Late recurrence of post-dural puncture headache
Post-dural ponksiyon baş ağrısının geç dönem rekürensi

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Summary
The term post-dural puncture headache (PDPH) refers to a common complication that occurs after accidental dural puncture. One of the diagnostic symptoms of PDPH is a postural headache, which worsens dramatically while sitting or standing and is relieved mostly by lying down. This symptom is caused by a cerebrospinal fluid (CSF) leak, leading to decreased CSF pressure or low CSF volume, which provokes a shift of intracranial contents and traction on pain-sensitive structures in the upright position. PDPH is commonly a self-limited condition and remits spontaneously within 2 weeks, or becomes less severe after surgical intervention to seal the leak with autologous epidural blood patch (EBP). Although recurrence of spontaneous intracranial hypotension following an EBP is not rare, spontaneously late recurrence of PDPH has been rarely reported. The purpose of this paper is to discuss this case with late recurrence of PDPH after 10 months following EBP.

Keywords: Epidural blood patch; post-dural puncture headache; recurrence.

Introduction
Intracranial hypotension is caused by low cerebrospinal fluid (CSF) pressure and characterized by postural headache. Post-dural puncture headache (PDPH) occurs in 10–40% of patients who have a lumbar puncture (LP). \[1,2\] PDPH generally occurs within 5 days of LP, caused by CSF leakage through the dural puncture. \[3,4\] PDPH usually starts within 24 h after LP and disappears in 50–90% of patients after treatment with an epidural blood patch (EBP). \[5\]

PDPH is usually a benign condition; however, it can have serious morbidities, such as cranial nerve palsy, seizures, and subdural hematoma. \[6\] Recurrence of spontaneous intracranial hypotension (SIH) is not rare. \[7,8\] On the other hand, spontaneous late recurrence of PDPH is unexpected. The following case presents a spontaneous late recurrence of PDPH after EBP, which is a rare condition.

Case Report
A 33-year-old woman with a 10-year history of a severe headache was admitted to a neurology clinic in another hospital. At first, the headache was diagnosed as migraine and it was observed that headache attacks occurred twice a week. The patient had been suffering from a chronic tension-type headache for 5 years, and she had been receiving treatment (methylphenidate) for attention deficit and hyperactivity disorder.
The brain magnetic resonance image (MRI) revealed an increased intensity in the T2-weighted images of both frontal areas. The LP was performed in that hospital. The CSF was normal in the microbiology and biochemistry analyses; however, the CSF pressure was not reported. The headache became more painful 4 days after LP. The patient’s pain increased when she got into an upright position, whereas it was resolved in 10 min following bed rest. It was also reported that Valsalva maneuvers and head movements triggered the headache. A brain MRI with contrast revealed diffuse dural enhancement, herniation of the cerebellar tonsils, and increased pituitary gland size (Fig. 1). There were non-specific T2 hyperintense lesions in the bilateral frontal areas. The cervical, thoracic, and lumbar MRI were reported as normal; the findings led us to suspect PDPH.

The patient’s postural headache was not relieved after 2 days of conservative treatment (bed rest, sufficient oral fluid intake, and caffeine). The EBP was performed. The patient’s venous blood was collected from the antecubital vein under aseptic conditions, and 15 mL was gently injected into the L3–L4 space. The patient lay down in the Trendelenburg position for 4 h after the procedure. When the patient returned to an upright position 48 h later, her postural headache was resolved.

One month later, she was reexamined. The postural headache was resolved, but she was still suffering from a chronic tension-type headache. The brain MRI with contrast was normal (Fig. 2). The headache intensity decreased within 2 months.

Ten months later after the initial EBP, the patient was admitted after suffering postural headache for 15 days. The brain MRI was reperformed and diffuse dural enhancement was detected (Fig. 3a). She was rehospitalized and computed tomography (CT) myelography was performed. There was a CSF leakage in the upper lumbar level (Fig. 3b). The LP was performed. The contrast-enhanced T1W axial image shows resolution of dural enhancement (arrows).
formed, and the CSF opening pressure at the lateral decubitus position was 60 mm H2O. The microbiology and biochemistry were normal. The second EBP was performed and the patient’s postural headache was relieved immediately. Three weeks later, she was free of postural pain. The diffuse dural enhancement was regressed in the brain MRI (Fig. 3c).

Discussion

The risk of a PDPH after accidental dural puncture is approximately 50%. Most PDPH cases can be resolved within a week with conservative management. However, in a small group of cases, symptoms may persist for weeks, months, or even years. Rarely, it can become chronic, with the longest reported headache after LP lasting 5 years. Although EBP has an apparent benefit, there have been a few reports of late spontaneous recurrence of PDPH after EBP. A 31-year-old woman with possible lupus vasculitis had a recurrence 5 weeks after treatment. Three patients developed PDPH following dural punctures after 3, 9, and 50 years, and the authors concluded: “Once a PDPH patient always a PDPH patient.” Wilton et al. reported a patient who had a 19-month persistent headache after the procedure.

The patient described here was diagnosed with PDPH and her postural headache was not relieved with conservative management within 2 days. After EBP had been performed, her headache was relieved. One month later, despite a continued chronic tension-type headache, no postural headache was observed and the brain MRI was normal.

She was readmitted with a severe postural headache 10 months later. She had no history of obvious or trivial trauma except from coughing. The MRI detected diffuse dural enhancement. CT myelography was applied and upper lumbar level CSF leakage was revealed. EBP was reperformed and her postural headache was relieved. Three weeks later, she was reexamined and she was free of postural pain.

Contributory risk factors for PDPH recurrence have been speculated on. It is not known why some patients develop SIH after LP and EBP. The existence of connective tissue disorders, joint hypermobility, ectatic dural sacs, multiple meningeal diverticula, and dilated nerve root sleeves has been thought to contribute to dural weakness in patients with SIH. Although the CSF leakage is repaired and shows a marked improvement with EBP, the connective tissue matrix can be vulnerable. It can be speculated that a very low grade of CSF leak may continue, and mild or minimally symptomatic CSF hypovolemia may be maintained. Trivial trauma, such as coughing, vomiting, sneezing, pulling, pushing, or heavy lifting, can cause a tear in the previously injured lumbar dural structure.

Conclusion

In this case, late recurrence of PDPH was encountered without known risk factors. Appropriate follow-up reveals a number of patients experiencing incomplete relief, failure, or recurrence of symptoms. The literature concerning late recurrence of PDPH has generally been insufficient. It is important that these issues are resolved in the future through well-designed clinical investigations.

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