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Transient lower limb pain following accidental thoracic subarachnoid insertion of an epidural catheter

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Abstract
Transient sensory disturbances, including dysesthesia or neurologic deficits in the lower extremities or buttocks have been described as complications of neuraxial anesthesia. We report a case of transient lower limb pain following the accidental placement of an epidural catheter into the thoracic subarachnoid space. A 31-year-old woman was scheduled to undergo laparoscopic myomectomy. An epidural catheter was accidentally inserted subarachnoid at the T12–L1 intervertebral space with a 2-ml test dose of 2% lidocaine, and was promptly removed. Fulgurant pain and allodynia extending over the L2–5 areas of the left lower limb and buttock started immediately postoperatively. We treated the persistent pain in our patient with epidural infusion of local anesthetics and steroids during her hospital stay, and with carbamazepine and a tricyclic antidepressant after her discharge from the hospital. All signs of allodynia had disappeared on postoperative day 25. Sagittal and axial T2-weighted magnetic resonance imaging (MRI) at the Th12 level revealed a small high-intensity area without mass effect in the ipsilateral dorsal column. The patient's clinical course and MRI diagnosis suggested the inhibition of descending inhibitory pathways resulting from a lesion of the spinal cord as the possible etiology of the transient lower limb pain and allodynia.

Key words Transient lower limb pain · Descending inhibitory pathways · Spinal cord lesions

Introduction

Transient neurologic symptoms (TNS) following spinal anesthesia are defined as transient but distressing back pain postoperatively, involving the buttocks or legs [1], or as pain or dysesthesia in the legs or buttocks, with or without back pain [2]. Randomized studies have revealed a 4%–33% incidence of TNS after lidocaine spinal anesthesia [3].

The possible causes of TNS are as varied as specific local anesthetic toxicity, needle trauma, neural ischemia secondary to sciatic stretching, patient positioning, the pooling of local anesthetics secondary to the use of small-gauge pencil-point needles, muscle spasm, myofascial trigger points, early mobilization, or irritation of the dorsal root ganglion [1]. The intrathecal administration of highly concentrated local anesthetics is known to increase the glutamate concentration in cerebrospinal fluid, resulting in histopathologic changes in neurons of the lumbar spinal cord in animal models [4–6]. Although lidocaine is more likely to cause TNS than other local anesthetics, there is no evidence that this painful condition is associated with any neurologic pathology [7].

We report a case of transient lower limb pain that developed after the accidental placement of an epidural catheter into the thoracic subarachnoid space.

Case report

A 31-year-old, 160-cm, 55-kg woman with a myoma of the uterus was scheduled to undergo laparoscopic myomectomy. Her medical history and preoperative profile were unremarkable. Three milligrams of midazolam was given as premedication 1 h before the surgery. In the operating room, the patient was placed in the right lateral position and a 17-gauge Tuohy epidural needle was introduced at the T12–L1 intervertebral space, using a midline approach. The needle tip was advanced until there was loss of resistance to the injection of air. After negative aspiration, an open-ended epidural catheter with an outside diameter of 1 mm was inserted 5 cm rostral past the tip of the needle. The patient complained of momentary pain radiating to the lower back, which disappeared immediately after the catheter was

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pulled out a few centimeters. We ascertained the disappearance of the radiating pain by direct verbal confirmation with the patient. After no blood or cerebrospinal fluid was drawn during an aspiration test, a 2-ml test dose of 2% lidocaine was injected and the catheter was fixed in position. Subsequently, the patient was placed in the supine position. Shortly thereafter, the patient described a loss of temperature sensation in the lower extremities, and while an attempt was made to confirm the position of the catheter tip, cerebrospinal fluid was aspirated from the catheter when negative pressure was applied with a syringe. Twenty micrograms of fentanyl and 2 ml physiological saline were injected through the catheter, and the catheter was promptly removed.

Induction of anesthesia was initiated with 100 mg propofol and 8 mg vecuronium, and an 8-mm-diameter endotracheal tube was inserted. Anesthesia was maintained with 1%–2% sevoflurane in 50% oxygen balanced with nitrogen. The patient was placed in the lithotomy position with slight Trendelenburg tilt. The peritoneal cavity was inflated with carbon dioxide, and the surgery was accomplished without any respiratory or circulatory complications. Postoperatively, the patient was moved to the recovery area. As she did not complain of any pain and did not reveal lower extremity weakness, she was returned to her ward. Two hours later, she developed fulgurant pain extending from the left buttock down to the tip of a toe.

The day after the surgery, the patient visited our pain clinic for pain assessment and physical examination. Pain intensity was evaluated on a 0- to-100-mm visual analog scale (VAS; a score of 0 was defined as no pain, scores of 10 to 30 as mild pain, and scores of 40 to 100 as moderate-severe pain [8]). She reported allodynia and fulgurant pain with a VAS score of 70 mm extending over the L1 to S1 area of the left lower extremity. Physical examination confirmed that the patient could walk independently, had normal kinetic function of the left quadriceps muscle, grade 5 on manual muscle testing, and normal reflexes of the patellar and Achilles tendons. We inserted an epidural catheter into the epidural space 5 cm rostral from the L2–3 interspace, and injected 6 ml of 1% mepivacaine and 80 mg methylprednisolone. A continuous epidural infusion of 0.5% mepivacaine at the rate of 1.5 $ml \cdot h^{-1}$ was initiated following the confirmation of reduced area of lower limb pain.

On postoperative day 2, numbness was demonstrated over the L2–5 areas of the left lower limb, and allodynia was restricted to only the left plantar aspect. Continuous epidural infusion of 0.5% mepivacaine was maintained, and a bolus injection of 5 ml of 1% mepivacaine and 40 mg methylprednisolone was administered. On postoperative day 4, the area of numbness was restricted to the ventral aspect of the left leg and foot. The area of allodynia was, however, enlarged to L3–5. Therefore, another bolus injection of 5 ml of 0.5% mepivacaine and 40 mg methylprednisolone was administered. The pain intensity gradually reduced from a VAS score of 25 mm on postoperative day 7, to a score of 18 mm with allodynia at L4–5 on postoperative day 13. The continuous epidural infusion of 0.5% mepivacaine was stopped on day 13 and the catheter was removed. The patient was discharged on postoperative day 15. On postoperative day 21, her pain intensity was 16 mm on the VAS score, but the allodynia still remained. She was administered carbamazepine at 100 $mg \cdot day^{-1}$ and amitriptyline at 10 $mg \cdot day^{-1}$. All signs of allodynia had disappeared on postoperative day 25.

Sagittal and axial T2-weighted (T2WI) magnetic resonance imaging (MRI) scans at the Th12 level, taken on postoperative day 21, demonstrated a small high-intensity area (arrow in Fig. 1) without mass effect in the left dorsal aspect of the spinal column that was possibly an old atrophic change (Fig. 1).

Discussion

Transient sensory disturbances, including dysesthesia or neurologic deficits in the lower extremities, have been described as complications of neuraxial anesthesia. Previous reports of transient neurologic symptoms (TNS) were related to bolus doses or the continuous injections of local anesthetics into the lumbar subarachnoid space. We report a case of transient lower limb pain that resulted from the accidental placement of an epidural catheter into the thoracic subarachnoid space in a female patient. To our knowledge, this is the first reported case of a thoracic spinal cord lesion resulting from catheter insertion causing clinically significant transient lower limb pain.

The patient revealed normal motor function of the extremities and did not develop bowel or bladder disturbances throughout the clinical course. The postoperative T2WI MRI showed a possible old small atrophic change extending into the dorsal horn of the ipsilateral dorsolateral fasciculus to the affected lower limb at the same vertebral level as the needle insertion. Therefore, we inferred that the sensory disturbances in our patient originated in the thoracic spinal cord. Previous reports [9,10] have described thoracic spinal cord injury arising from accidental dural puncture or the accidental placement of an epidural catheter, resulting in transient numbness or permanent paraparesis due to spinal edema or hematoma. According to these reports, compression of the spinal cord was a potential cause of the sensory or motor deficits. It is unclear whether the MRI-delineated lesion in our patient compressed the spinal cord, because the high-intensity area was small and

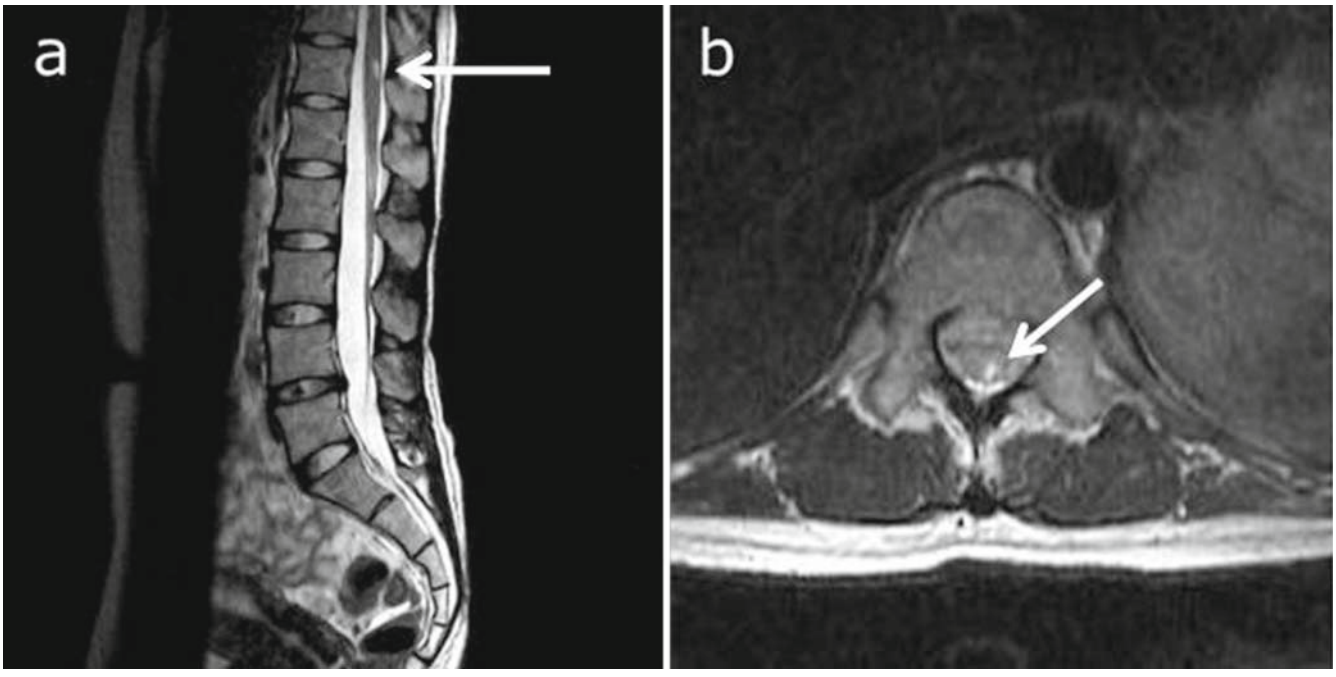

Fig. 1a,b. T2 weighted sagittal (**a**) and axial (**b**) magnetic resonance images depicting a small high-intensity area (*arrows*) in the dorsal left aspect of the spinal column at Th12

limited. We speculate, however, that such a small lesion of the dorsal column would be capable of causing impairment of neural transmission. Pubols et al. [11] reported an increase in spontaneous activity and exaggerated responses to C-fiber input after a thoracic dorsolateral funiculus lesion resulting from lidocaine blockade in cats, and Sandkühler et al. [12] demonstrated that lidocaine-induced lateral funiculi blocks in the cord significantly reduced ipsilateral descending inhibition. These reports suggest that the unilateral inhibition of descending inhibitory pathways at the thoracic level might produce increasing ipsilateral spontaneous activity and responses to nociceptive stimuli. In addition, it has been reported that spinal anesthesia with the use of less than 3% lidocaine does not decrease the incidence of TNS [13, 14]. The possible causes of pain and allodynia in our patient were regarded as arising from the inhibition of descending inhibitory pathways due to a lesion induced by the catheter or by lidocaine injection into the thoracic spinal cord.

TNS starts within 24 h of the initiation of spinal anesthesia and remains after complete recovery from spinal anesthesia. Zaric et al. [7] reported that the symptoms had disappeared spontaneously by the tenth postoperative day. The severe pain and allodynia in the lower limb in our patient commenced several hours after the surgery, and lasted for 25 days. Current therapeutic options for TNS include opioids, nonsteroidal anti-inflammatory drugs (NSAIDs), muscle relaxants, and symptomatic therapy [1,7]. We treated the persistent pain in our patient with epidural infusions of local anesthetics and steroids during her hospital stay, and with carbamazepine and amitriptyline (a tricyclic antidepressant) after her discharge from the hospital. Steroids and tricyclic antidepressants have been suggested to play a role in prompt recovery from spinal cord lesions and the activation of descending inhibitory pathways.

According to our patient's clinical course and MRI diagnosis, we believe that inhibition of the descending inhibitory pathways resulting from a lesion of the spinal cord may have been the possible etiology of her transient lower limb pain and allodynia. Further studies are needed to clarify the role of the descending inhibitory pathways in transient lower limb pain.

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