Introduction

Cranial nerve dysfunction and post-dural puncture headache (PDPH) is an unfortunate condition that can occur after spinal anesthesia or accidental dural puncture. They may also take many years to occur after epidural anesthesia. PDPH is aggravated by standing or sitting because of cerebrospinal fluid (CSF) leakage through the dural defect.

The side effects of the Tuohy needle used for epidural anesthesia are due to prolonged CSF leakage and delayed closure of a dural defect leading to intracranial hypotension. The decrease in CSF volume and pressure leads to the sliding of the intracranial structures and traction on pain-sensitive contents in the brain [1-3]. Cases of cranial nerve dysfunction after a dural puncture were reported in the literature previously. The most affected nerve is nerves abducens. At such a time, the epidural blood patch is the standard therapy [3]. The author reports a case of a sixth and twelfth cranial nerve recurrent palsies at 3 and 4 years following combined spinal-epidural anesthesia for cesarean section.

Case Report

A 36-year-old multigravida (G2 P1) at full-term pregnancy requested a cesarean section (C/S) delivery. The patient had her first delivery via C/S under spinal anesthesia. The patient did not undergo any other lumbar spinal procedure before. The patient does not have any known neurological condition (e.g. multiple sclerosis, etc.). An 18-gauge Tuohy needle was inserted at the first attempt at the L3-L4 interspace via a midline approach.

The epidural space was located using the loss of resistance to saline technique. There was no dural puncture with the Tuohy needle. By adopting the ‘needle-through-needle’ technique, a 27-gauge Whitacre needle was used for the injection of intrathecal local anesthetic.
On the first postoperative day, she reported symptoms of a headache at the frontal and occipital sides, worsening at standing or sitting positions. The treatment of post-dural puncture headache and palliative measures consists of bed rest, hydration, and intake of analgesics (every 6 hours, up to a maximum of 2 g a day [acetaminophen 250 mg + propyphenazone 150 mg + caffeine]). Seven days later, she had no complaints and was discharged.

In the postpartum process, she had headaches at 3-4-month intervals and had no improvements by analgesics. She had no visual or auditory symptoms. In this process, since the patient was not in clinical follow-up, no investigation was conducted for the recurrent headache. Two years following the C/S, she reported a symptom of double vision. The symptom also carried on for 3 months and worsened day by day. On the physical examination, she exhibited an inability to move the right eye into the lateral gaze. A diagnosis of *abducens* nerve palsy caused by accidental dural puncture was made.

*Abducens* nerve palsy in this patient recovered spontaneously with conservative therapy.

Three and a half years following the C/S, she had difficulties in speaking. Four years after C/S, she noticed a shape defect on the right side of her tongue. At first, she was under the care of the Department of Otolaryngology-Head and Neck Surgery while in consultation with the department of neurology. No pathology was determined by MRI and nerve conduction study.

Four years following the C/S, the complaint of double vision recurred, and the patient was taken under the care of the department of neurology in another medical center. She was admitted for a detailed investigation. During the neurologic examination, her speech had minimal dysarthria. The right half of her tongue was atrophic. The diagnosis was the 12-nerve palsy. She had no other meningeal or focal neurologic symptoms.

Cranial MR was normal. In the MR angiography, there was widespread congestion in the cranial venous structures. Carotid vertebrobasilar Doppler USG was normal. The diagnosis was intracranial hypotension. After the MR angiography, MR myelography was also conducted. A doubtful contrast fixation or leak was seen in the lumbar 3-4 and 4-5 right lateral and lumbar 4-5 right inferior neural foramens. An LP was conducted, and CSF pressure was measured as 93mmH2O, which was normal. After myelography, the patient stated that the complaints became more intense.

The headache got better by lying down. The epidural blood patch was decided with the aim of a therapeutic blockade. After obtaining an informed consent from the patient regarding both the procedure and the report, an epidural blood patch (EBP) with her blood (15-20 mL) was administered to the patient. EBP was conducted by an experienced anesthetist under aseptic conditions. After the cannula was removed, the routine vital monitoring was maintained while the patient was laying on her back for 2 hours. In the sitting position and two hours after the procedure, the headache was clearly regressed. Then, a spinal MR was conducted again. The areas where there were leakages were plastered with the epidural blood. In the following days, she had no headache. The patient was discharged three days later. During the follow-up visits in the subsequent two years, the patient did not experience pain and her neurological examination was normal, except for the *Hypoglossus* nerve palsy.

**Discussion**

The author reports a case with a recurrent sixth and twelfth cranial nerve palsy 3 and 4 years after an unintentional dural puncture. The mechanisms behind PDPH are still unknown. It is believed that PDPH is an effect of sliding of the brain downward and traction on pain-sensitive structures because of the leakage of CSF [1, 3, 4].

The size of the dural puncture and time of brainstem compression due to CSF leakage vary among patients and are dependent on the gauge of the needle used [4].

The incidence of accidental dural puncture with the Tuohy epidural needle is 0.04-6%. PDPH is a complication of epidural analgesia with an incidence of 1-3%. More than 80% of these patients develop PDPH [5]. Loss of resistance to air confers a higher risk of dural puncture compared to a loss of resistance to fluid [6].

In one report, a patient developed a post-dural puncture headache and diplopia after an administration of the sequential combined spinal-epidural labor analgesia via a 25-gauge Sprotte spinal needle and a 16-gauge Tuohy epidural needle [7]. Another report described a case of headache and abducens palsy after an administration of spinal anesthesia via a 25-gauge pencil-point spinal needle [3]. In another report, it was described that a case of headache and vocal cord paralysis occurred after an administration of spinal anesthesia via a 25-gauge Quincke needle [8].

Although the success rate for relieving PDPH by EBP in many studies range from 56% to 98 % [3, 9], it does not reliably reverse the *abducens* nerve palsy because neural demyelination occurs beforehand. Early EBP within 24 hours of ocular symptoms was beneficial in restoring CSF pressure with partial resolution of diplopia, other case reports describe the failure of *abducens* nerve palsy to respond to an EBP even when the patch is performed early.

Because *abducens* nerve palsy rarely develops before the 4th day after a dural puncture (although it can occur as early as the 1st day after a dural puncture), or after the resolution of a headache, several researchers suggested that conservative treatment for post-dural puncture headache should be abandoned after 4 days in favor of an EBP to prevent the development of *abducens* nerve palsy [3]. For the patient of this report, it did not reliably reverse the hypoglossal nerve palsy because neural demyelination and muscular atrophy occurred.

Despite the high incidence of headache as a result of dural puncture with a Tuohy needle, the anesthetist needs to think of a different diagnosis, such as intracranial hematoma [10], intracranial tumors [11], pituitary apoplexy [12], cerebral venous thrombosis [13], migraine, chemical or infective meningitis [14] and nonspecific headache.

Although more rarely, cranial nerve dysfunction after dural puncture was also reported. Usually, the oculomotor, trochlear, facial, vestibulocochlear, trigeminal, hypoglossal [15], vagus [16], or abducens nerves can be affected. The most affected nerve is nerves *abducens* (3). Palsy is unilateral in 80% of cases. Magnetic resonance imaging may reveal signs of intracranial hypotension, including meningeal...
enhancement, subdural effusions, engorgement of venous sinuses, and downward brain displacement.

These findings are consistent with intracranial hypotension. However, they are not specific to abducens nerve palsy [3,17]. In the present patient’s MR angiography, there was widespread congestion in the cranial venous structures while in the contrast-enhanced spinal MR, a doubtful contrast fixation or leak was seen in the lumbar 3-4 and 4-5 right lateral and lumbar 4-5 right inferior neural foramen. The areas where there were leakages were plastered with the epidural blood patch.

Vasculopathy (29%), tumor (16%), multiple sclerosis (12%), inflammation (8%), and trauma (6%) are the most common causes of the noniatrogenic abducens nerve palsy [18].

Headache almost always precedes the development of ocular changes. Abducens abnormalities occurred in 2.6% of the patients with headache. Abducens nerve palsy remains a diagnosis of exclusion that requires consideration of other potential neurologic and ophthalmic abnormalities, even when it is associated with dural puncture. If the abducens nerve palsy is an isolated neurologic deficit that occurs within 3 weeks of dural puncture and is preceded by a spinal headache, it is likely a consequence of dural puncture [3]. Unlike this case, the recurrent sixth nerve palsy occurs 3 and 4 years after the post-dural puncture.

In a review that included five patients, the abducens nerve palsy ranged from 1 to 12 days and the duration of complete recovery spanned from 2 weeks to 6 months. Thus, it was believed that the loss of CSF caused the transient hearing loss [19], abducens nerve palsy, dural venous sinus thrombosis [20], and subdural hematomas [21,22].

However, there are occasions when blood patches appear to be effective in treating the headache. Once the diagnosis of PDPH is ascertained, we suggest the conservative therapy option and an assessment of the progression of the symptoms.

In the present case, an epidural blood patch prevented the occurrence of abducens nerve palsy when performed at the onset of double vision. As it was in this case; six cases, which were reported occurrence of assessment of the progression of the symptoms.


