The authors present a case in which paraplegia developed following administration of caudal epidural steroid injection (ESI) and discuss the different pathophysiological mechanisms involved. The authors strongly recommend that 0.5% bupivacaine should be used with caution for caudal epidural injection.

**Keywords**: caudal epidural injection; bupivacaine; corticosteroids; paraplegia.

**INTRODUCTION**

The authors would like to report a case of paraplegia following a caudal epidural steroid injection (ESI). This case report discusses not only various complications of ESI in general, but also the specific pathophysiological mechanisms implicated in the development of paraplegia after epidural injection in particular.

**CASE REPORT**

A 56-year-old otherwise fit and well female presented to Accident and Emergency department with severe right sided leg symptoms. The clinical examination and subsequent MR scan confirmed a right sided L5/S1 disc prolapse with S1 nerve root compression but no distal neurological deficit. A caudal epidural injection was advised.

The procedure involved image guided placement of an 18 gauge epidural needle through the sacral hiatus under local anaesthesia, with the patient in prone position. An epidural catheter was fed through the needle and was advanced up to the L5/S1 disc space. An epidurogram was obtained to confirm the location of the catheter in relation to the epidural space. A total of 20 ml of the drug containing a mixture of Triamcinolone and Bupivacaine (0.5%) was injected after confirming a dry tap. In the recovery room, we noticed that she developed profound hypotension and bilateral grade zero power in the lower limbs within 20 minutes from the time of epidural injection. The level of the temporary neurological deficit reached up to the T12 dermatome.

Fluid resuscitation was immediately commenced. The consultant anaesthetist suggested that the weakness could be due to a possible spinal anaesthesia. An urgent MR scan was arranged to rule out formation of a spinal haematoma or another space occupying lesion. Her lower limb neurology was closely...
monitored. MR scan did not show any evidence of a haematoma.

Subsequently, the patient regained full sensory function in both lower limbs in 16 hours and full motor function in 20 hours following the procedure.

**DISCUSSION**

Epidural steroid injection is recommended in patients with radicular pain or pain suggestive of radiculopathy (16). The value of caudal epidural steroid injections (ESI) in treatment of low back pain and sciatica is controversial (3). The rationale for administration of ESIs is based on the assumption that inflammation of the spinal nerve root causes radicular pain and the epidural corticosteroids relieve this pain, allowing time for healing and physical therapy. It is believed that ESIs are mainly effective in treating acute radiculopathy at intermediate term follow-up, even though it was found to have no long-term benefit (17). Absolute contraindications to ESI include systemic infection, local infection at the site of the planned injection, bleeding disorder or anticoagulant therapy, allergy to glucocorticoids, and patient refusal (5).

Although epidural steroid injection is a common tool of “conservative” therapy in patients with radicular pain (16), it is not inherently benign (13). Several rare complications have been reported in the literature. They include local discomfort, infection, steroidal side effects, dural puncture, postdural puncture headache (PDPH), epidural haematoma, and nerve injury (1). Other rare complications include increased back pain, facial flushing, vasovagal reactions, episodes of nausea, increased leg pain (6) and even transient blindness (18). Most complications are more related to the invasiveness of the procedure than to the injection itself (14).

The most common technical complication of ESI is inadvertent dural puncture, which has an incidence as high as 7% (2). PDPH occurs in 20% to 50% of all dural punctures (12). Paraplegia following administration of epidural injection has been reported in the literature but varied pathophysiological mechanisms were suggested. Tripathi et al (16) reported a case in which an accidental “intracord injection” of steroid solution during epidural block using fluoroscopy in a conscious patient, caused paraplegia. The patient reported paraesthesia in the legs during injection (16). McLain et al (13) reported a unique case of transient, profound paralysis after ESI “without fluoroscopy control”, which resolved over a 2-3 hours period. In their opinion the period of recovery was consistent with an acute but brief compressive injury and inconsistent with an anaesthetic effect. They proposed three possible mechanisms (a) inadvertent thecal penetration during injection may have produced an atypical anaesthetic block; (b) loculation of the injected fluid may have caused a transient compressive lesion; or (c) intrathecal injection may have produced an iatrogenic arachnoid cyst. In our patient recovery took over 15 hours which does not fit into any of the mentioned mechanisms.

Stoll et al (15) reported a case of myelopathy developed in an otherwise fit and healthy male eight days after ESI due to development of epidural haematoma formation. They mentioned that epidural haematoma formation is a common complication after ESI in patients with coagulation disorders, though their case represents an exception to that. Cases of paraplegia have been reported after transforaminal steroid injection in which either an anatomical variation of artery of Adamkiewicz (10) or vascular injury leading to an infarction of the spinal cord (9,11) were implicated in the pathophysiology. Development of an epidural abscess has been implicated as one of the causes for neurological deficit after ESI (7).

Craig et al (8) reported a case of severe flaccid paraplegia after administration of epidural injection for labour and delivery, which took 16 months to recover. They attributed this to the 1.5% benzyl alcohol preservative contained in a 0.9% saline solution used for post delivery epidural injection. But the solution that was used in our case did not contain such chemicals. Bilir et al (4) reported a case of cauda equina syndrome after epidural injection of triamcinolone and bupivacaine, which developed 3 hours after injection and resolved completely after eight hours. They proposed that it is most probably due to the anaesthetic effect of the injection and suggested that continued vigilance for neurologic deterioration after epidural steroid
injections is important. In our patient, the same mixture was used but the strength of bupivacaine used was 0.5%. We assume that the 0.5% strength could have been the underlying reason for the patients to develop paraplegia not only within shorter time after the procedure but also for a prolonged duration of the anaesthetic effect.

**CONCLUSION**

The surgeon and the patient should be made aware that transient paraplegia is a possible complication after caudal epidural injection. We strongly recommend that 0.5% bupivacaine should be used with caution for caudal epidural injection.

**REFERENCES**