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Delayed focal neurological deficit secondary to a cervico-thoracic spinal cord epidural haematoma

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Abstract This is a report of an elderly woman who developed focal neurological deficit in association with a cervico-thoracic spinal epidural haematoma. Symptoms developed several days after the initial injury and subsequently resolved without surgical intervention. The unusual features of this presentation are discussed.

Key words Focal neurological deficit · Spinal epidural haematoma

Introduction

The incidence of post-traumatic spinal epidural haematoma has been estimated at about 1–2% [2]. The condition typically presents with neurological deterioration soon after injury. Surgical decompression is usually advocated in an attempt to reverse neurological deficit, although recovery has been reported without surgery [1].

This report describes an elderly woman who developed a delayed localised motor deficit in association with a post-traumatic cervico-thoracic epidural haematoma which resolved without surgical intervention.

Case report

An 85-year-old woman was involved in a road traffic accident, in which the car she was driving rolled over. She sustained a head injury but did not lose consciousness. On admission she was found to have an injury to her lower cervical spine without neurological deficit. A CT scan of her neck revealed fractures through the spinous process of C6 and through the laminae of C7 and T1. An epidural haematoma was also noted.

Three days following this injury she developed a significant motor deficit in her right leg. Examination revealed a flicker of movement (1/5) in her quadriceps and in tibialis anterior. Sensation in the right leg was normal and there was no neurological abnormality of the left leg. Furthermore, there was no neurological deficit of either upper limb and cranial nerve function was intact.

She did, however, develop reduced bladder control. It was initially felt that her symptoms were more consistent with an intracerebral event and she underwent a CT scan of her head. This was normal.

She subsequently underwent an MRI scan of her neck (Fig. 1), which confirmed the extent of the epidural haematoma and also showed evidence of signal change within the cord opposite the C7/T1 disc. The patient and her family were counselled regarding management options, and it was decided to take an expectant approach because of her rather unusual features and the incomplete

Fig. 1 The extent of the epidural haematoma is shown by the MRI scan, which also reveals signal change within the cervico-thoracic spinal cord

nature of her loss. Three days following the onset of motor loss, some evidence of improvement was noted. Three weeks following the onset of weakness, her symptoms had almost completely resolved.

At follow-up 6 months later, she was fully mobile with normal bladder function.

Discussion

Interpretation of this patient's neurological symptoms was influenced by the focal nature of the deficit. An epidural haematoma of this size and in this location might be expected to produce a rather more widespread neurological deficit. We therefore suspected that this elderly woman had sustained a cerebrovascular accident. However, the CT scan of her head, which was performed 24 h after onset of her symptoms, did not show any evidence of either an infarction or an intracranial haemorrhage. The high density of an intracranial haemorrhage usually appears at once, while the characteristic low-density changes of an infarction may become apparent within hours of the onset of symptoms, and usually by 24 h or so [3]. We were therefore confident that her symptoms were not due to an intracranial event.

The subsequent MRI not only confirmed the extent of the epidural haematoma, but also showed signal change within the central part of the spinal cord at the site of maximum compression from the epidural haematoma (Fig. 1). Although we cannot exclude any pre-existing change at this site, this signal change may represent either an area of haemorrhage or acute oedema, which would explain both the focal nature of the neurological deficit and the subsequent recovery.

The patient might be considered fortunate that her cervical spinal canal was not otherwise narrowed, which would have placed her at greater risk of developing a central cord syndrome at the time of her injury. Instead, it would appear that a more gradual onset of spinal cord compression developed as a result of an expanding epidural haematoma, and that this resulted in the development of a focal, transient neurological deficit.

The long-term outcome in this case has justified our initial non-operative approach. We would emphasise, however, that our usual management of patients who have major neurological impairment with spinal cord compression due to an epidural haematoma is urgent surgical decompression.

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