Case Report

Early Progressive Charcot Spinal Arthropathy after Non-Traumatic Spinal Cord Injury

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Abstract

Neuropathic Charcot spinal arthropathy (CSA) is a rare and destructive process that develops in the absence of deep sensation and that affects the intervertebral discs and adjacent vertebral bodies. Initially and for many cases, with a long time asymptomatic evolution, diagnosis is usually made at an advance stage of bone destroying lesions with spine dislocation. The differential diagnosis of CSA includes infection (bacterial, fungal and tuberculosis) tumor, aseptic spondylodiscitis in spondyloarthropathies and Paget disease. Disc puncture can be done to obtain cultures and reject disc space infection. In our opinion, surgical treatment for spine stabilization by circumferential fusion is indicated in patients with instability or failure of conservative treatment that could be probed in non-aggressive cases. We describe a quickly progressive case of CSA in a non-traumatic SCI patient and make a review of literature.

ABBREVIATIONS

SCI: Spinal Cord Injury; CSA: Charcot Spinal Arthropathy; ICU: Intensive Care Unit

INTRODUCTION

Neuropathic Charcot spinal arthropathy (CSA) is a rare and destructive process that develops in the absence of deep sensation and that affects the intervertebral discs and adjacent vertebral bodies [1,2]. This condition results from the loss of protective sensation and joint protective mechanism secondary to any condition affecting the deep sensory pathways [2]. Initially reported by Kronig in 1884 in a patient with tabes dorsalis, today is rare with the disappearance of tertiary syphilis and now is found in patients after traumatic spinal cord injury (SCI) [2,3]. Initially and for many cases, with a long time asymptomatic evolution, diagnosis is usually made at an advance stage of bone destroying lesions with spine dislocation [1-3]. We describe a quickly progressive case of CSA in a non-traumatic SCI patient and make a review of literature (Figure 1).

CASE REPORT

A 50-year-old man was admitted to his district hospital because of respiratory failure and altered level of consciousness. His trachea was intubated, connected to mechanical ventilation and admitted to the ICU. Tracheostomy was performed. Two weeks later, paraplegia was detected. Spine X-Ray and

Figure 1 X-ray lateral view of thoracolumbar spine at admission in our Hospital Previous spondyloarthropathy and laminectomy. Vertebral body destruction affecting posterior arch and spine dislocation.
MRI revealed T12 vertebral body destruction, a big epidural haematoma from T5 to L4 with cord compression and changes in spinal cord intensity from T10 to L3. We can not show that images because there were performed in his primary Hospital. A T10-L1 laminectomy was performed to drainage the haematoma, and samples were taken for culture and biopsy. No germs or malignant cells were found on tissue samples.

The patient was a lifelong smoker, consumed more than 80 g of alcohol per day and ten years before had been diagnosed of dilated cardiomyopathy with moderate left ventricular dysfunction. He also suffered from chronic atrial fibrillation, type II diabetes, sleep apnea syndrome and morbid obesity. Medications included amiloride + hydroclorothiazide, digoxin, acenocoumarol, bisoprolol and omeprazole (Figure 2).

Three weeks after symptoms started he was moved at the ICU of our hospital, the Spanish National Hospital for SCI patients (Hospital Nacional de Parapléjicos, Toledo) at admission in our Hospital he presented an SCI ASIA A grade, located at T11 level. He had several respiratory and systemic complications (atelectasis, pleural effusion with chest drainage, tracheobronchitis, catheter-related bacteriemia, adrenal failure and one episode of acute delirium). Differential diagnosis was made between infection, tumor, aseptic spondylodiscitis in spondyloartropathies and Charcot disease. Due to spinal instability, surgery was indicated. Circumferential fusion from T9-L2 with pedicle screws and rods and a titanium mesh cage was performed (Figure 3). Tissue samples were sent to the histopathology and microbiology laboratories (bone, bone marrow and fibrocartilaginous tissue with fibrosis and haemorrhage, without bacteria or malignant cells) CSA was diagnosed [4,5].

After surgery he was discharged to the intermediate respiratory unit, where weaning from the respirator was achieved. Clinical examination revealed SCI level T11 ASIA A. His further hospital course was unremarkable and he was discharged nine months later, on finishing his rehabilitation program. He has been followed every six months at the outpatient department; since his discharge four years ago. Vertebral fusion was achieved and no loss of correction was observed. His neurological status is stable, without modification of his SCI level.

DISCUSSION

Pathophysiological mechanism of CSA is unknown (1,2) the excessive biomechanical loads transmitted to the thoracolumbar spine though aggressive transfer activity has been proposed as a cause as most of the lesions appear in the lumbar spine, where increased lumbar mobility may predispose to excessive forces during self-transfer activity [6] but most of young patients with SCI do this kind of activity, including sport, without having been reported a higher incidence of CSA, for example, in paralimpic athletes. The differential diagnosis of CSA includes infection (bacterial, fungal and tuberculosis) tumor, aseptic spondylodiscitis in spondyloartropathies and Paget disease [1,2].

Main symptoms are deformity, loss of sitting tolerance, an audible click, progressive pain and changes in neurological status [7]. The occurrence of autonomic dysreflexia while moving, that is aggravated when sitting instead of alleviated, should prompt the investigation of an underlying cause in the lower spine [6]. It seems to be triggered by spinal instability and affect approximately 7% of patients with CSA [7]. Radiological signs suggestive of CSA over infection are discovertebral destruction involving the facet joint, disc vacuum phenomenon, osseous joint debris, spondylolisthesis and joint disorganization [8]. Simple X-ray examination of the spine can give as the diagnosis MRI and TC should be use to confirm it and see the extension. We usually use disc puncture can to obtain cultures and reject disc infection.
Most of the contemporary series of CSA have defined an almost exclusive, proportion of cases directly related to traumatic SCI, ASIA. A grade [6] occurs only caudal to the level of the injury [1,2,6] but, as in our case, we can find quickly progressive CSA in patients with recent non-traumatic SCI. Latrogenic instability after decompressive laminectomies and/or transfer of nonphysiological loads to segments adjacent to previously fused regions are also factors that may contribute to the development of CSA [6] and that could be the cause in our patient.

Because the low incidence of CSA it has been difficult to establish a consensus as to its optimal treatment. For some authors, surgery is necessary for all CSA cases to stabilize the “broken spine”, prevent death, neurologic complication, kyphotic anquilosis or infection [2,4-6]. For others, conservative treatment is the reference [3]. Non-unions, infections and relapses are the most commonly reported complications with a reoperation rates as high as 40% in old cases [3]. So, nowadays, if surgery is necessary, circumferential fusion is recommended for most of the authors with the use of a four rods construction in same patients to avoid this high rate of failure [2,6]. The goal of surgery is to obtain a solid fusion of the spine and prevent deformity [4].

CONCLUSION

Suspicion and early detection of CSA by symptoms is essential to make a diagnosis and avoid progressive spine deterioration. Simple X-ray examination of the spine can give as the diagnosis. MRI and TC should be used to confirm it and see the extension. Disc puncture can be done to obtain cultures and reject disc space infection. In our opinion, surgical treatment for spine stabilization by circumferential fusion is indicated in patients with instability or failure of conservative treatment that could be probed in non-aggressive cases.

REFERENCES