Case Reports

Subdural Intrathecal Catheter Placement: Experience with Two Cases

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Background: Subdural migration of epidural catheters is well known and documented. Subdural placement of intrathecal catheters has not been recognized. Two cases of sudural placement of intrathecal catheters are presented.

Objective: The possibility of subdural migration of epidural catheters and its manifestations has been well documented. The following 2 cases demonstrate that intrathecal catheters can enter the subdural space upon placement.

Case Reports: The first case is a 52-year-old male with multiple sclerosis receiving a pump for intrathecal baclofen. It worked well for 10 years, but after 2 months of inadequate relief despite a 2-fold increase in baclofen, the catheter was imaged. The catheter pierced the arachnoid in the lower thoracic spine and tunneled subdural. It then pierced the arachnoid again, re-entering the cerebrospinal fluid (CSF) in the cephalad portion of the thoracic spine. Over time, the tip became covered with tissue, preventing direct CSF communication and causing subdural drug sequestration.

The second case is a 54-year-old male with chronic bilateral lower extremity pain having a pump placed for pain control. Because of inadequate relief after implantation, the catheter was imaged. It pierced the arachnoid at L4-L5 but became subdural at T12-L1. At the time of surgical revision, the catheter was pulled back to L2. Repeat imaging showed it to be entirely subarachnoid, and analgesia was restored.

Conclusions: These cases differ from others in the literature because the catheter was apparently subdural at the time of initial implantation. As these 2 cases demonstrate, this placement may manifest immediately, but it may remain undetected for a prolonged period. Initial subdural placement should be considered along with catheter migration into the subdural space in the differential of a malfunctioning pump.

Key words: intrathecal catheter, subdural, migration

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The possibility of subdural placement of epidural catheters and their concomitant manifestations has been well documented (1-4). Subdural migration after catheters have been implanted has also been reported (5-10). We present 2 cases in which the catheter did not migrate but instead was passed subdural during the initial placement.

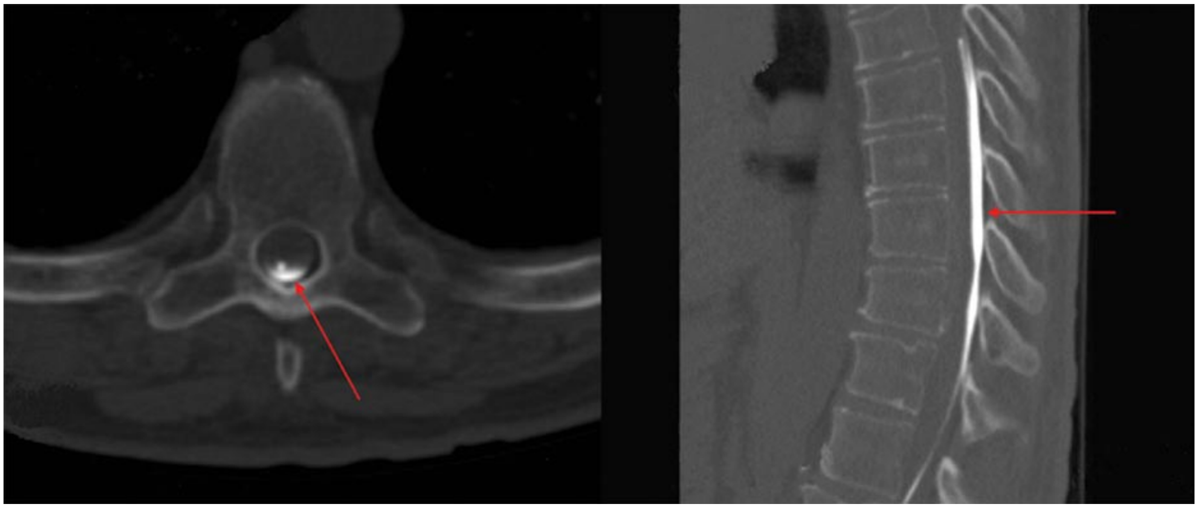

Fig. 1. *Demonstration of catheter in the subdural space. In the axial view, the arrow is pointing to the subdural space. There is a circular dot of enhancement which is the catheter, and the contrast is collecting in the subdural space below it. In the sagittal view, the arrow points again to the subdural space where the contrast is unevenly distributed.*

First Case Report

A 52-year-old male with multiple sclerosis underwent intrathecal catheter and pump implantation 10 years prior to presentation to manage spasticity with intrathecal baclofen. The intrathecal baclofen controlled the spasticity until 2 months prior to consultation. Over these 2 months, the treating neurologist doubled the delivery of the baclofen infusion without response. The programmable pump was evaluated and found to be functioning and emptying appropriately. The integrity of the catheter was evaluated. Side port access demonstrated free flow cerebrospinal fluid (CSF). A CT scan with and without contrast revealed the intrathecal catheter entering the subdural space at T12-L1 and traversing through the subdural space until approximately T2-T3. The injected contrast enhanced the subdural space from T1-T12. Only a faint trace of contrast was identified in the CSF (Fig. 1).

Second Case Report

The second case is a 54-year-old male with a past medical history of hypertension, anxiety, benign prostatic hyperplasia, and a 5-year history of chronic bilateral lower extremity pain with decreases in both proprioception and sensation. He was found to have 5 herniated disks, and after being treated unsuccessfully with oral oxycodone, neurontin, and Cymbalta, he came into the hospital for an intrathecal catheter trial. The patient's pain was excruciating and intractable, and in order to develop a balanced analgesic, drugs were titrated separately. Dilaudid was chosen to attenuate pain response via mu agonist activity. Bupivicaine was used to potentiate the analgesic effect of the opioid and to address small nociceptive fiber conduction. Clonidine was utilized for its central alpha agonist activity which has been shown to be analgesic. This combination of drugs ameliorated his pain, and the decision was made to implant an intrathecal pump. The catheter was placed at the L4-L5 level with its tip at T8. Following implantation, the patient experienced inadequate pain relief. The catheter position was evaluated with a CT myelogram. It was found to be intrathecal at L4-L5 but entered the subdural space at the T12-L1 level (Fig. 2). The catheter was revised by withdrawing the catheter to the L2 level and was seen to be entirely in the subarachnoid space (Fig. 3). After the repositioning, analgesia was attained.

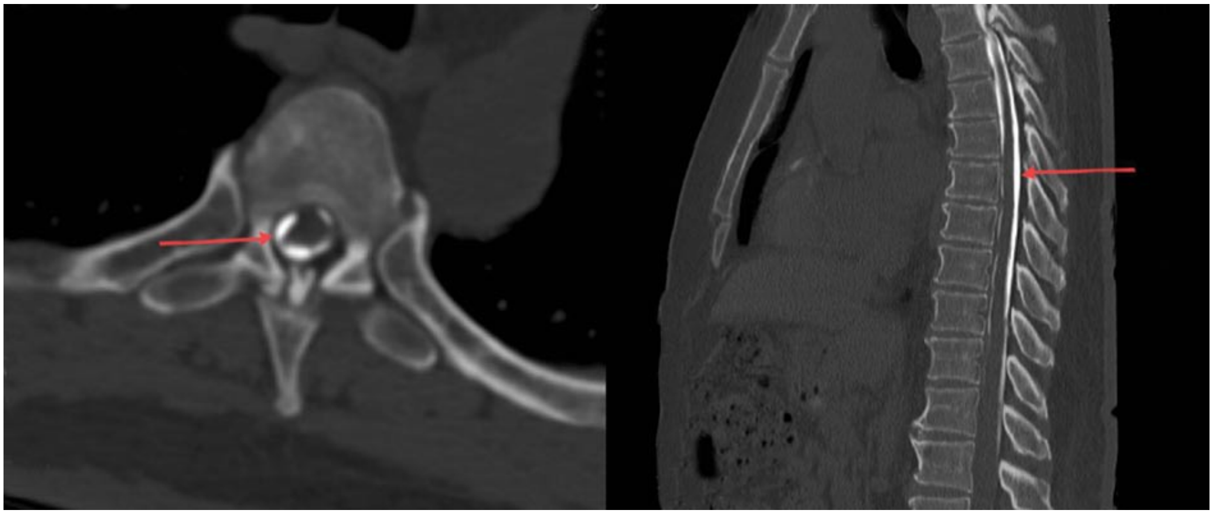

Fig. 2. *This figure demonstrates the catheter in the subdural space. In the axial view, the arrow is pointing to the subdural space. The contrast shows a discontinuous ring around the spinal cord. In the sagittal view, the arrow is pointing to the subdural space where the contrast is not flowing along the spinal cord.*

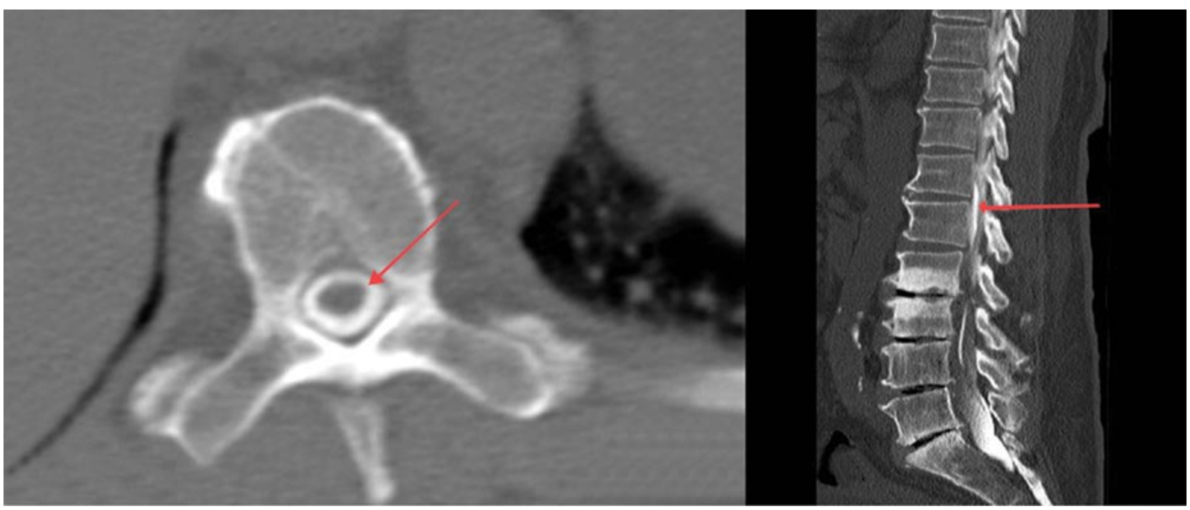

Fig. 3. *This figure illustrates the catheter after it was pulled back into the intrathecal space. In the axial view, the arrow identifies a well-defined uniform ring surrounding the spinal cord. The arrow in the sagittal view also shows the contrast flowing evenly around the cord.*

Discussion

These cases differ from others reported in the literature because migration into the subdural space did not occur over time but rather occured (and, in the first case, was unrecognized) at the time of implantation. During the initial placement, the catheter likely caused a rent in the arachnoid membrane, and the intrathecal catheter passed subdurally along the course of its advancement. Migration in other cases would occur from erosion of the catheter tip through the arachnoid membrane until it was located subdurally.

In the first case report, the patient responded well to the intrathecal baclofen infusion for nearly a decade. The catheter could not have migrated because of the significant length of catheter in the subdural space. It is improbable that the amount of catheter that was subdural could have gotten there merely through migration. Instead, it is likely that the tip of the catheter pierced the arachnoid membrane after traversing the subdural space, delivering the drug directly into the CSF. We postulate that over time, the tip of the catheter which protruded through the arachnoid membrane became covered with tissue, preventing the drug from entering the CSF. The baclofen solution then became sequestered in the subdural space. The small amount of fluid delivered (100 to 200 mcl/day) would be unable to track back along the catheter where it entered the subdural space and, therefore, would not exit into the CSF. This is supported by the fact that 4 mL of contrast were administered for the study, and essentially all 4 mL were sequestered in the subdural space. The CSF that we were able to aspirate came from the site of entry of the catheter into the subdural space at the T12-L1 level. The CSF tracked along the catheter as suction was applied with the syringe, drawing the CSF sudural along the catheter to its tip.

In the second case, during insertion the catheter entered the subdural space shortly after entering the CSF. By retracting the catheter to a level below where it had entered the subdural space, it again became entirely located in the subarachnoid space. This allowed the drug to go directly into the CSF, providing analgesia.

Initial subdural catheter placement should be considered along with migration into the subdural space as part of the differential of a malfunctioning pump. As these 2 cases demonstrate, the subdural location can manifest shortly after catheter placement, but it can also be present for a prolonged period of time before difficulties with the catheter arise. Drug withdrawal as well as recurrence of symptoms may occur as a result.

The question of how to identify and solve the problem of catheter positioning remains. Ross et al(10) suggested implanting a pump with the catheter entering the spinal canal adjacent to the pump, leaving less distance from which the catheter could migrate. Another solution would be to perform routine CT scans with contrast at the time of catheter implantation. However, the cost effectiveness would depend on the true incidence of catheter placement into the subdural space at initial insertion. The lack of knowledge about this incidence makes it difficult to recommend routine use of contrast administration during placement. Until such questions are answered, it is important to continuously expand the differential diagnosis of catheter malfunction and develop new methods to evaluate and treat these complications.

Concluson

These cases differ from others in the literature because the catheter was apparently subdural at the time of initial implantation. As these 2 cases demonstrate, this placement may manifest immediately, but it may remain undetected for a prolonged period. Initial subdural placement should be considered along with catheter migration into the subdural space in the differential of a malfunctioning pump. Intrathecal catheters can pass into the subdural space. This possibility needs to be included in the differential with intrathecal analgesia is inadequate.

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