CASE REPORT

Accidental subdural injection of local anaesthetic: diagnosis by pressure measurement and response to aspiration of injectate

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Summary

A healthy 22-year-old man received an initial injection of 12 mL of lignocaine/bupivacaine solutions (2 mL test, then 10 mL) into an epidural catheter. This produced a satisfactory regional blockade that seemed to be epidural, but, when a supplementary 6 mL injection was given 1 h later, the patient developed impaired motor function as far as the upper cranial nerves, with loss of pinprick sensation to the shoulder. The emergence of fluid dribbling freely from the catheter prompted measurement of the pressure, which was 36 mmHg. The fluid was proved not to be cerebrospinal fluid (CSF) by the absence of glucose (on dextrostix), by the appearance of turbidity with added thiopentone, and later by microscopy. Slow aspiration of 7 mL of the presumed injectate reduced the pressure in the catheter to 8 mmHg, which promptly reversed the additional excessive blockade, allowing surgery to proceed uneventfully. The retrieval of injectate argues strongly that the catheter tip had found its way subdurally, and the promptness of the reversal with aspiration argues for a mechanical rather than a pharmacological cause for the extensive neurological dysfunction after the second injection. Pressure measurement and aspiration may be helpful in other similar cases.

Keywords: REGIONAL ANALGESIA; EPIDURAL ANAESTHESIA; SUBDURAL BLOCKADE.

Introduction

Inadvertent injection of local anaesthetic into the subdural space is a recognized potential complication of the epidural analgesic approach, whether because of initial malplacement or subsequent migration of the epidural catheter. It is one cause of an unexpectedly profound and extensive conduction block with supplementary doses of local anaesthetic [1±4]. The block may be delayed in onset and be accompanied by unconsciousness, respiratory arrest, hypotension, bradycardia [2,3] and aphonia [5]. It is distinguishable from the effects of inadvertent intrathecal injection by the aspiration of the local anaesthetic injectate rather than CSF from the epidural catheter. The conventional management, as for a total spinal injection, is supportive and expectant – to maintain pulmonary ventilation and stable haemodynamic responses until the local anaesthetic effect wears off. This case report describes a more proactive therapy–aspiration of injectate from the catheter. It provides an insight into the possible mechanism of blockade.

Case report

An otherwise fit 22-year-old man presented for urethroplasty for post-traumatic stricture. He weighed
55 kg and was 160 cm tall. One hour after he had been premedicated with pethidine 50 mg and promethazine 25 mg intramuscularly (i.m.), an 18-g Tuohy needle was placed by a midline approach into the L3–4 interspace, with the patient in the lateral position – the epidural space being identified by the hanging drop technique. The tip of an epidural catheter (18-g Mini-pack system 2, Portex Ltd, UK) was passed 3 cm into the epidural space. After an uneventful test dose of 2 mL of lignocaine 2% with adrenaline 1:200,000, an easy injection was made of 10 mL of a 50:50 mixture of 2% lignocaine and 0.5% bupivacaine, which produced anaesthesia to pinprick up to T8. Surgery was started in the lithotomy position with monitoring of ECG, blood pressure (which fell modestly), pulse oximetry and the skin temperature of the big toe.

By 60 min, the block had receded to T6, with restoration of normal blood pressure and fall in skin temperature. After testing and aspirating nothing from the epidural catheter, a further 6 mL of the local anaesthetic mixture was given. Ten minutes later, the patient became hypotensive (74/45 mmHg) and bradycardic (58 beats min−1), a condition that responded to atropine 0.6 mg and infusion of 300 mL of Ringer’s lactate solution in 10 min. A few minutes later, the patient complained (in a feeble voice) of difficulty in breathing. He was breathing at a rate of 24 min−1 and tidal volume of about 125 mL by Wright’s respirometer, and with paradoxical chest movements although there was no obstruction: the pulse oximetric readings fell from 98 to 84% until the introduction of assisted face-mask ventilation with 100% oxygen. He remained conscious but had miosis and difficulty with eye opening. The upper level of insensitivity to pinprick was C6. Testing for the lower levels of motor and sensory block could not be undertaken without interfering unacceptably with surgery. Subarachnoid injection of the supplementary dose of local anaesthetic was suspected, but it was noted that fluid was dribbling unusually freely from the opened injection port of the epidural catheter. The fluid became turbid with the addition of 2.5% thiopentone 0.5 mL and was negative to glucose on dextrose [6]. When the hub of the catheter was connected to a standard pressure transducer (Hewlett Packard, USA) zeroed at the level of the mild axilla, it recorded a pressure of 36 mmHg. These observations suggested that the injected anaesthetic solution had been injected into, and become loculated in, the subdural space.

Further aliquots of the fluid were slowly aspirated to a total of 7 mL, which brought the pressure in the epidural catheter to 8 mmHg. Further testing of the aspirate with thiopentone and dextrostix continued, and later formal reports for biochemistry and cytology confirmed that it was not CSF. The reduction in the pressure recorded from the catheter was followed within 5 min by the restoration of satisfactory diaphragmatic breathing [7]. Aminophylline 200 mg was given intravenously (i.v.) to assist diaphragmatic contractility [8–10] and the tidal volume increased to 350 mL, with an increase in oximetric saturation, pulse rate and blood pressure. After 30 min during which surgery was completed, the patient was fully conscious and orientated with normal voice, pupil reactivity and eye movement. In view of this and for fear of recreating the conditions that had brought about the temporary neurological deficits, it was decided not to pursue radiological investigations of the placement of the epidural catheter. The catheter was removed without further use or incident.

The patient was discharged on the 7th post-operative day after recovery uncomplicated by headache or neurological deficit.

Discussion

This patient presented the typical clinical picture of subdural injection of local anaesthetic through an epidural catheter. The differential diagnosis of epidural or intrathecal injection was made by recording a higher pressure from the epidural catheter than is normal for an epidural injection [11]. The tests indicated that the aspirate was not CSF and a reduction in the pressure resulted from aspiration. The response i.e. fall in pressure also emphasized the therapeutic importance of this manoeuvre. The decision not to pursue radiological investigation was in keeping with published recommendations [12,13].

Our presumptive explanation for the events in this case was that both injections went subdurally, that the addition of the second to the first caused the extraordinary spread to the cervical segments and that its aspiration brought about the recession of block. The rapidity of the recovery after the aspiration argues against the most obvious causative mechanism that
the extension of block was due directly to the pharmacological effects of local anaesthetic bathing the affected nerves [14]. This rationale underlies manoeuvres to prolong the local persistence of local anaesthetic by adding adrenaline [15], colloidal substances [16], or altering the pH to increase the ionization [17,18]. Had the effects been pharmacological, they would be expected to persist after the aspiration of injectate until the local anaesthetics had dissociated from binding to nerves.

It seems more likely that the more cephalad neurological deficits were brought about mechanically, possibly by distortion of nerve roots by the cephalad passage of the injectate in subdural channels that did not bring it directly into contact with the nerve roots. This is consistent with the high pressures recorded from the epidural catheter and the speed with which recovery took place once the pressure was relieved. A similar explanation has been advanced for the neurological sequelae of subdural haematomata [19].

The identification of this mechanism is important because it provides the basis for therapeutic action that can have more or less immediate effects.

References

3 Reynolds F, Speedy HM. The subdural space: the third place to go astray. Anaesthesia 45: 120–123.


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