LUMBAR sympathetic block (LSB) is an effective therapy for reflex sympathetic dystrophy of the lower extremity by blocking sympathetic efferent impulses to the affected limb.1 Complications of LSB are rare2 and usually minor, such as backache and stiffness,1 and are due to needle trauma to the paraspinal muscles. Subarachnoid injection is a recognized complication of LSB.2 In the following case report we describe a patient in whom postdural puncture headache (PDPH) occurred following LSB performed with the assistance of fluoroscopic guidance. Fluoroscopic confirmation of needle placement helped clarify that PDPH after LSB may be due to dural sleeve puncture outside the spinal canal, rather than intraspinal dural puncture.

Case Report

A 55-yr-old, 100-kg man with a history of left lower extremity pain after trauma to the left thigh 8 yr earlier was referred to our pain clinic. In the past the patient was suspected of having reflex sympathetic dystrophy and treated with trigger point injections, nonsteroidal anti-inflammatory agents, transcutaneous electrical stimulation therapy, and LSB. The LSB relieved his pain temporarily, and subsequently the patient underwent an operative lumbar sympathectomy 6 yr prior to referral to our clinic.

Documenting complete sympathetic denervation after a lumbar sympathectomy is important to determine whether all the sympathetic fibers to the extremity have been interrupted.3 Therefore, a skin conductance response4 test was performed, which indicated that this patient still had sympathetic activity persisting in the left leg. The patient still described chronic burning pain, allodynia, and intermittent cold sensations to the entire leg. This combined with the history of relief of pain with LSB and the fact that he still had sympathetic activity to the leg led us to recommend a diagnostic LSB.

An LSB was performed under fluoroscopic guidance to identify the metallic surgical clips marking the cephalad and caudal extent of the previous surgical sympathectomy and to facilitate accurate needle placement in reference to these clips. The patient was placed in the right lateral decubitus, and the technique of needle insertion was based on that of Mandl.2

A 22-G 1.5-cm spinal needle (Havel's, Cincinnati, OH) was inserted 8 cm to the left of midline under fluoroscopic guidance at the L2 level. During the insertion, paresthesia was elicited to the middle of the patient's left thigh. At the same time the anesthesiologist performing the block noted the sensation that the needle had contacted periosteum. Fluoroscopic image on lateral view showed the needle to be at the anterior wall of the intervertebral foramen at the L1-2 level. It was necessary to traverse this area to get to the superior aspect of the L2 vertebral body cephalad to the sympathectomy clips. The needle was withdrawn and repositioned to the anterolateral aspect of the superior border of L2 cephalad to the sympathectomy clips. Correct needle placement was verified with anteroposterior and lateral fluoroscopic images and confirmed with 0.2 ml of radiographic contrast (iohexolate meglumine 60%, Conray, Mallinckrodt Medical, St. Louis, MO). A total of 1.2 ml 0.5% bupivacaine in one-third strength Conray was injected with intermittent negative aspiration. Fluoroscopic imaging showed good spread of the solution along the anterior border of the vertebral bodies from the inferior aspect of T1-2 to the surgical clips at L2. The patient stated he had complete relief of the leg pain at the end of the procedure.

Three days later the patient called complaining of posterior neck pain and occipital headache that had begun the day after the LSB was performed. The pain increased with standing and was relieved by assuming the supine position. The patient also complained of nausea, but denied vomiting, fever, or chills. The positional headache persisted after another day of bed rest and oral hydration including liquids containing caffeine. At this point the patient complained of headache pain with a score of 10 (on a visual analog scale of 0–10) when standing and 4 when supine. Using the technique of Jarvis,4 a total of 500 mg caffeine sodium benzoate in 1 L lactated Ringer's solution was given over 1 h. The pain was rated at a score of 1 while supine 40 min after the infusion began. Another 1 L lactated Ringer's solution was infused over the next hour, during which time the headache resolved completely both supine and upright. The patient continued to have complete resolution of his headache during his normal activity through the next week and at follow-up 7 months later.

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CASE REPORTS

Discussion

With LSB, complications tend to be minor, but reported complications include: puncture of a major vessel or renal pelvis, subarachnoid injection, genitofemoral neuralgia, perforation of an intervertebral disc, infection, and chronic back pain. Dural puncture during paravertebral somatic nerve block has been reported, presumably due to needle entrance into dural root sleeves. It has been suggested that puncture of these elongated dural sleeves may cause PDPH secondary to cerebrospinal fluid leakage. These dural root sleeves have been identified to extend beyond the intervertebral foramen and to be present on lumbar radioculography.

Postdural puncture headache is a frequent complication of spinal anesthesia. One theory of the cause of PDPH relates to the loss of cerebrospinal fluid through the dural puncture. Belief in this theory supports many treatments of PDPH: epidural blood patch, epidural saline infusion, and epidural dextran 40 infusion. These treatments all involve placement of a substance at the site of dural puncture that can physically close the dural flap preventing further cerebrospinal fluid leakage, or create a physical pressure increase in the epidural space due to the volume of fluid infused. This pressure increase may be transmitted to the cranial cavity relieving the caudal displacement of the brain and tension on its anchoring structures.

Secher suggests that the primary factor involved in a PDPH is an intracranial vascular component. The finding that intravenous caffeine sodium benzoate rapidly provides relief in the majority of subjects supports this idea. The vascular changes that occur following loss of cerebrospinal fluid after dural puncture include distention of cerebral vessels, increased cerebral blood flow, and increased brain volume. Caffeine sodium benzoate has been shown to produce cerebral vasodilatation, increased cerebral vascular resistance, and decreased cerebral blood flow.

This patient's PDPH was treated with 2 L lactated Ringer's solution and 500 mg caffeine sodium benzoate. The PDPH was relieved with this combination, but it is not known whether the rehydration with the lactated Ringer's solution alone would have relieved the headache pain. If the PDPH was not relieved with the rehydration and caffeine sodium benzoate, the patient may have had relief with an epidural blood patch.

Epidural blood patch has failed in attempted treatment of PDPH after LSB in a previous case report in which the puncture of the dura was presumed to be outside the vertebral canal. The placement of blood into the epidural space with a blood patch may not reach the site of dural puncture with an LSB. The mechanism of action of caffeine sodium benzoate is far removed from the relatively inaccessible dural puncture site. Caffeine sodium benzoate may prove to be the most effective therapy for treating PDPH after LSB with a 22-G needle.

This is the first report of PDPH after LSB using fluoroscopic imaging. At no time during the procedure did fluoroscopic imaging show the needle to be within the vertebral canal. This is consistent with the fact that puncture of the dura outside the vertebral canal (i.e., dural sleeve) may contribute to the formation of PDPH. Fluoroscopic imaging showed the needle to be located at a point where dural sleeves are known to exist. During LSB, when the needle tip is located in the dural sleeve area, either fluoroscopic imaging or recognition that the needle depth is too superficial (in cases done without fluoroscopy) should prevent inadvertent local anesthetic injection in most instances. Therefore, it is important that practitioners recognize that PDPH may develop after LSB without developing signs of subarachnoid or subdural anesthesia.

References

Inflammatory Cutaneous Reactions to Epidural Catheters


THE authors' Acute Pain Service, at a university medical center in Seattle, has treated more than 5,000 postoperative patients with epidural analgesia. Over a period of 8 weeks, six patients developed a cutaneous inflammatory reaction to nylon epidural catheters.

Case Reports

Case 1

A healthy 66-yr-old woman was admitted for total abdominal hysterectomy and bilateral salpingo-oophorectomy. She gave a history of allergy to rubber, horse serum, adhesive bandages, and several types of tapes.

Before induction of general anesthesia an epidural catheter was inserted at the L3–4 interspace and established using 2% lidocaine with epinephrine (2:100,000) (lidocaine). Following the Acute Pain Service protocol, 5% povidone iodine (Betadine) spray was used prior to catheter placement. Thereafter, tincture of benzoin and a Tegaderm® dressing (10 × 50 cm) were used to secure and cover the catheter.

For postoperative analgesia she received an epidural dose of 2 mg morphine (1 h before the end of surgery) and was subsequently prescribed 2 mg epidural morphine every 6 h as needed. On the second morning after surgery (40 h after epidural catheter insertion) she complained of "soreness" only at the point of entry of the catheter, where there was also a "circle of inflammation and a small amount of swelling." It was decided that the best course of action would be to remove the catheter and extend the course of perioperative antibiotics (cefazolin). Upon removal there was a small amount of serous discharge, but it also became evident that there was an inflammatory "trail" which traced the exact course of the epidural catheter from her lower back, up to and over her shoulder (Fig. 1). The "trail" was about 5–6 mm wide and persistent, albeit faded, until her discharge on postoperative day 6.

On examination, she has at all times had a normal range of back movement and has been neurologically intact.

Case 2

A 58-yr-old woman was admitted for a total abdominal hysterectomy and bilateral salpingo-oophorectomy. Her only relevant medical history was that she was allergic to "sulfa and Betadine." She consented to a combined epidural and general anesthetic. An epidural catheter was inserted in the lumbar region, using chlorhexidine antiseptic. It was covered with Tegaderm® dressing, after a light spray of tincture of benzoin. She was given 2% lidocaine and had a block to T8–9.

For postoperative analgesia she received 3 mg epidural morphine toward the end of surgery and was prescribed 3 mg epidural morphine every 6 h, as required. Apart from some mild nausea she had no problems. The epidural catheter was removed 70 h after surgery. A red line was noted on her back, which was several millimeters wide and traced the course of the catheter (Fig. 2). The rash faded over 1–2 days. Examination of her range of motion and nervous system were normal.

Case 3

A healthy 61-yr-old man was admitted for a radical cystectomy and urinary diversion procedure. His epidural catheter was inserted at the L3–4 interspace following Betadine® antiseptic spray. Tincture of benzoin and a Tegaderm® dressing were applied. He was given 2% lidocaine for peroperative analgesia. Postoperatively 0.0625% bupivacaine with 2 µg/ml fentanyl was administered as an epidural infusion (8 ml/h).