arthropathy, is a rare and potentially devastating consequence of spinal cord injury. The degeneration seen in patients with Charcot spine occurs secondary to the loss of innervation to the spinal facet joints [1]. The most common cause is traumatic spinal cord injury, though syphilitic tabes dorsalis frequently seen in the past [2]. Patients often complain of increased kyphosis, a "cracking" sound in their back during movement, increasing pain, or increased spasticity. Treatment of this condition ranges from observation, immobilization with bracing or jacket, or surgical stabilization. In this report, we describe a patient diagnosed with Charcot spine 32 years after a complete cervical spinal cord injury, and treatment with a posterior approach for stabilization.

Case Report

A 52-year-old male presented to our hospital with complaints of worsening of his chronic low back pain over the prior 6 months. His pain was exacerbated with movement, he heard a "grinding" noise in his back, and he also found it more difficult maintain an upright posture in his wheelchair. He denied fevers, chills, or change in motor or sensory function. His past medical history was significant for a fall from a large height 32 years prior, which resulted in a complete C6-7 spinal cord injury and paraplegia.

The patient's physical exam was remarkable for a C7 sensory level, weakness in hand intrinsic muscles, and no movement in the lower extremities. Inspection of the patient's back revealed prominent midline spinous processes in the lower thoracic region without any evidence of edema or erythema. On admission, the patient's white blood cell count was normal; however, both his sedimentation rate and C-reactive protein were elevated at 44 and 3.94, respectively. A CT scan of the thoracic spine revealed erosion of the T10 and T11 vertebral bodies, distraction and retrolithesis of T10 on T11, a large heterogeneous fluid collection extending into the paravertebral soft tissues, and hypertrophic osteophytosis (Figure 1A and 1B). Also of note on the CT scan are bridging anterior osteophytes. This ankylosis is also likely the result of many years of paraplegia, lack of innervation to the vertebral joints, and long-standing microtrauma. MR imaging of the spine demonstrated the large fluid collection with transection of the thecal sac and spinal cord at T10-11, along with the prior known spinal cord injury at C6-7 (Figure 2).

Given the patient's clinical presentation and signs of progressive

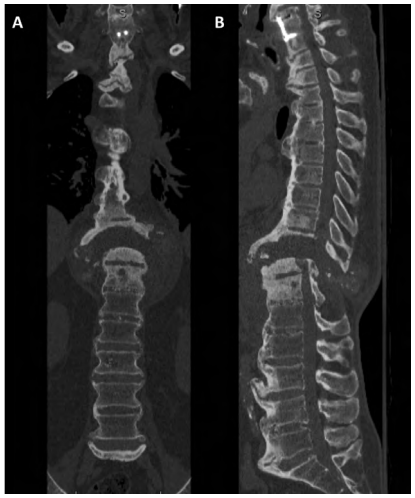

Figure 1A and 1B: Coronal and sagittal CT scan without contrast revealed erosion of the T10 and T11 vertebral bodies, distraction and retrolithesis of T10 on T11, a large heterogeneous fluid collection extending into the paravertebral soft tissues, and hypertrophic osteophytosis.

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degenerative changes with worsening kyphosis, the patient underwent a T7-L3 posterior instrumented fusion for stabilization. Through the same incision, an extracavitary approach was utilized for placement of an expandable titanium case for anterior column support (Figure 3). The pathologic level was obvious, with gross instability and large associated fluid collection. Intraoperatively, it was discovered that he had a transected thecal sac with induration and scarring of the proximal and distal ends of the dura, such that there was no CSF leak. The patient had no complications and was discharged to rehab on postoperative day 4. After the operation, he experienced a significant reduction in pain and noted improved posture in his wheelchair.

Discussion

Charcot spine is rarely seen, but remains an important pathology to recognize amongst patients who experience spinal cord injury. Impairment of joint innervation with loss of proprioception leads to excessive stress and distraction on the joints. This repetitive trauma results in damage to the cartilage, chronic synovial effusion, and ultimately, destruction and subluxation of the joint [3]. Often, hypertrophic osteophytosis also occurs in the surrounding soft tissue. The thoracolumbar and lumbosacral levels are affected the most, due to the high mechanical stress in these areas, and in most cases, only one level is involved [4]. For this reason, amongst paraplegic patients, those who remain active appear to constitute a high-risk demographic, due to the forces exerted on the spine induced by sitting in a wheelchair and during transfers.

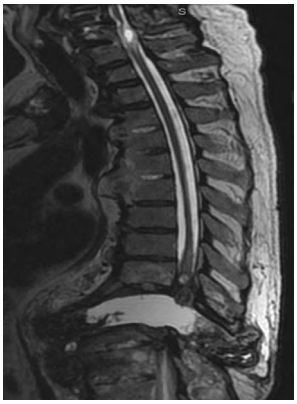

Figure 2: MR imaging of the spine demonstrated the large fluid collection with transection of the thecal sac and spinal cord at T10-11. Also note the prior spinal cord injury at C6-7.

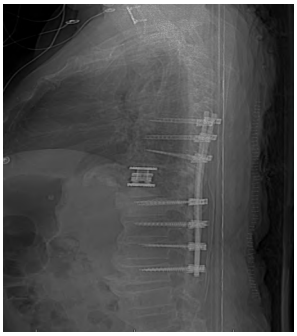

Figure 3: Post-operative x-ray demonstrating a T7-L3 posterior instrumented fusion and expandable titanium case for anterior column support.

As previously mentioned, traumatic spinal cord injury is the leading cause of Charcot spine. However, infections or neoplastic pathologies can lead to the condition, as can syringomyelia. Typically, patients who develop Charcot spine present decades after the initial injury, with reports ranging from as few as 11 to as many as 77 years after the initial trauma [4].

Frequently, an increase in pain, spinal deformity, instability when sitting, and an audible "cracking" sound is described by patients. In patients with complete spinal cord injuries, an increase in spasticity may also be seen. For patients with some preserved neurologic function, a worsening in motor or sensory status can also occur [5].

Radiographic characteristics of the condition consist of marked disc and vertebral body destruction, fluid collections and hypertrophic osteophytosis, instability, and deformity [6]. Therefore, diagnostic imaging tests, including dynamic flexion/extension x-rays, CT scan, and MRI, are all important in the evaluation of the Charcot spine patient. Dynamic x-rays are utilized to determine the severity of instability and movement of the spine around the affected level. CT scans illustrate the bone quality, pedicle sizes, degree of hypertrophic bone formation and are helpful in surgical planning. The spinal cord, thecal sac, and surrounding soft tissues are better evaluated by MRI. Also, MRI is can be helpful in differentiating Charcot spine from alternate diagnoses, such as infection and tumor.

There are no Charcot spine specific criteria to confirm the diagnosis. Instead, the history, imaging, physical exam finding, and patient's symptoms must be assimilated into a Charcot spine diagnosis. The main differential diagnoses of Charcot spine are infectious and neoplastic processes, as these conditions share common findings of bony erosion, osteophytosis, and paravertebral mass formation. Post laminectomy instability, and pseudoarthrosis of an ankylosed spine are two other diagnoses that must be considered. Clinical history (recurrent infections, subsequent traumatic event, etc.), as well as a needle biopsy, can be very helpful delineating one diagnosis from another.

Treatment ranges from conservative observation to immobilization with a brace or jacket, or surgical stabilization. Immobilization may slow or stabilize the progression of the disease, but should be considered in patients unwilling to undergo, or unfit for surgical intervention. As mentioned, many patients, especially those treated conservatively, undergo a biopsy to rule out an infectious or neoplastic process, which may mimic some of the findings seen in Charcot spine [4]. Surgical treatment, with the goal of spinal stabilization, is commonly recommended due to the progressive and destructive nature of the disease [7]. Ideally, all three spinal columns are augmented and supported. Typically surgeons utilize a posterior approach with pedicle screws, and a cage is inserted, if possible, to support the anterior spine column [8]. Bone morphogenetic protein and additional rods have also been used to increase the rate of fusion [9]. Anterior only, or combined anterior and posterior have been performed in the past.

Charcot spine, although rare, can be a painful and serious complication of spinal cord injury. Therefore, providers must be cognizant of the entity, and spinal cord injury patients must have regular, long-term clinical and radiographical follow-up, even years after the injury. Patients should be evaluated yearly for 5 years after the traumatic event, and every 3 years subsequently, or more frequently if symptoms progress or a neurologic deficit observed.

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