Eur Spine J (2007) 16 (Suppl 3):S318–S321
DOI 10.1007/s00586-007-0343-9

CASE REPORT

Shortening osteotomy for the treatment of spinal neuroarthropathy following spinal cord injury. A case report and literature review

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Received: 27 November 2006 / Revised: 18 February 2007 / Accepted: 22 February 2007 / Published online: 22 March 2007

Abstract The development of Charcot's arthropathy of the spine secondary to spinal cord injury is rare and reports in the literature concerning it's surgical management are limited. Arthrodesis is the recommended treatment for painful and unstable neuropathic joints. Traditionally this involves extensive debridement of the affected joint with anterior and posterior instrumented fusion and autograft to bridge the defect. This paper reviews the reported surgical management of post-traumatic spinal neuroarthropathy in the recent literature and presents a case where sound fusion was achieved by a shortening osteotomy and end-to-end apposition of the fresh bleeding bony surfaces of the adjacent vertebral bodies. The patient reported marked improvement of symptoms post-operatively without any complications of surgery. CT scan at 13 months confirmed successful bony union. Clinical follow up was completed to 3 years. This technique eliminated the need for anterior surgery and extensive autograft thus reducing surgical morbidity.

Keywords Charcot joints · Spinal fusion · Shortening osteotomy · Spinal cord injury

Introduction

Neuropathic (Charcot) spinal arthropathy was initially described in association with tabes dorsalis. Other causes have included syringomyelia, leprosy, cord tumours, spinal tuberculosis, cerebrovascular accidents, diabetes and alcoholic neuropathy [3, 13, 16, 21]. Park et al. [15] in a review of English literature found only 29 reported cases of spinal neuroarthropathy occurring after spinal cord injury.

The exaggerated degenerative process seen in Charcot arthropathy occurs when the protective sensory mechanisms of the intervertebral and apophyseal joints (pain and ligamentous stretch reflexes) are lost leading to progressive subluxation and even frank dislocation. As the afferent proprioceptive fibres are destroyed, repeated trauma ensues during patient transfer without proper host inhibition. Charcot arthropathy of the spine most commonly affects the lumbar vertebrae due to the increased weight bearing of the spine at this region and the absence of rib support. Two pathological subtypes of neuroarthropathy have been described: (1) atrophic neuroarthropathy characterised by massive bone resorption and (2) hypertrophic neuroarthropathy, which demonstrates extensive bone formation and osteophytosis. The differential diagnosis in early Charcot arthropathy includes severe osteoarthritis, osteomyelitis, Paget's disease and destructive tumours. The characteristic ''exploded'' appearance of all three columns demonstrated on CT scanning in the later stages of the disease is rarely seen in other pathologies and helps to establish the diagnosis [2, 5, 17]. However definitive tissue histology and culture from the involved joint is still mandatory to exclude infection or malignant change.

The most common presenting complaint is ill defined, burning pain around the level of the involved vertebrae followed by an increase in kyphotic deformity leading to a loss of seating balance. Patients may also report a ''grinding'' or ''clunking'' sensation in their backs when transferring. Above the major splanchnic outflow level of T6, autonomic dysreflexia has also been reported in

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relation to spinal Charcot arthropathy secondary to spinal cord injury [14].

The recommended surgical treatment for painful and unstable neuroarthropathy of the spine is arthrodesis. Surgery described has included posterior instrumented fusion with anterior strut grafting and combined anterior and posterior instrumentation with extensive debridement and autologous grafting.

Case report

We report a surgical technique where sound fusion of the neuropathic pseudoarthrosis was achieved by a shortening osteotomy and end-to-end apposition of the adjacent vertebral bodies by a single-stage direct posterior approach eliminating the need for extensive autograft to bridge a large bony resection.

A 38-year-old male, who at the age of 17 (in 1985) had a motorbike accident, was left with a complete spastic paraplegia below T5 after sustaining a fracture dislocation of the dorsal spine. The initial treatment at the time of injury had been a T2–T9 fusion with Harrington Rod fixation. He rehabilitated well becoming independent of self-care including wheelchair transfer. He returned to full time employment as a clerical officer and became a keen fisherman in his spare time.

Eighteen years later, in April 2002 he presented with a dull aching pain in the dorsolumbar area and described a sense of clinking and crunching in his back on movement. He also described a change in neural status with sudden loss of his leg spasticity together with a loss of erectile function and the development of flaccid areflexia. A CT scan showed a cleft within the body of T10 and a characteristic vacuum sign [11] (Fig. 1).

With the patient under observation, a year later his symptoms worsened and examination revealed gross postural instability in the dorsolumbar spine. He had no history of fever or weight-loss. Radiographs at that stage showed complete posterior dislocation of T9 on T10 in addition to new bone formation indicating the development of a Charcot joint (Fig. 2).

Operative technique

Through a posterior approach, the previous Harrington Rods were removed and a tissue sample was taken from the neuropathic segment and sent for culture and histological assessment. Exploration of the affected level showed complete destruction of the posterior elements leading to a pseudoarthrosis with extensive fibrosis and a bursal cavity filled with clear fluid. Complete resection of the sclerotic avascular bone was achieved by removal of the T10 vertebrae as well as the adjoining endplates of T9 and T11. With the aid of pedicle fixation three levels above and below the resection, the dislocation was reduced and the spine shortened to achieve direct bone-to-bone contact between the remaining T9 and T11 vertebrae. Allograft bone graft was laid over the decorticated posterior elements for an end-to-end combined anterior and posterior fusion (Fig. 3).

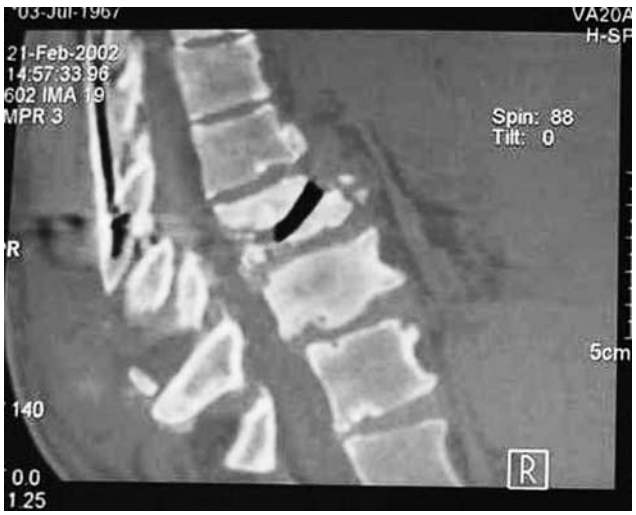

Fig. 1 CT at presentation showing cleft sign

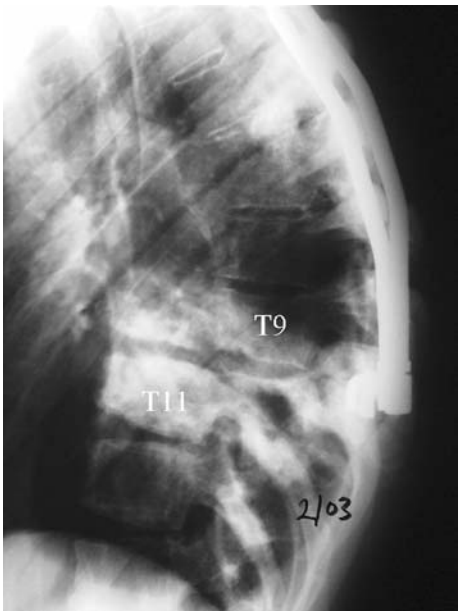

Fig. 2 Lateral radiograph one year after presentation showing the T9–T10 dislocation

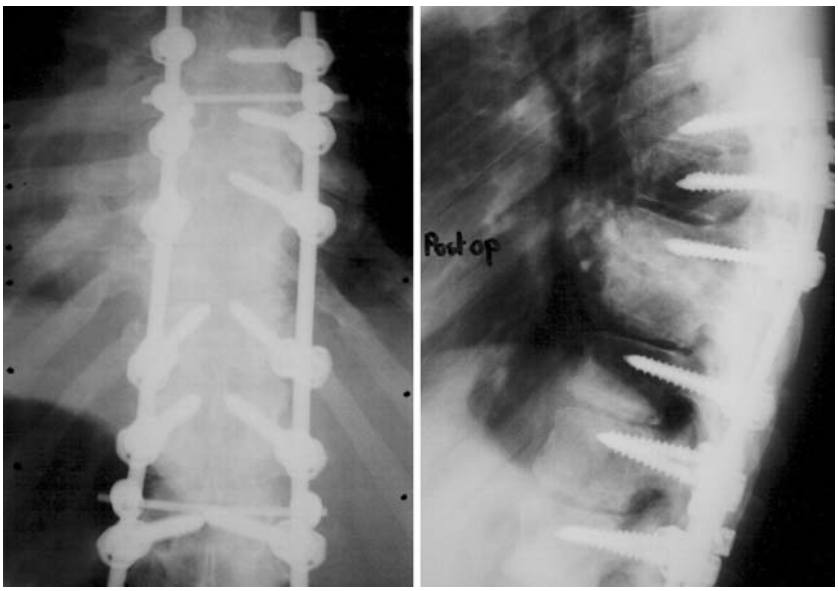

Fig. 3 Post-operative AP and lateral radiographs showing reduction, spinal shortening and bone-to-bone contact

Outcome

The patient was allowed to mobilize in his wheelchair by the second week after surgery and a TLSO was used for 5 months. After surgery, the patient reported marked improvement of symptoms and felt more secure while sitting in his wheelchair. Clinically he had a straight spine with no tenderness. Radiographs and CT scans at 13 months demonstrated a satisfactory arthrodesis between the bodies of T9 and T11 (Fig. 4). The patient has remained symptom free during the 3 years since his operation.

Discussion

Although uncommon, the possible development of neuropathic spinal arthropathy should be considered as a late complication in a long-term paraplegic patient after spinal cord injury. Treatment of a symptomatic Charcot spine should be directed towards vertebral stabilization to avoid complications secondary to instability. The first reported instrumentation for Charcot spine was in 1978 when Slabaugh et al. [18] described a technique where the pseudoarthrosis at T11 to L1, was resected and with the aid of Knodt compression rods and iliac crest bone graft, an end-to-end fusion was achieved shortening the spine by two vertebral levels. The patient was kept recumbent in a cast for 6 months whilst the anterior and posterior fusion consolidated. Devlin et al. [7] published a series of eight patients treated surgically for post-traumatic neuropathic spine. six of the eight underwent combined anterior and posterior fusion and instrumentation, five with vascularized transfers (rib-4, fibula-1). Two patients were treated with single-stage posterior procedures including decompression and grafting of the anterior bony defect. Post-operatively the patients were

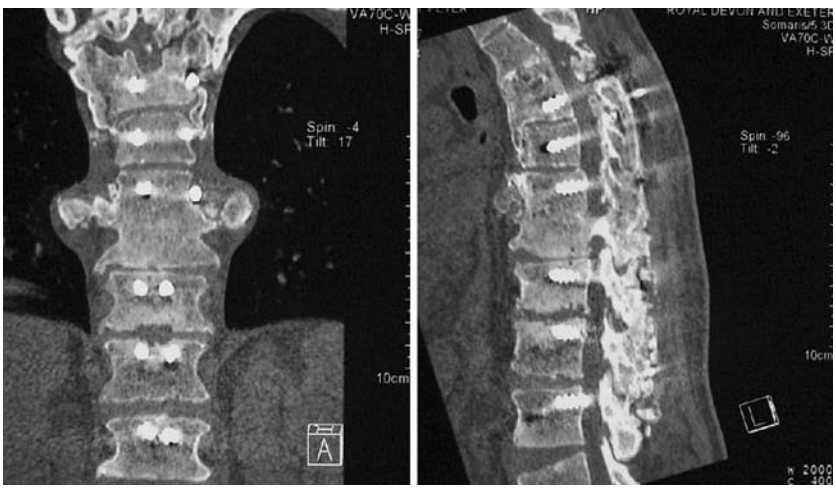

Fig. 4 CT scan showing bony union between bodies of T9 and T11 at 13 months follow up

mobilized without the use of orthoses. Complications occurred in five patients including three infections (two requiring further surgery) and three instrumentation related problems (two requiring further surgery). At final follow up one patient had a persistant pseudoarthrosis. The largest series on the treatment of neuropathic arthropathy of the spine after traumatic paraplegia was published in 1992. Brown et al [4] described 15 cases, 8 of which were treated surgically. All had both anterior and posterior fusions, six had posterior instrumentation alone with an autogenous anterior strut graft and two had combined anterior and posterior fusion with autogenous grafting. Complications reported included two failures of instrumentation fixation and two superficial wound infections. During the follow up period (averaged 3.1 years) it was noted that three patients developed a second level of charcot arthropathy distal to their previous fusion. Arnold et al. [1] described a further two cases, one treated with posterolateral decompression with tibial interbody graft and posterior instrumentation, the other underwent lateral extracavitary decompression with anterior interbody rib graft and posterior instrumentation. No complications were reported within the short follow-up period. More recently, Vialle et al. [20] published a series of nine cases of Charcot spine, five post-traumatic and all treated with circumferential fusion. Four cases where fused with combined anterior and posterior approach, one by posterior approach only. In one-patient instrumentation failure was responsible for delayed anterior fusion leading to repeat surgery including posterior instrumentation and posterolateral fusion.

Previously described methods of surgical treatment involve extensive debridement of the pseudoarthrosis (including vertebrectomy) with bridging of the often-massive defect with autologous bone graft followed by anterior and posterior fusion. This is high risk, demanding surgery involving significant morbidity to an already compromised patient and requires prolonged rehabilitation. We report a technique where sound fusion was achieved by a single-stage shortening osteotomy and end-to-end apposition of the adjacent vertebral bodies without anterior surgery. In our case, the overlapping and the bayonet position of the dislocated spine allowed safe shortening of the spine without threat to the surrounding soft tissues and major vessels. Load sharing was achieved anteriorly by direct compressive bone-to bone contact once the avascular, sclerotic bone of the pseudoarthrosis was resected. The operation was performed entirely through a direct posterior approach and eliminated the need for autologous bone graft harvesting.

It is acknowledged that any fusion procedure adds abnormal stresses to adjacent levels and so theoretically could potentiate the development of further distal Charcot joints in the neuropathic spine. This is especially pertinent in the young, active paraplegic patients working within the community who are involved in self-transfer activities and sport. Long-term clinical and radiological monitoring is required to determine the prevalence of further Charcot joints developing in the remaining unfused spinal motion segments.

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