Sudden onset of subarachnoid block after subdural catheterization: a case of arachnoid rupture?

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Summary
We describe a patient who received an apparently uneventful extradural block in labour but developed rapid extension of neural block within minutes of receiving her first incremental dose 2 h later. Computed contrast tomography revealed radio-opaque dye within both the subdural and subarachnoid spaces, but none within the extradural space. This case report demonstrates that subdural spread of low-dose local anaesthetics is not always clinically distinguishable from extradural analgesia and that the arachnoid membrane may subsequently perforate with potentially serious consequences. (Br. J. Anaesth 1996; 76: 322–324)

Key words

Case report
A 23-yr-old nulliparous Asian woman requested extradural analgesia for pain relief in early labour. The patient was in good health and there were no contraindications to the procedure. She was of slim build and her spine was easily palpable. The operator (D.W.E.) had previously performed approximately 50 extradurals without complication. With the patient in a sitting position, an 18-gauge Tuohy needle was inserted into the extradural space at the L3–4 interspace without difficulty. Loss of resistance to air was detected at 4 cm and an extradural catheter (Portex, Kent) was inserted with minimal resistance. After removal of the Tuohy needle, however, it was estimated that no more than 1 cm of catheter was lying within the extradural space. Consequently the procedure was repeated using the same interspace and a new catheter inserted without difficulty such that an estimated total of 4 cm lay within the extradural space. The extradural needle was not rotated on either attempt and the patient did not complain of pain, paraesthesia or headache during insertion of the catheter. Nothing could be aspirated from the needle or catheter on either occasion.

After a further negative aspiration test with the patient lying on her side, a 3-ml dose of 0.25 % bupivacaine was given via the catheter. This test dose produced no detectable sensory or motor changes and after 5 min another 7 ml of the same solution were given. Satisfactory analgesia was achieved approximately 5 min later and there was no change in arterial pressure. When so instructed, the patient was able to raise both legs from the surface of her bed. No further formal assessment of the height of the block was made.

Two hours later, further analgesia was requested. With the patient in a semi-recumbent position, and after a negative aspiration test, a pre-mixed proprietary preparation containing bupivacaine 12.5 mg and pethidine 25.0 mg in saline 10 ml (Delta West, Perth, Australia) was injected by the attending midwife via the extradural catheter. Five minutes later, the patient complained of nausea, shortness of breath and numbness in her hands and face. An urgent request by the midwife for an anaesthetic review was met within minutes.

On examination, the patient was found to be drowsy but responsive to questions and commands. She was unable to move either of her lower limbs but retained a weak bilateral handgrip and was still able to lift her head from the pillow. Anaesthesia to cold was present over the entire chest wall below the clavicles but there was no sensory loss over the face and neck. Marked nystagmus was observed. The pupils were small and equal, and it is possible that bilateral ptosis was present, although it was difficult to be certain because of the patient’s ethnic origin and her state of drowsiness. There was no obvious injection of the sclerae and both nostrils were patent, but the presence of a bilateral Horner’s syndrome could not be excluded.

During this period of assessment, the patient remained remarkably calm and co-operative. Her arterial pressure did not change and there were no signs of respiratory depression or fetal distress. Despite complaining of some shortness of breath, her vital capacity was recorded as 1.5 litre. She was given supplementary oxygen and pulse oximetry revealed an oxygen saturation of 99 %. In view of these findings, it was decided not to administer naloxone unless there was any deterioration in her state of consciousness or ventilation.

A provisional diagnosis of subarachnoid catheter migration was made and instructions given that further analgesia would be administered only by an anaesthetist. Over the next 2 h there was complete recovery of neural function. Five further top-up doses consisting of 0.25 % bupivacaine 2 ml were given.

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given for pain during the rest of her labour. On each occasion, analgesia occurred within the duration of a contraction and lasted for an interval of 45–60 min. Incomplete motor block of the lower limbs returned after each injection. Eight hours after insertion of the extradural catheter, a healthy baby was delivered with the aid of outlet forceps.

Although we strongly suspected on clinical grounds that the catheter tip had ultimately become situated within the subarachnoid space, we were puzzled why the response to the initial dose had been so uneventful. In which compartment(s) had the catheter been originally placed? Did the catheter migrate from the extradural or subdural space, or was it in the subarachnoid space from the outset? To try and answer these questions we felt it would be useful to confirm the position of the catheter radiologically. The patient agreed to this proposal and gave her informed consent for computer tomography (CT) using contrast medium.

Four hours after delivery the patient was transferred to the CT scanner and 3 ml of iodinated contrast medium (iopamidol 300 mg ml\(^{-1}\)) were introduced via the extradural catheter; 5-mm thick contiguous axial CT scans were then performed through the upper lumbar spine. This showed contrast medium only within the subdural and subarachnoid spaces while the extradural space was entirely contrast-free (fig. 1).

The day after delivery, the patient developed typical features of a dural puncture headache which gradually became more severe. An extradural blood patch was performed on day 4 and her headache disappeared after 3 h. She remained headache-free when discharged from hospital 2 days later.

Discussion

Subdural catheterization is a well recognized complication of extradural analgesia with an estimated incidence, according to one retrospective study, of 0.8% [1]. The diagnosis in that study, however, was based solely on clinical criteria and was not confirmed radiologically. The true incidence of subdural catheterization, therefore, remains unknown because a definitive diagnosis depends on CT imaging, an investigation that is justified only if catheter misplacement has been suspected clinically.

While two attempts were made to site the extradural catheter in our patient, the procedure was straightforward on both occasions; the first catheter was pulled out accidentally when the needle was removed. There was certainly no reason to believe that the dura had been perforated, injured or otherwise predisposed to catheter misplacement on either occasion, although such events can never be entirely excluded.

It has been claimed by various authors that subdural local anaesthetic spread can be diagnosed, or at least strongly suspected, on clinical grounds. According to Collier [2] for example, subdural spread is typically accompanied by: relatively slow onset of neural block (i.e. 15–20 min with further progression over the next 20 min); limited motor block; only moderate hypotension; progressive respiratory depression and inco-ordination rather than sudden apnoea; and recovery after 2 h [2]. Other clinical clues have also been suggested, such as: exaggerated hypotension and unexpectedly high motor block [1, 3, 4]; relative sparing of motor function in the lower limbs [4]; unilateral block [5]; the presence of a Horner’s syndrome [3]; and, possibly, acute headache during injection [6]. Without radiological confirmation, however, the reliability of these clinical signs in distinguishing between extradural, subdural and subarachnoid injection is conjectural.

One of the important features in this case report is that our patient displayed none of the above symptoms or signs after the initial therapeutic dose.
Aspiration of both needle and catheter was negative, and there was no abnormal response to the test dose. Thus there were no grounds to believe that the catheter was other than correctly sited in the extradural space. Yet the CT scan clearly demonstrated no spread within the extradural space. This finding implies, therefore, that subdural spread of local anaesthetic drugs when given in low dosage is not necessarily clinically distinguishable from extradural or even subarachnoid spread.

In a previous case report, we described an obstetric patient in whom an extradural catheter was subsequently shown to have been confined solely within the subdural space [6]. We now report a patient in whom we believe the catheter was initially within the subdural space but later became situated intrathecally, as postulated in the review by Reynolds and Speedy [7]. While it has been found to be impossible to puncture the intact postmortem dura with a Portex catheter, the thinner arachnoid membrane is easily perforated [8]. Thus it is not difficult to appreciate how readily transarachnoid migration of a subdural catheter might occur in clinical practice.

In our patient, the clinical response to subsequent doses was so distinct from that following the initial dose that we felt confident in assuming that the catheter had become situated intrathecally. This was confirmed not only radiologically but also by the fact that she later developed a spinal headache. An alternative explanation might be that the side holes of the extradural catheter were situated within both the subdural and subarachnoid spaces from the outset and that any disparity in clinical response depended entirely on the vagaries of differential flow [9, 10]. However, the fact that our patient responded so consistently to subsequent doses, and that the extradural space was altogether free of radio-opaque dye makes this alternative explanation, we believe, exceedingly unlikely.

Fortunately, the initial intrathecal dose was not sufficient to cause severe respiratory or circulatory depression in our patient and resuscitation was not required. This is now the second time a mixture of bupivacaine 12.5 mg and pethidine 25 mg has been given intrathecally in our unit without causing serious harm or injury [11]. Our experience thus supports the contention that extradural management during labour is associated with an intrinsically greater margin of safety when local anaesthetics are used in low doses [12].

If our interpretation of events concerning this case report is correct, then presumably a subdural catheter can perforate the arachnoid membrane and become situated within the subarachnoid space at any time. This, too, has important implications. Although our patient received intermittent bolus doses, there is no reason to believe that other regimens, such as continuous infusions or patient-controlled extradural analgesia, are immune to transarachnoid migration. Our case report helps to explain just how easily subdural catheter placement may escape notice, and reinforces the need to meet recommended standards of supervision and management among all patients receiving extradural analgesia in labour, irrespective of the drug delivery system.

References