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Non-traumatic acute paraplegia caused by cervical disc herniation in a patient with sleep apnoea

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ABSTRACT

A 57-year-old man with obstructive sleep apnoea presented with acute progression of myelopathy into paraplegia resulting from cervical disc herniation at C4-C5 and C5-C6 levels. There was no associated history of trauma. Rapid progress to paraplegia from non-traumatic cervical disc herniation rarely occurs. Diagnostic and treatment modalities are discussed. The possible relationship between cervical myelopathy and sleep apnoea is also discussed.

Keywords: myelopathy, non-traumatic paraplegia, sleep apnoea, intervertebral disc herniation

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INTRODUCTION

Non-traumatic acute myelopathy leading to paraplegia resulting from cervical disc herniation is rare. To date, there have been only five reported cases[1,2]. Though patients with established cervical myelopathy may have respiratory complications including sleep apnoea[3-6], there is no previous report of sleep apnoea as a presentation of cervical disc herniation. We report a rare case of a patient with a history of sleep apnoea who developed rapidly-progressive myelopathy leading to paraplegia secondary to cervical disc herniation.

CASE REPORT

A 57-year-old Chinese man presented to our emergency department with neck pain for two days, associated with worsening numbness over the right side of his body and his left upper limb. There was no history of trauma and he had no past history of cervical spine problem. He also has diabetes mellitus, managed on dietary control. He was recently diagnosed to have obstructive sleep apnoea (OSA) about six months prior to the current presentation. He weighed 88kg then and was assessed by the otorhinolaryngologist to have multiple levels of narrowing in his upper airway. He had been receiving nasal continuous positive airway pressure therapy for the problem of OSA, and was responding well to this treatment.

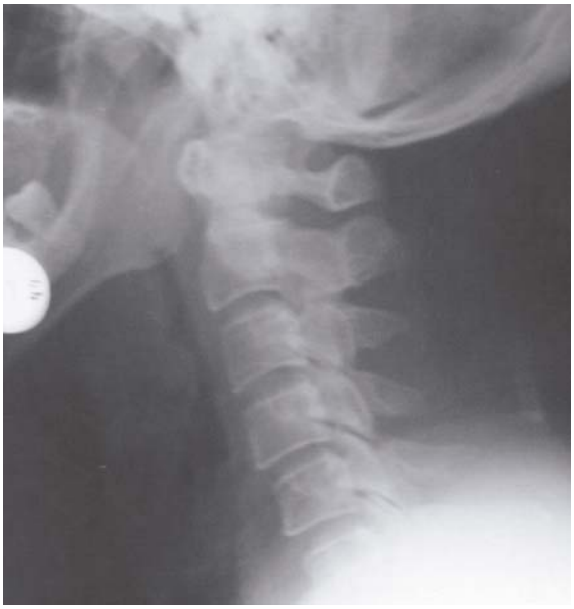

Fig. 1 Pre-operative lateral cervical radiograph shows osteophytes at multiple levels.

At presentation, the patient was still able to move all four limbs and lower limbs power was graded to be five. Deep tendon reflexes of the upper limbs were normal but he was hyperreflexic in the lower limbs. Plantar reflexes were downgoing. A cervical spine radiograph showed osteophytes at multiple levels, and a diagnosis of cervical spondylosis was made (Fig. 1). He was admitted in view of persistent numbness and increasing neck pain. When reviewed two hours later, he had developed bilateral lower limb weakness, and examination revealed the absence of power. Power of the distal part of upper limbs were graded to be zero but was still present over C5 myotome bilaterally, graded three over right side and four over left side.

Emergency magnetic resonance (MR) imaging showed a large extruded disc at C4-C5 level, causing severe spinal stenosis and compression of the spinal cord. There was also a sizeable disc protrusion at C5-C6 level. Increased signal of the spinal cord was also seen (Fig. 2).

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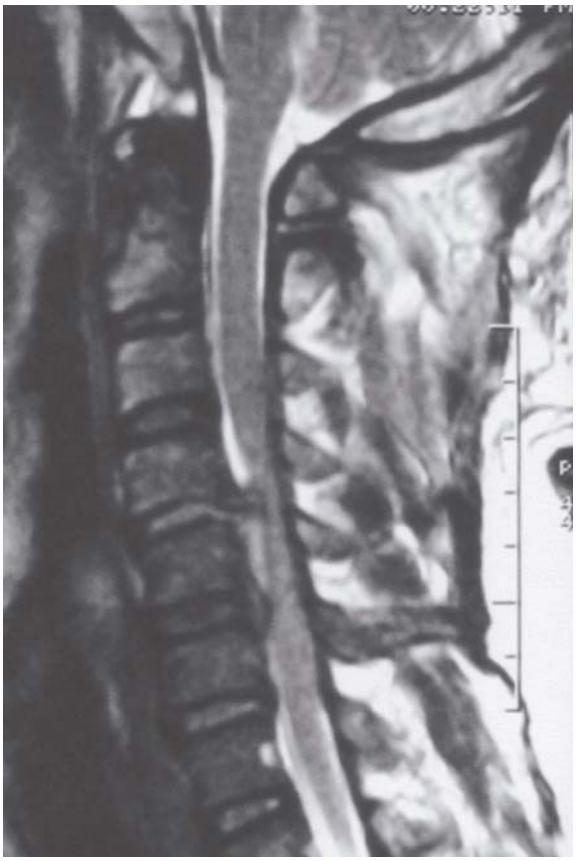

Fig. 2a Sagittal T2-W MR image shows the C4-C5 and C5-C6 discs protruding posteriorly. There is increased signal intensity in the spinal cord from C4 to C6 levels.

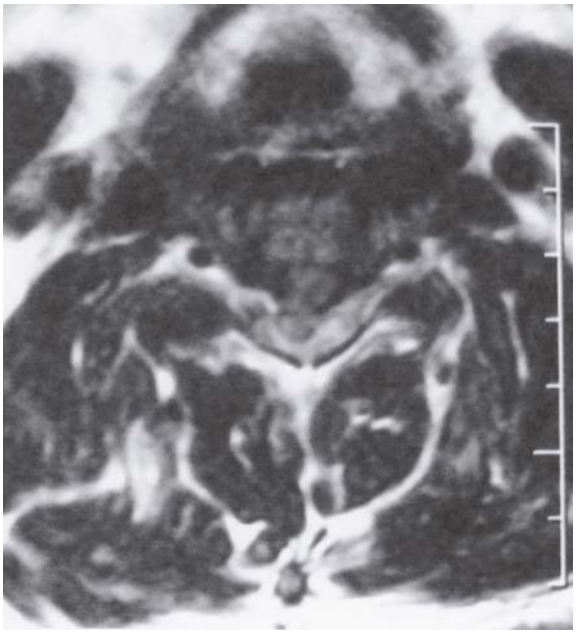

Fig. 2b Axial T2-W MR image shows that the cervical cord is compressed at C4-C5 disc level.

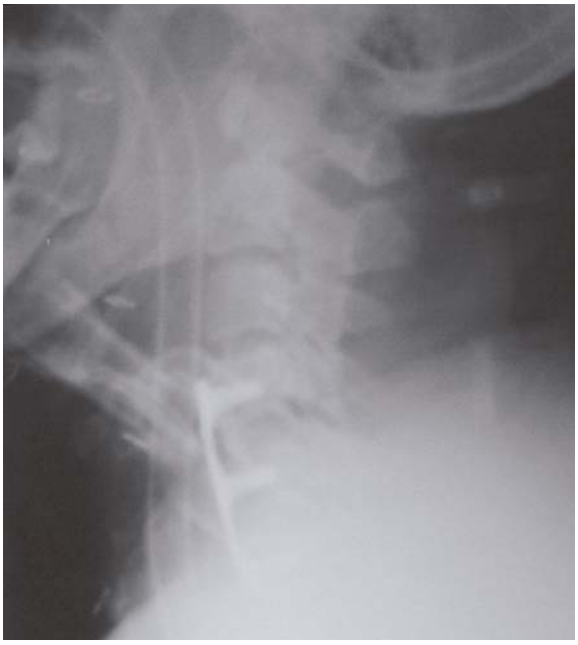

Fig. 3 Post-operative lateral cervical radiograph shows C4-6 plating.

A decompression operation was subsequently performed. An anterior approach was taken and C4-C5 and C5-C6 discectomies were done. The cervical spine was stabilised with two tricortical iliac crest bone grafts and placement of a cervical plate (Fig. 3).

Post-operatively, the neurological recovery was unfortunately dismal. Patient was able to move the shoulder as power at C5 myotome was preserved but there was no significant recovery distally, even after six months. His recovery was also complicated by several recurrent episodes of pneumonia, resulting in recurrent respiratory failure and need for ventilatory support.

The lack of intercostals muscle power and recurrent pneumonia resulted in the need for a permanent tracheostomy and persistent oxygen supplement therapy. Patient also continued to have episodes of sleep apnoea despite the insertion of the tracheostomy. The patient is currently still receiving intensive chest physiotherapy and incentive spirometry, while being weaned off his oxygen support.

DISCUSSION

Cervical disc herniation resulting in non-traumatic acute myelopathy is rare. There have only been five reported cases of non-traumatic acute paraplegia resulting from cervical disc herniation[1]. Cervical cord compression occurs more often in Asian men because this group has a constitutionally narrower spinal canal and a higher incidence of ossification of the posterior longitudinal ligament which predisposes to disc prolapse[7,8].

This case illustrated the rapid progression from initial presentation to complete paraplegia. Suzuki

et al[1] had previously warned about the possibility of this rapid progression of myelopathy secondary to non-traumatic cervical motion associated with physical examination or changing position during surgery. This should certainly be kept in mind in the management of patients with myelopathic symptoms. The disruption of the blood supply via the anterior spinal artery and its branches add to the local compressive effect of the prolapsed disc, and may be the cause of the rapid progression in such cases.

MR imaging showed a large prolapsed cervical disc which was compressing the spinal cord. Indeed prolapse of the cervical disc may not be seen on radiography, and so this investigation, as our case showed, cannot be relied on to exclude prolapsed intervertebral disc disease[9]. The increased MR imaging signal of the spinal cord in our case is similar to findings in previous reports[1,2]. This indicates the presence of cord oedema and gliosis, and is consistent with histological findings of ischaemia-reperfusion injured spinal cord seen in experimental animals[10]. This suggests that early diagnosis with timely decompression for the spinal cord may be crucial in preventing severe secondary spinal cord injury.

Fehlings et al reviewed the current evidence pertaining to the role and timing of surgical decompression[11]. Based on a meta-analysis of the literature, they found that there is strong biological rationale, based on animal models, to support the concept that early decompression improves outcome after acute spinal cord injury. Unfortunately, this data has not been reproduced in human trials and the relevant time frame for surgical decompression in humans remains unclear. In the same review, the authors noted that studies supporting the role of surgical decompression in humans are limited largely to non-randomised prospective studies, case series and retrospective studies; and accordingly, surgery can be considered only a practice option. They noted the need to conduct well-designed and executed randomised controlled prospective trials on spinal cord injury to fully evaluate the role and timing of decompression surgery[11].

Sleep apnoea is the periodic reduction or cessation of breathing during sleep. It can be a result of a reversible or permanent narrowing of the upper airway, and it will then be termed obstructive sleep apnoea. There are past studies that explore association of sleep apnoea as a cause of hypertension, ischaemic heart disease, stroke and road traffic accident[12], but none linking it as a cause of either cervical disc herniation or cervical myelopathy.

However, conversely, cervical spinal cord problems including trauma[3], surgery[4], infarction[5] and tumour[6] have been reported to result in respiratory dysfunction, including sleep apnoea. Howard et al reported four patients with infarction of the anterior portion of the cervical cord complicated by respiratory insufficiency, including sleep apnoea[5]. There is a descending respiratory pathway described that subserved autonomic and voluntary ventilatory control[13]. It is postulated that the lesions in the cervical cord may disturb these descending pathways, leading to apnoea during sleep and inattention[5].

In our case, patient's sleep apnoea problem may be contributed by his cervical myelopathy. Although our patient had a high body mass index which was associated with OSA and was assessed to have multi-level upper airway obstruction, he continued to have episodes of sleep apnoea, even after a tracheostomy. The persistence of sleep apnoea despite relief of any upper airway obstruction following the tracheostomy suggested a contributing central neurological cause. We postulate that the cervical myelopathy from the herniated cervical disc compressing against his spinal cord provided the neurological cause to his sleep apnoea. This process may have started before his neurological symptoms of weakness. As in other reported cases[3,5], the occurrence of apnoea in such patients resulted in problem in weaning patients off ventilatory support and this should be anticipated.

In conclusion, rapid progress to paraplegia from cervical disc herniation, though rare, does occur. Early diagnosis with decompression surgery to avoid severe injury to the spinal cord is critical. Sleep apnoea may therefore be persistent in a patient with cervical myelopathy.

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