

Postdural Puncture Headache with Abdominal Pain and Diarrhea

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Dural puncture or a rent in the dura with prolonged cerebrospinal fluid leakage may cause noninfectious arachnoiditis and may be associated with urinary and fecal incontinence. Visceral dysfunction is common for patients with noninfectious arachnoiditis of the lumbosacral nerve roots after dural puncture. We report a case of postdural puncture headache associated with abdominal pain and

diarrhea. An epidural blood patch was performed, and all symptoms resolved after 5 days. After exclusion of organic and psychological disorders, this treatment might be considered for patients who are experiencing abdominal pain and diarrhea after dural puncture.

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The most common complication of spinal anesthesia (SA) is postdural puncture headache (PDPH) due to cerebrospinal fluid (CSF) leak (1). PDPH may present as a postural frontal, frontotemporal, or occipital headache that is worsened by erect posture and improved by recumbence. Continuous loss of CSF through the dural puncture site causes a decrease in cerebral pressure. In the upright position, the decreased pressure fosters stretching of the nerves and vessels, which produces the symptoms of PDPH. Atypical presentations of postdural puncture symptoms have been reported (2,3); however, the actual cause of symptoms could not be established conclusively. Recent evidence indicates that arachnoiditis or a local inflammatory process might be the etiology for these symptoms (4–6). We report a case of prolonged PDPH complicated by abdominal pain and diarrhea treated with an epidural blood patch (EBP), which resulted in complete relief of all symptoms.

Case Report

A 20-yr-old man (75 kg; 182 cm) presented for arthroscopy of the right knee. Medical and surgical histories were unremarkable. Specifically, he had no history of gastrointestinal problems, headache, or psychiatric illness. SA with a 25-gauge Quincke needle was performed after three attempts at the L3-4 vertebral interspace. The patient did not report pain on needle placement or injection; no blood in the CSF was noted. Tetracaine 12 mg in 10% glucose (2.4 mL) and epinephrine 0.1 mg were injected, resulting in a T10 sensory level. The surgery lasted 75 min with the patient in the supine position.

Six hours after dural puncture, the patient experienced the gradual onset of a dull, throbbing headache that was completely relieved in the supine position but was exacerbated immediately when he was sitting or standing. He was advised to remain in bed and received fluid administration (3 L/d by mouth) and oral acetaminophen (1300 mg/d by mouth). The next day, he complained of persistent headache associated with neck pain, nausea, and vomiting. EBP was suggested to relieve the patient's symptoms, but he refused. The symptoms persisted over the next several days. On the 12th day after SA, he experienced a sudden and acute onset of abdominal pain and three to four episodes of diarrhea with small-volume, loose stools, which were accompanied by mucus discharge, bloating, fecal urgency, and incomplete evacuation, especially after meals. Three days later, colonoscopy was performed because of worsening symptoms of abdominal pain and diarrhea. No mucosal or organic disease was found, and the diagnoses of functional abdominal pain syndrome, functional diarrhea, and unspecific functional bowel disorder were considered. The duration of symptoms associated with abdominal pain and diarrhea in this patient was not >12 wk, a requirement for diagnosis of irritable bowel syndrome (IBS). However, IBS could not be

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completely excluded. Loperamide and kaolin (kaolin and pectin) were prescribed, with minimal benefit. Psychosocial factors were excluded after consulting with psychologists. An EBP with 12 mL of autologous blood was administered at the L3-4 vertebral interspace on the 31st day after SA. One hour after EBP, the patient noted complete relief of headache and neck stiffness. The abdominal pain and diarrhea dramatically improved on the first day and were completely relieved on day 5 after EBP without any oral antidiarrheal drugs.

Discussion

We report a case of a prolonged PDPH for 31 days associated with abdominal pain and diarrhea for 20 days. A diagnostic and therapeutic EBP was performed, and all symptoms resolved, including headache, neck pain, nausea, vomiting, abdominal pain, and diarrhea.

The arachnoid layer contains small vessels that appear to be immunologically reactive and are more susceptible to an inflammatory and edematous reaction that leads to fibroblast proliferation. Hannerz et al. (7) reported more magnetic resonance imaging (MRI) enhancement of meningeal structures during postlumbar puncture headache compared with an MRI performed when the symptoms had disappeared. Moreover, Aldrete (4) suggested that an incidental dural puncture or rent in the dura with prolonged CSF leakage may cause noninfectious arachnoiditis. In addition, the adjacent neural tissue—such as spinal cord, ganglia, nerve roots, brain, or cranial nerves—may also be involved. Therefore, arachnoiditis is not only a local anatomical phenomenon. Aberrant nociceptive stimuli may cause severe burning pain, stabbing and throbbing sensations, and dysesthesia. In cases of lumbosacral nerve root involvement, asymmetric perineal numbness, vesicular, rectal, or sexual dysfunction is common (4). Therefore, our patient probably had noninfectious arachnoiditis after SA, although we did not perform an MRI examination.

Al-Chaer and Traub (8) stated that in the presence of local inflammation, visceral afferents become sensitized and respond to previously innocuous natural stimuli. Additionally, inflammation can cause pain by sensitizing low- and high-threshold receptors, as well as previously “silent” or unresponsive receptors. Inflammatory mediators released locally decrease the receptor firing threshold and, by peripheral sensitization, augment and perpetuate the transmission of noxious stimuli. Sarkar et al. (9) demonstrated a reduction in the pain threshold to electrical stimulation of the non-acid-exposed proximal esophagus and a concurrent reduction in the latency of the esophageal evoked potentials from this region after a 30-minute infusion of 0.15 M HCl acid into healthy human distal esophagus. They concluded that the hyperexcitability of

spinal cord sensory neurons might induce visceral hypersensitivity in humans and generate the symptoms of functional gastrointestinal disorders. Moreover, Traub (10) found that the visceral information carried by the pelvic nerves converges onto spinal neurons in the lumbosacral segments of the cord with those carried by the splanchnic nerves on thoracolumbar segments. In addition, Ji and Traub (11) stated that neuroplasticity may originate from the response of *N*-methyl-*D*-aspartate receptors to signaling from innocuous colonic stimuli, leading to excessive activity at these receptors which could produce central sensitization and result in visceral hyperalgesia. Taken together, the motility disturbances of bowel function might be due to visceral hyperalgesia by both peripheral and central mechanisms (8). Therefore, many IBS patients exhibit a number of extraintestinal pain symptoms, such as back pain, migraine headaches, heartburn, dyspareunia, and muscle pain, that are consistent with visceral, cutaneous, and central hyperalgesic mechanisms (12). Recently, Hyland and Butterworth (13) reported a patient with a varicella-zoster virus infection who presented with severe abdominal pain that may have resulted from varicella-zoster virus reactivation and inflammation of the dorsal root ganglion. The abdominal pain resolved over three days after the onset of IV acyclovir treatment. Moreover, Aldrete (4) stated that visceral dysfunction was common for patients with noninfectious arachnoiditis of the lumbosacral nerve roots after dural puncture. Headache, neck pain, and abdominal pain associated with diarrhea were also present in our patient; thus, we postulate that PDPH caused visceral hyperalgesia that resulted from noninfectious arachnoiditis in the lumbosacral plexus.

An EBP might treat arachnoiditis by increasing epidural and subarachnoid pressure and decreasing meningeal inflammation. An EBP acts as a gelatinous tamponade that first prevents CSF leakage and then allows the normal reparative healing of the fibroblast cells that form a collagen seal on the tear in the dura. In animal studies, Di Giovanni et al. (14) showed that complete healing of a dural tear took approximately one week. Luijckx and De Jaegere (15) showed that brain MRI revealed meningeal thickening and enhancement in a patient who had spontaneous intracranial hypotension. This finding disappeared after increasing CSF pressure and diminishing cerebral vasodilation by an EBP three weeks later. These findings demonstrated that it took time for the diffuse meningeal enhancement and, presumably, inflammation to resolve after EBP and suggest that the resolution of meningeal inflammation played a role in recovery after PDPH.

With performance of the EBP, the tamponade effect stops leakage of CSF and relieves traction on brain structures. Descending nociceptive transmission from

the anterior cingulate cortex (ACC) and the prefrontal cortex (PFC) may induce functional bowel disorders (16). CSF leakage may cause downward brain traction, which may induce ACC and PFC firing, and this may increase the perception of visceral pain (17). Thus, the tamponade effect of EBP may stop pathologic nociceptive transmission from the ACC and PFC.

In conclusion, after exclusion of organic and psychological disorders, an EBP may be considered for the treatment of patients experiencing abdominal pain and diarrhea after SA or recent dural puncture.

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