

## SHORT REPORT

# Intrinsic spinal cord lesions complicating epidural anaesthesia and analgesia: report of three cases

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Serious neurological complications related to epidural anaesthesia and analgesia are only rarely reported. We describe the clinical and radiological features of three patients who sustained intrinsic spinal cord lesions after attempted epidural catheterisation. In each case there was an early onset of motor and sensory impairment after the procedure and MRI demonstrated similar, extensive, paracentral, high signal intensity lesions within the cord on T2 weighted images. Possible mechanisms to explain these MRI appearances are discussed. It is proposed that the most likely cause of these lesions was direct trauma to the spinal cord during the procedure and subsequent injection of fluid into the spinal cord producing localised hydromyelia. The prognosis in each case was for a gradual recovery of motor function but spinothalamic sensory impairment and severe spontaneous pain over the affected area persisted.

Epidural anaesthesia and analgesia are used widely during surgical procedures and for pain control. They are generally regarded as safe and effective,<sup>1</sup> although complications may occur during placement of the epidural catheter or from the effects of the drugs given during the procedure.<sup>2</sup> Despite the potential risks, the frequency of severe, permanent neurological complications related to epidural catheterisation, based on prospective and retrospective studies, seems to be extremely low at roughly 0.1–1/10 000 procedures.<sup>3–8</sup> Radiculopathy, cauda equina syndrome, and myelopathy are all recognised causes of permanent neurological disability after epidural anaesthesia and analgesia.<sup>9</sup> Some underlying mechanisms have been implicated in the aetiology of these syndromes. Compression of the spinal cord or nerve roots by extradural abscess or haematoma formation, arterial and venous infarction of the spinal cord, nerve root trauma during catheter placement, and chemically induced arachnoiditis have all been implicated in causing permanent neurological disability after epidural procedures.<sup>10–14</sup>

Lumbar epidural injection may also precipitate a severe and widespread lumbosacral polyradiculopathy in the presence of pre-existing spinal stenosis.<sup>9, 15</sup> Complications resulting from direct trauma to the spinal cord during attempted epidural catheterisation have only rarely been reported.<sup>16–18</sup> We describe the clinical and radiological findings of three cases of intrinsic spinal cord damage occurring as a result of cervical, thoracic, and lumbar epidurals.

## CASE REPORTS

### Patient 1

A 46 year old man presented to the orthopaedic team with pain in his neck radiating to the left shoulder. A plain radiograph disclosed osteoarthritis of the left acromioclavicular joint and after failure of conservative treatment with analgesics the lateral end of the left clavicle was excised. After sur-

gery the pain persisted and 7 months later a cervical epidural at C6–7 was attempted for pain relief. It proved technically difficult to insert the epidural catheter and therefore a solution of 10 ml 0.25% bupivacaine and 80 mg methylprednisolone acetate in 10 ml normal saline was injected directly through the epidural needle. Shortly after the procedure he developed weakness and numbness of the right arm and hand. This was followed by severe neurogenic pain affecting the whole of the arm, neck, and upper chest on that side. On subsequent examination mild weakness of the right upper limb was noted with absent reflexes and diminished pain and temperature sensation between C3–T4 on the right. Vibration and proprioception were preserved. Magnetic resonance imaging of the cervical and thoracic spine performed 6 days after the procedure disclosed an extensive area of high signal intensity on T2 weighted images within the right paracentral region of the cord extending from C2 to T1 (fig 1). Over the next few weeks power gradually returned to normal although reflexes remained absent in the right arm and there was persistent alteration in pain and temperature sensation from C3–T4 on the right. Paraesthesia was replaced by severe spontaneous pain, which was burning in quality and still troublesome 18 months later.

### Patient 2

A 69 year old man underwent a right hemicolectomy for carcinoma of the caecum under general anaesthesia. Postoperatively, while the patient was still anaesthetised, a thoracic epidural was performed at T7–T8 with 10 ml 0.1% bupivacaine and 50 µg fentanyl using the loss of resistance to air technique. Soon after the injection there was a brief episode of hypotension when his blood pressure dropped from 120/70 to 70/30. This lasted about 10 minutes. On waking from the general anaesthetic he complained of severe pain and weakness in both arms and upper trunk. A cautious injection of a further 1 ml 0.25% bupivacaine was administered through the epidural catheter resulting in an exacerbation of the pain. The catheter was therefore removed. Subsequent neurological examination showed severe weakness of both upper limbs, worse on the right. There was diminished sensation between C3 and T10 on the right and between T2 and T6 on the left. All upper limb reflexes were absent or diminished and plantar responses were bilaterally extensor. The next day MRI showed a diffuse central cord lesion extending from C2 to T1, which was hyperintense on T2 weighted images (fig 1 B) and hypointense on T1. Signal changes were more extensive and conspicuous on T2 weighted images suggesting peripheral oedema as well as central cavitation. He was treated with high dose intravenous methylprednisolone given as a bolus of 30 mg/kg followed by an infusion of 5.4 mg/kg over 23 hours. Over several weeks power gradually improved in the upper limbs but he continued to experience an unpleasant burning sensation in his right arm and upper trunk. A repeat MRI 3 months later showed resolution of the oedema within the cord but the cavity remained unchanged.



**Figure 1** (A) Patient 1: T2 weighted axial section through the plane of C5/6 showing a small, well defined cavity within the right side of the cord. (B) Patient 2: sagittal T2 weighted image of the thoracic spine showing extensive high T2 signal in central cord substance varying in intensity and conspicuity. (C) and (D) Patient 3: sagittal T2 weighted image of the lower thoracic/upper lumbar spinal segment and T2 weighted axial image through T11 showing well defined asymmetric cord cavities of CSF intensity involving the lowermost thoracic segments and upper part of the lumbar expansion.

### Patient 3

A 31 year old primagravida underwent a combined spinal and epidural procedure at a documented level of L2–3 before an elective caesarean section. A bolus dose of 2 ml 0.5% bupivacaine and 25  $\mu$ g fentanyl was initially injected into the subarachnoid space through a 26 gauge spinal needle. During this procedure she experienced considerable local discomfort and brief shooting pain down the right leg but adequate analgesia was achieved before surgery. An epidural catheter was then introduced to a length of 3 cm beyond the tip of the needle. The patient later reported that she experienced further bouts of severe pain radiating down her right leg when additional epidural analgesia was administered for postoperative relief of pain. After removal of the epidural catheter she continued to complain of pain and weakness in the right leg. On examination she was noted to have mild weakness of the right leg with diminished pain and temperature sensation to T10 on that side. Vibration sense and proprioception were relatively preserved. The right knee jerk was diminished but all other deep tendon reflexes were normal and the plantar responses were bilaterally flexor. Magnetic resonance imaging of the lower thoracic and lumbar cord showed two asymmetric areas of signal change within the cord, isointense with CSF, lying either side of the midline extending from T11 to L1 on the right and T11 to T12 on the left (fig 1 C and D). Over the next 10 days power gradually returned to normal in the right leg.

There was some improvement in sensation although she continued to experience pain in the lower back and right leg, which has persisted after 4 years of follow up.

### DISCUSSION

Severe neurological complications resulting from epidural anaesthesia and analgesia are rarely reported. The above patients all developed motor and sensory impairment after epidural catheterisation. Subsequent MRI on all three patients disclosed similar lesions within the spinal cord corresponding to the segmental levels of motor and sensory impairment identified on clinical examination. These were hyperintense on T2 weighted images and hypointense on T1. In each case the distribution of the lesion was tubular, clearly demarcated, and not typical of the distribution of an infarct attributable to arterial occlusion or hypoperfusion. No other pathological changes that could explain the clinical findings were identified on the scans. We therefore propose that the mechanism responsible for the early onset of neurological symptoms was direct penetration of the spinal cord during attempted epidural catheterisation and subsequent injection of fluid into the substance of the cord, producing localised hydromyelia. It is difficult to envisage the blunt tip of the catheter passing upward within the spinal cord, contributing to the lesion, but this is also a possibility. The injection of drug solutions may, however, explain the extension of lesions seen

on MRI, many segments above the point of catheter insertion, due to high fluid pressure exerted within the cord. This could also account for the splitting of the lesion into two separate cylindrical columns at T12 level seen in patient 3. However, because the MRI in this patient showed the cord terminating at the normal L1 level, we have difficulty explaining the site of the lesion on the basis of the anaesthetic procedure documented in the notes. It is conceivable that inadvertent injection into a spinal root, during the initial subarachnoid injection or after placement of the epidural catheter, led to tracking of fluid up into the cord. Alternatively, the segmental level of injection might have been higher than assumed from reference to the conventional anatomical landmarks. A recent study assessing the ability of anaesthetists to identify a marked lumbar interspace by palpation of these anatomical landmarks disclosed that the correct position was identified in only 29% of cases and errors ranged from one space below to four spaces above the presumed level.<sup>19</sup> A similar discrepancy was noted in the case reported by Barontini *et al*,<sup>16</sup> where it was postulated that the procedure may have been performed at a level higher than that suggested in the patients' records.

Experimental studies have indicated that all local anaesthetic agents are potentially neurotoxic.<sup>20</sup> Polyethylene glycol contained in methylprednisolone acetate is also known to cause necrosis of neuronal tissue.<sup>21</sup> Additional damage may therefore have occurred from the direct toxic effects of the active drugs within the cord.

To date there have been three previous reports, involving a total of four patients, where direct trauma to the spinal cord during attempted epidural catheterisation has been implicated as the possible cause of neurological complications.<sup>16-18</sup> In the case described by Bromage and Benumof,<sup>18</sup> an air bubble was identified within the cord of a patient who was left paraplegic after an attempted thoracic epidural using the loss of resistance to air technique to identify the epidural space. The remaining reports have described lesions similar, although less extensive, to those identified in our patients. With the exception of the patient described by Barontini *et al*,<sup>16</sup> all previous cases have occurred when the epidural was performed under general anaesthesia or intravenous sedation where any immediate sensory symptoms of needle trauma to the spinal cord would have been masked.

Although cases such as these are likely to be rare, we strongly endorse previous recommendations that epidural anaesthesia and analgesia should preferably not be initiated under general anaesthesia.<sup>17, 18</sup> If further pain relief is required postoperatively the epidural catheter should ideally be placed before administering the general anaesthetic or after the patient has regained consciousness. Intravenous sedation should also be used with caution and the patient should remain sufficiently awake to be able to respond to significant painful stimuli. Severe local or radiating pain should then be regarded as an indication of possible mechanical stimulation of the cord or nerve roots and the needle withdrawn immediately. The additional use of fluoroscopic guidance during needle placement does not guarantee against accidental trauma to the cord.<sup>17</sup>

Any patient in whom there is a suspicion of direct trauma to the spinal cord during attempted epidural catheterisation should undergo detailed neurological assessment. If symptoms and physical signs persist, MRI of the cord should be performed and this may demonstrate the typical appearances disclosed in our cases. The possible value of high dose steroid treatment in this setting is unknown but it should be considered. Use of methylprednisolone within 8 hours has been shown to improve neurological outcome in other forms of acute spinal cord injury.<sup>22</sup>

The prognosis in all three cases was for progressive recovery of motor function over a period of days to months. This may reflect the resolution of oedema and reabsorption of some of

the fluid within the lesion. Unfortunately, spinothalamic sensory impairment and severe spontaneous pain over the affected area has persisted in all three patients.

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