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Inadvertent extra-arachnoid (subdural) injection of a local anaesthetic agent during epidural anaesthesia

A case report

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Summary

Profound hypotension occurring in a patient 2 hours after initiation of combined general and epidural anaesthesia for a myocutaneous free-flap graft was found to be attributable to subdural/extra-arachnoid injection of 0.5% bupivacaine. The initial diagnosis was based on a negative aspiration test, a delayed widespread sensory and sympathetic block, and the absence of any other obvious cause for the hypotension. This was confirmed by myelography, which demonstrated an extension of the contrast medium predominantly posteriorly in the spinal canal with excessive spread along the nerve roots. Posture and coughing did not affect the spread.


The incidence of subdural/extra-arachnoid injection following attempted epidural placement of a needle or catheter for anaesthesia or analgesia has been estimated to be 0.82%, although an incidence of 10% has been reported following epidural space cannulation for myelography.1 Lubenow et al.1 suggested 5 clinical criteria (2 major and 3 minor) for the diagnosis of subdural/extra-arachnoid injection of local anaesthetic agents. Both the major criteria (a negative aspiration test and an unexpected widespread sensory block after epidural injection) and any one of the 3 minor criteria (delayed onset, variable motor block and sympatholysis out of proportion to the dose of local anaesthetic injected) confirms the diagnosis.

This report illustrates an instance of subdural/extra-arachnoid cannulation and injection of a local anaesthetic agent in which the aberrant catheter placement was confirmed radiographically. The complication presented a particular diagnostic problem when it occurred under the umbrella of general anaesthesia.

Case report

A 31-year-old man presented for a myocutaneous free-flap graft to the right leg. The choice of anaesthetic technique was general combined with epidural anaesthesia in order to maximise graft blood flow.3

A 16G Tuohy needle and epidural catheter was inserted through the L3/4 interspace with the patient in the left lateral position, the epidural space being identified by loss of resistance to air. Neither cerebrospinal fluid (CSF) nor blood could be aspirated after siting of the catheter. Intrathecal and intravascular placement was further excluded by administering a test dose of 2 ml 2% lignocaine with adrenaline 1/200 000 via the catheter.

A dose of bupivacaine 0.5%/15 ml was injected via the catheter, and this produced surgical anaesthesia of the lower extremities extending up to the 10th thoracic dermatome within 20 minutes. No further injections were given via the epidural catheter. The level of the block seemed fixed at 30 minutes and general anaesthesia was induced with thiopentone and suxamethonium, the trachea was intubated and the patient ventilated. Anaesthesia was maintained with air, oxygen, halothane and pancuronium. In addition to other routine modalities, monitoring included that of radial arterial pressure and central venous pressure (CVP).

The patient remained haemodynamically stable for 2 hours after the administration of the local anaesthetic agent, during which period he received 2 000 ml of Ringer's lactate solution. Then, without any obvious cause, the blood pressure and CVP started to fall. Despite active fluid administration, the blood pressure dropped to 50/0 mmHg and the CVP dropped to 0 within the next 15 minutes without a change in the pulse rate, suggesting a high sympathetic block. Phenylephrine was added to the fluid infusion and titrated against blood pressure, and surgery and anaesthesia were terminated leaving the epidural catheter in place. It took approximately 35 minutes for the blood pressure to stabilise. When the patient was fully awake and extubated it was found that the anaesthetic block extended to the 4th thoracic dermatome and appeared patchy in nature. Analgesia persisted for 4 hours after the initial injection of the local anaesthetic agent.

After obtaining the patient's consent, 15 ml of iopaminol (200 mg/ml) was injected via the epidural catheter and radiographs of the spine were taken (Fig 1), confirming subdural/extra-arachnoid placement of the epidural catheter.4,5

The patient suffered no permanent sequelae.

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Clinical subdural/sub-arachnoid injection of a local anaesthetic agent presents with unexpectedly widespread neural blockade following negative aspiration and test-dose injection. Associated symptoms, such as pupillary dilatation and hypotension, are consistent with high sympathetic blockade. The onset time of the exaggerated block is delayed and there is variability in the extent of motor block. Furthermore, the block is often patchy in nature and full sensation can take as long as 5 hours to return.

In most instances the diagnosis of subdural/sub-arachnoid block is based on clinical criteria, particularly the late onset of an extended block. Other cases of subdural/sub-arachnoid placement of both local anaesthetic agents and contrast medium have been confirmed radiologically.

Subdural/sub-arachnoid block can be distinguished from high subarachnoid block in that the former generally produces more moderate hypotension, has a slower onset of anaesthesia and causes progressive respiratory depression rather than sudden apnoea. Contrast medium injected into the subdural space has a typical radiological appearance. The spinal cord is not clearly defined, there is excessive extension of the contrast medium along the nerve roots and this is found predominantly posteriorly to the canal. In the longitudinal plane, the spread is mainly cephalad. On screening, the spread is not affected by changes in posture and by coughing, as would be expected with subarachnoid injection.

A widespread block, out of proportion to the volume of local anaesthetic injected, relatively late onset and long duration of a patchy block following negative routine tests for intrathecal and intravascular cannulation was found in our patient.

This clinical picture was suggestive of subdural/sub-arachnoid injection of local anaesthetic agent. The report illustrates the problems of this complication of regional anaesthesia when the presenting signs are those of profound hypotension presenting during general anaesthesia. In these circumstances, the dermatome level reached by the block cannot be determined until the patient has regained consciousness, and the anaesthetist is thus deprived of one of the major signs (widespread sensory blockade) and one of the minor signs (variable motor block) that characterise the condition. Furthermore, the additional minor sign of disproportionate sympathetic loss is less certain to be attributable to an unusual block during a procedure in which significant blood loss is a feature and also when the effects of general anaesthetic agents also contribute to sympatholysis.

This presentation of the complication under general anaesthesia poses special problems in diagnosis and anaesthetists should be aware of the possibility when the major manifestation is one of cardiovascular collapse.

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