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An Unusual Cause of Delayed Postmyelogram Headache

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Headache, with a reported incidence of from 7.5 to 77%, is a common complication of diagnostic myelography. 1 The incidence of headache is influenced by the type and volume of contrast,2 site of injection,3 size of needle, and patient positioning after the procedure.4 The frequency of postmyelogram headache is reportedly greater than that of post-dural puncture headaches (PDPH) after spinal anesthesia. This increased incidence may be attributable to factors other than cerebrospinal fluid (CSF) leak at the dural puncture site, such as irritation of the meninges by the contrast dye, withdrawal of CSF, and patient positioning during the procedure.5 Onset of PDPH usually occurs within 36 h of the procedure, particularly in ambulatory patients, and only rarely after 5 days.6 Most postmyelogram headaches are self-limited, but they have been reported to last for up to 19 months after the original puncture.7 We report an unusual cause of a delayed PDPH, occurring 7 days after myelography, and its resolution with epidural blood patch.

CASE REPORT

A 71-yr-old man presented to his local hospital reporting increasing pain in his chest wall and upper back over the previous 2 months. This had not been relieved by acetaminophen and codeine. He also reported persistent cough, malaise, and weight loss during the previous month. Over the several days prior to admission, he had experienced difficulty ambulating and reduced sensation in both legs.

Positive findings on physical examination included increased muscle tone and normal strength in both legs. There was decreased proprioception bilaterally but normal perception of pin prick and light touch. His testes were atrophic and a rock-hard left prostatic mass was found adherent to the left lateral rectal wall. He was transferred to our institution, where a prostatic needle biopsy was performed, revealing adenocarcinoma, and treatment with flutamide was started.

Due to his skeletal pain and neurologic findings a tentative diagnosis of spinal metastases was made, and myelography was carried out on the day after admission. Using a 22-G spinal needle, 8 ml iohexol (Omnipaque[®]) was instilled at the L2-L3 level and flowed into the high thoracic region, where a complete obstruction to flow occurred at the level of the second thoracic vertebra. Sclerosis and destruction of this

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vertebral body and of both adjacent second ribs were seen. A second puncture was carried out with a 22-G needle at C1-C2, and 5 ml of contrast was instilled; its caudal flow was blocked at the T2 level. Computerized tomography scanning revealed a circumferential epidural soft tissue mass at this level, blocking the flow of contrast. Also seen was a mass indenting the dura at the L5 level.

On day 3 postadmission, dexamethasone was prescribed. By the next day, subjective improvement of proprioception in his legs was reported. On this day the first of a course of four daily radiotherapy sessions was performed on his upper thoracic and lower lumbar spine. Beginning on day 5 he was positioned sitting upright in a bedside chair several times a day. On day 7 he was noted to have marked neurologic improvement and was ambulating with assistance. On day 9 he complained of a severe bifrontal headache, worse with upright posture and relieved when supine. He denied ever having had a similar headache prior to that day.

On day 12, the anesthesia service was consulted to assist in diagnosis and treatment of the persistent headache. Although it seemed unusual for PDPH to have begun 7 days postmyelography despite the patient's upright position and his ambulation, the patient's description of the headache was characteristic of PDPH. With patient consent an L2–L3 epidural blood patch was undertaken using 15 ml autologous blood. He was kept supine for 4 h, and then he ambulated. The headache did not recur, and the patient was discharged home on day 15 postadmission.

DISCUSSION

We have reported the case of a typical postmyelogram headache occurring with an unusually delayed onset. The patient had two dural puncture sites, but a number of factors point to a dural tear at the lumbar level as the cause of his headache. The incidence of postmyelogram headache after C1-C2 puncture has been shown to be approximately one-half that after the lumbar approach, and its onset is usually sooner.8 This supports the theory that the pressure gradient between the subarachnoid and epidural spaces determines the amount of CSF leakage; the subarachnoid pressure is higher at the lumbosacral than at the cervical level with the patient in an upright position. This patient had an epidural tumor obstructing the flow of CSF and thereby preventing it from reaching the lumbar dural tear but not the cervical site. The headache experienced by this patient was therefore less likely to have been caused by CSF leak at the C1-C2 level. It is probable that the systemic corticosteroid treatment and spinal radiotherapy reduced the tumor mass and allowed the restoration of CSF flow; this was confirmed not radiologically but indirectly, by neurologic improvement. Thus, the onset of a side effect actually signified a positive therapeutic effect.

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There is abundant evidence implicating CSF leak at the dural puncture site as the cause of PDPH. It generally is believed that this CSF leak causes a decrease in CSF pressure and subsequent traction on pain-sensitive intracranial structures. Supporting this hypothesis are studies showing an increase in the incidence of PDPH with increasing needle size, the presence of decreased CSF pressure in patients with PDPH, and direct visualization of the CSF leak at subsequent laminectomy.9 An epidural blood patch is believed to act by forming a gelatinous plug over the dural leak and promoting fibroblast activity and collagen deposition over the clot to permanently seal the dural tear.7 This mechanism, along with the proven effectiveness of epidural blood patch, 10 provides additional evidence for CSF leak as the cause for prolonged PDPH.

We have presented a case of a postmyelogram headache with an unusually delayed onset. We propose that obstruction of CSF flow, and its subsequent restoration with treatment, led to delayed CSF leak and headache. Epidural blood patch yielded complete relief. This proposed mechanism further substantiates CSF leak as the cause of PDPH and the effectiveness of blood patch in this situation.

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External Compression of the Abdominal Aorta Reversing Tetralogy of Fallot Cyanotic Crisis

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Cyanotic spells are not rare during anesthesia in children with Tetralogy of Fallot (TOF). Classically, the treatment of this life-threatening crisis is pharmacologic. 1-3 During open-chest operations, surgeons may briefly clamp the thoracic aorta to reverse hypoxic spells. 4 We describe how a similar maneuver consisting of manual external compression of the abdominal aorta quickly interrupted severe cyanotic episodes.

Key words: Anesthesia, pediatric, cardiac: tetralogy of Fallot. Complications: cyanosis. Measurement technique: oximetry. Monitoring.

CASE REPORTS

Case 1. This 6.9-kg, 6-month-old boy with TOF experienced profound hypoxic episodes with increasing frequency. Total repair was scheduled. He was given a bottle of water with sugar and 1 mg oral propranolol at 3:00 AM. At 7:00 AM, 1 mg morphine hydrochloride and 0.2 mg atropine sulfate were injected intramuscularly. The child was calm on arrival to the operating room. Peripheral hemoglobin oxygen saturation (Spos) noted from a pulse oximeter probe (Oscar, Datex, Finland) placed at the left big toe was 98%. Anesthesia was induced smoothly with up to 2.5% halothane in oxygen. A saphenous vein was cannulated and nasotracheal intubation performed. A sidestream capnograph was placed using a special pediatric connector (Oscar, Datex). Halothane was discontinued, and 1 mg diazepam, 50 μ g fentanyl, 1 mg pancuronium bromide, and 20 ml of a 5% albumin solution then were given intravenously (iv). Mechanical ventilation was begun. A femoral artery and the right internal jugular vein were cannulated. Seventy five μg fentanyl was added. Central venous pressure was 10 mmHg, heart rate 120 beats per min, and Spo. > 96%, and blood pressure had decreased slightly, from 92/65 to 85/47 mmHg. Twenty milliliters 5% albumin solution had been given to compensate for blood losses due to percutaneous catheter insertions.

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