

Unrecognised spinal cord compression as a cause of morbidity

SB O'Neill, JP McCann

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SUMMARY

Predicting outcomes is important in planning patient management and rehabilitation. Two cases, one with illustrative radiology, are described. Each presented with potentially preventable morbidity, secondary to unrecognised compression of the spinal cord. Detailed history and examination may have revealed the underlying problem: a condition with potential associated long-term morbidity.

INTRODUCTION

In this paper, we describe two cases in which there was a delay in reaching the diagnosis of spinal cord compression, leading to increased morbidity in each case. We would suggest that, in each case, a full history and thorough clinical examination would have prompted investigation of the underlying problem, thereby resulting in lessened morbidity. We also suggest that where response to treatment including rehabilitation is not as expected, a clinical explanation should be sought.

CASE REPORTS

Case 1 A 61-year-old lady presented with a fracture at the right ankle following a fall. She reported feeling that her leg had given way. Five months later she presented, following a further fall, with an intracranial haemorrhage. Her initial Glasgow Coma Scale (GCS) was 12/15. CT (computerised tomography) scanning revealed a left parietal extradural haematoma which was subsequently evacuated. Post-operatively, she made a good functional recovery; however mild weakness of her right leg persisted, despite full recovery of function in her right arm, which at outpatient review prompted further investigation.

A detailed history revealed a four-year history of reduced balance and falls, two of which resulted in the aforementioned problems. Detailed neurological examination revealed a sensory level at T6 and bilaterally extensor plantar reflexes. An MRI (Magnetic Resonance Imaging) of her



Fig 1. Sagittal MRI of spine revealing a well-defined rounded intradural tumour mass, characteristic of meningioma at the level of the 3rd and 4th thoracic vertebrae.

Royal Victoria Hospital, Grosvenor Road, Belfast
BT12 6BA

Department of Rehabilitation Medicine:

SB O'Neill, MB, MRCP, Specialist Registrar in
Rehabilitation Medicine

JP McCann, MD, FRCP, FRCPI, Consultant in
Rehabilitation Medicine

Correspondence to Dr O'Neill

E-mail: soneill@doctors.org.uk

spine revealed a tumour characteristic of a meningioma at the level of the 4th thoracic vertebra (Fig. 1). Some 13 months following her initial injurious fall she underwent thoracic laminectomy and excision of a T4 level intradural extramedullary tumour. Histology confirmed a meningioma. Following an initially stormy postoperative course she has recovered power in her legs and her overall mobility and balance have improved.

Case 2 A 34-year-old man with avascular necrosis of the left femoral head underwent left total hip replacement (THR). The avascular necrosis had occurred secondary to steroid treatment for Hodgkin's lymphoma which had been diagnosed ten years earlier. Subsequent to his assessment for THR, and two months before his surgery he was treated with ABVD (adriamycin, bleomycin, vincristine and dacarbazine) chemotherapy and steroids for acute spinal cord compression at the level of the 5th and 6th thoracic vertebrae (secondary to his Hodgkin's lymphoma). At the time of surgery there was no apparent neurological deficit, with only his painful hip limiting his mobility.

He experienced recurrent THR dislocation and a posterior lip augmentation device (PLAD) was implanted as a preventative measure. Weakness of the hip and knee flexors of Medical Research Council (MRC) grade two and three muscle power respectively was noted. Bowel and bladder function was normal. He was subsequently transferred for inpatient rehabilitation. A history of a dose dependent effect of dexamethasone on his strength was noted, with an increase in hip flexor power and the patient reported increasing back pain.

MRI of the spine at this time, eight weeks post-THR, revealed marked kyphosis and compression of the spinal cord to 3mm diameter by collapse due to lymphomatous infiltrate of the 5th and 6th thoracic vertebrae (Fig. 2). He underwent anterior and posterior decompression and corpectomy at this level with posterior stabilisation.

Following this he had a further period of inpatient rehabilitation. Pre and post spinal surgery lower limb American Spinal Injury Association motor scores¹ were 34 and 39, out of a maximum 50, respectively. He achieved an increase in his mobility and functional skills, which he retained until his death two years later due to Hodgkin's lymphoma.



Fig 2. Sagittal T2 weighted MRI of thoracic spine shows spinal cord compression and vertebral collapse at the level of the 6th Thoracic vertebra.

DISCUSSION

Simple trip and fall is a common presentation, however few will have spinal cord disease. Patients with mild limb weakness secondary to spinal cord compression may present in a similar manner and if a detailed history and examination is not performed, potentially reversible causes of spinal cord pathology may be missed. Moreover the patient will be susceptible to the potential injurious sequelae and morbidity of their spinal cord disease. Spinal cord compression, in the form of cervical spondylotic myelopathy, has been described as a cause of gait disturbance and falls in the elderly.²

In case one, diagnosis of the spinal cord compression at the time of initial presentation to hospital would have, in all probability, prevented the later fall and consequent head injury and extradural haematoma. Her failure to progress in rehabilitation, in retrospect seen to be secondary to her underlying thoracic meningioma, might have prompted further investigation at an earlier stage.

In case two the recurrent dislocation of the THR was in all probability due to weakness of the pelvic musculature secondary to spinal cord dysfunction. We would suggest that his mobility was reduced by the presence of spinal cord compression in addition to left hip joint pain. Spinal cord compression has been variably noted in up to 5% of patients with Hodgkin's lymphoma. There are no specific figures for long term survival following spinal cord compression.

Osteotomy is often considered in cases of avascular necrosis,³ given that THR is expected to require revision in 10 to 20 years. Our patient was offered THR, as his life expectancy was short. However, it is probably the case that, with clinical evidence of underlying spinal cord dysfunction, the use of PLAD would have been considered earlier; thus reducing the risk of the prosthetic hip dislocating⁴ and preventing the subsequent increase in his morbidity. There is little in the way of published evidence on the interrelationship between myelopathy and the outcome of joint replacement, although in patients with rheumatoid arthritis it has been demonstrated that subsequent development of cervical myelopathy is a limitation to good outcome following multiple major lower limb joint replacement.⁵ Missed compressive lesions, causing paraplegia, at the thoracic vertebral level have been reported following surgery for lumbar spinal canal stenosis.⁶

Prediction of rehabilitation outcomes is important in planning patient care, and depends upon an accurate diagnosis of the cause of a patient's impairments. These cases also demonstrate the need to review reasons for failure to achieve the expected outcome and where appropriate undertake investigation to seek concomitant pathology.

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