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Original Article

Neuropathic lumbar spondylolisthesis – a rare trigger for posture induced autonomic dysreflexia

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Study design: Case report.
Objectives: Description of a rare trigger for autonomic dysreflexia.
Setting: Princess Royal Spinal Injuries Unit, Sheffield.
Methods and Results: A case of Charcot's spine (neuropathic spinal arthropathy) in a woman with a traumatic T5 paraplegia is described. She developed symptoms of autonomic dysreflexia, brought on by changes in posture. The postural variation was attributable to a freely mobile neuropathic spondylolisthesis at the L4/5 level. A laminectomy performed for the implantation of a sacral anterior root stimulator was identified as a causative factor in the development of the neuropathic joint. Surgical stabilisation and fusion resulted in amelioration of her symptoms.
Conclusion: Neuropathic spine is a rare cause of autonomic dysreflexia that should be considered when other more common factors have been excluded. The development of Charcot's spine in the spinal cord injured population is facilitated by surgical procedures involving the vertebrae.
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Keywords: Charcot's spine; neuropathic spondylolisthesis; autonomic dysreflexia; postural variation; spinal cord injury; spinal fusion

Introduction

Since its original description by Bowley in 1890, autonomic hyperreflexia has been known under various names. Signs and symptoms of autonomic hyperreflexia are varied and include paroxysmal hypertension, bradycardia, headaches, diaphoresis, urinary retention, piloerection and sweating.[1] Less common complications have included myocardial infarction, seizures, intracerebral bleeds and visual deficits.[2]

It is believed that stimulation of pelvic or presacral nerves is one of the trigger mechanisms for this syndrome in patients with suprasacral spinal cord injury. A variety of stimuli induce dysreflexic symptoms, though the commonest is probably bladder distension.[3,4] The impulse from the stimulus travels to the spinal cord and then in a cranial direction in the lateral spinal thalamic tracts and dorsal columns. This precipitates a massive uninhibited sympathetic response through the intermediolateral cell column. In neurologically intact subjects this response is moderated by central inhibition. The unopposed sympathetic activity induces severe hypertension, which is perceived by carotid sinus and aortic body baroreceptors, which in turn stimulate the vasomotor centre and the brain stem. The resulting vagal response leads to the well-described picture of vasodilatation above the spinal cord lesion and vasoconstriction below the level of lesion.[3] However more recent work has failed to demonstrate a consistent increase in plasma noradrenaline levels. The increased responsiveness to pressors seen in spinal injured patients may be due to increased reactivity of resistance vessels or decreased neuronal reuptake from the synaptic cleft.[5]

Charcot's spine, first described by Kronig in 1884 in patients with tabes dorsalis has since been reported by various authors as occurring in traumatic paraplegia.[6–8] There is a predilection for Charcot changes to occur in the lumbar spine thought to be related to the absence of rib support and increased weight bearing function of this area of the spine. The underlying reason for the development of neuropathic joint is related to the absence of pain and joint sense due to neurological disease.[9] Neuropathic arthropathy is merely exaggerated joint disease that occurs in the absence of protective sensation in the joint and its synovium.[10] The denervated joint undergoes a mech-

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anical process of disintegration with changes in the articular and subchondral bone that lead to incongruity and articular fracture. An extensive laminectomy with removal of the osseous and ligamentous constraints of the posterior vertebral elements may initiate or potentiate the neuropathological process that leads to Charcot's arthropathy, as a consequence of increased instability of the affected joints.[7]

Autonomic dysreflexic symptoms precipitated in a paraplegic female with grossly unstable neuropathic spondylolisthesis that followed a previous spinal surgical procedure is described.

Case report

Mrs D is a 60-year-old woman who suffered a complete T5 paraplegia in 1990 when she was thrown from her horse in a riding accident. Her initial spinal injury consisted of a fracture of the fifth thoracic vertebra. She underwent Harrington instrumentation and fusion of her vertebral column injury. The postoperative period was uneventful and she was mobilised in 6 weeks following the injury. There was no relevant past medical history.

She learned to manage her bladder by intermittent self-catheterisations but was troubled by frequent leakage. Video urodynamic studies demonstrated a hyper-reflexic bladder. Perivesical injections of phenol carried out twice in 1991 and 1992 did not improve her incontinence. In view of the continuing problem with her bladder, a sacral anterior root stimulator (SARS) was implanted in 1993. She developed CSF leakage post operatively, but this resolved in a few days. Following the implantation, she developed good control over her bowel function but the desired control over her bladder function was not achieved.

During her initial rehabilitation she had troublesome spasms which were controlled with baclofen and dantrolene. In 1994 the spasms deteriorated and caused a significant functional handicap. Increasing doses of oral antispasmodics did not achieve satisfactory control of spasms. Later that year an intrathecal drug delivery system was implanted, with initial good control of her spasm. Radiographs taken at the time showed a grade 2 L4/5 spondylolisthesis, which was asymptomatic (Figure 1).

In 1998 following a repair of her rotator cuff, she developed an MRSA wound infection, which required prolonged use of teicoplanin and vancomycin. Towards the end of the same year she repeatedly attended the unit with problems of poor posture, altered bowel and bladder habits, frequent leakages and a general feeling of being 'washed out'. Extensive haematological, radiological and urological investigations failed to identify the underlying cause of her symptoms. MRI of her thoracic and cervical spine did not show any evidence of cavitatory changes or myelomalacia.

Intermittent failure of the baclofen pump was considered as one of the causes of her symptoms.

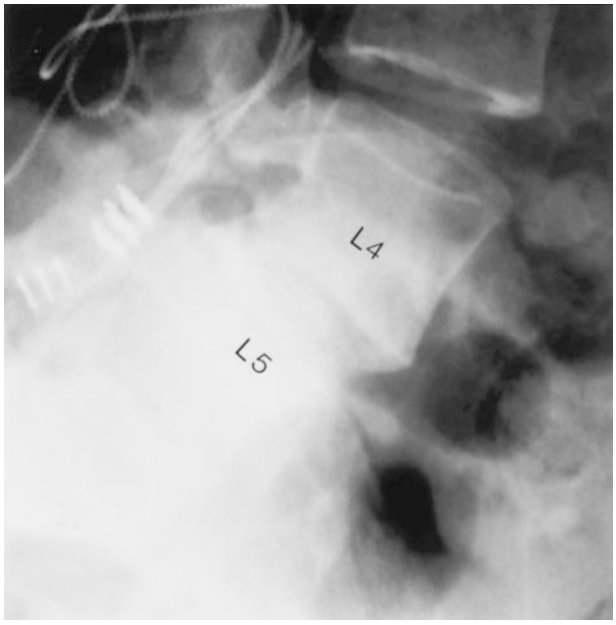

Figure 1 Lumbar spine showing grade II spondylolisthesis (1994)

The entire intrathecal delivery system was replaced in July 1998. She continued to experience symptoms of lethargy. Urinary continence could not be achieved by intermittent catheterisation because of her inability to transfer. A suprapubic catheter was inserted for bladder management.

In December 1998 she reported a grating and clunking sensation from her back when she sat up and moved around. Radiographs taken the same month showed a grade 3–4 spondylolisthesis at L4/5 level with neuropathic changes. The orthopaedic team were loath to intervene for fear of disturbing her SARS, which she continued to use for control of her bowels.

Three months later she began to describe dysreflexic symptoms including sweating, restlessness, flushing and hypersensitivity of skin which were brought on when she sat up, but subsided when she lay down. Examination showed that her blood pressure rose from 110/78 supine to 200/140 when the bed was tilted to 45 degrees. In the sitting up posture it dropped a little to 190/110, but when her dysreflexic symptoms were present, the blood pressure had risen to 260/120, but gradually declined to 120/70 when she was put back to bed. Radiographs in supine and sitting positions showed a freely mobile neuropathic spondylolisthesis at L4/5 which dislocated and reduced in the sitting and supine postures respectively (Figure 2).

She then underwent a posterior stabilisation and fusion from L3 to S1. The symptoms completely resolved in the post operative period. The spasms also eased considerably and she remains very satisfied with the procedure.

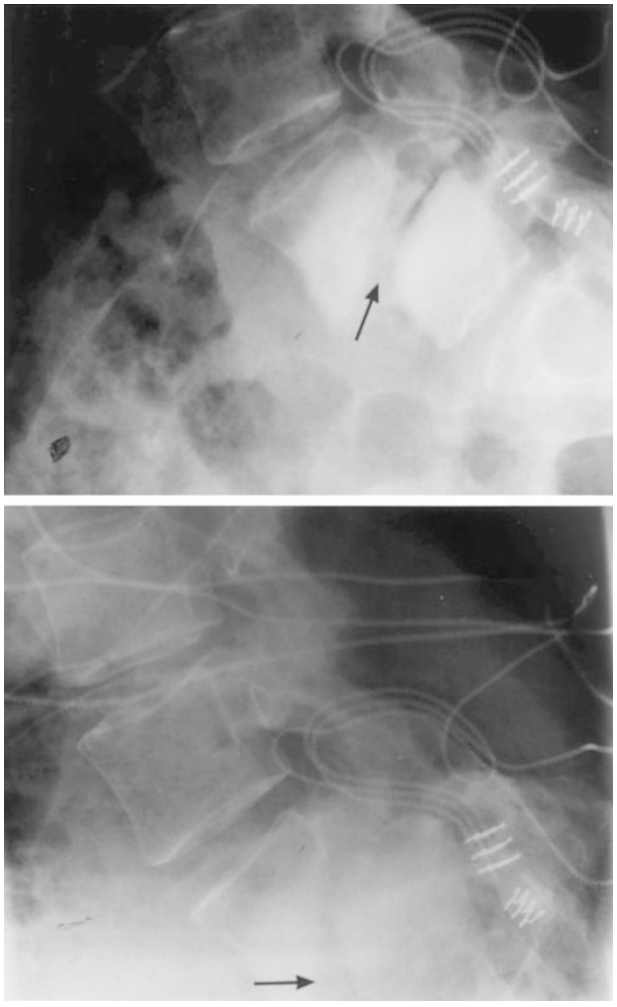

Figure 2 Top – Lumbar spine X-ray when patient was in supine position. Bottom – X-ray when the patient was sat up, showing significant forward movement of L4 on L5

Discussion

Charcot's spine, though rare, is not infrequently described in traumatic paraplegia. The relative increase in the reported cases of Charcot's spine in paraplegia may be a reflection of greater physician awareness and improved survival of spinal injured patients.[8] Common presenting symptoms have included back pain, change in spasticity, change in bladder function, changes in neurology and audible noises with motion.[7,8] Radiologically, initial hypertrophic bone formation is followed by destruction of end plate, massive periosteal bone formation and the formation of a giant pseudoarthrosis.[7,9,10]

Over a period of time, the patient we described above went through all these symptoms. Although it was recognised that she had grade 2 spondylolisthesis in 1995, its relevance to her symptoms were not fully appreciated. There was a reluctance to intervene surgically for fear of interfering with the sacral anterior root electrodes. The laminectomy employed while implanting SARS electrodes may have contributed to the development and exacerbation of the neuropathic joint. The role of surgical intervention on the spine as a contributory factor in the development and exacerbation of neuropathic joints is known.[6–9] A neuropathic joint causing autonomic dysreflexic symptoms has not been described before. In this patient the symptoms and blood pressure changes showed a direct relationship to posture. We suggest that in the sitting posture the joint dislocated forwards exerting pressure on the presacral plexus of nerves and the retroperitoneal viscera. A series of noxious stimuli, due to the dislocation of L4 on L5, triggered a massive sympathetic response resulting in her symptoms. As soon as she lay down the compression on her viscera and the stretch on the presacral nerves reduced, ameliorating the symptoms. Further evidence in support of the above hypothesis is the fact that symptoms ceased soon after stabilisation of her lumbar spine. Once frank dysreflexic symptoms were recognised with changes in blood pressure, stabilisation was carried out very quickly to prevent the development of severe, even life threatening complications. The choice of surgery on this particular occasion was influenced by coexisting hip contractures and the desire to do the minimum required. After extensive discussions between the orthopaedic team and the spinal team involved in her long-term care, a posterior instrumentation and fusion was carried out from L3 to S1, although an anterior procedure would have been the ideal.

In view of the serious consequences, physicians and health professionals should consider neuropathic spine as a rare cause of autonomic dysreflexia when other more common factors have been excluded.

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