CASE REPORT
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Acute Subdural Hematoma Following Spinal Cerebrospinal Fluid Drainage in a Patient with Freezing of Gait

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Background Headache is a common complication of lumbar puncture (LP). Although in most cases post-LP headaches are not severe and have a benign course, they can also be a manifestation of a potentially life-threatening complication such as subdural hematoma (SDH).

Case Report We describe a patient in whom a massive SDH developed after LP and cerebrospinal fluid (CSF) drainage, which were performed during the diagnostic evaluation of freezing of gait.

Conclusions SDH should not be excluded from the differential diagnosis of headache following LP, especially when there is a loss of CSF. **J Clin Neurol 2009;5:95-96**

Key Words freezing of gait, subdural hematoma, lumbar puncture, cerebrospinal fluid drainage, headache.

Introduction

In most cases headache following lumbar puncture (LP) is not severe and has a benign course,[1] but it can also be a manifestation of a potentially life-threatening complication such as subdural hematoma (SDH). We present herein a case of massive SDH after LP and cerebrospinal fluid (CSF) drainage.

Case Report

A 57-year-old man was admitted to our hospital with freezing of gait (FOG) and bradykinesia. He had suffered three minor ischemic strokes over the past 15 years, from which he had completely recovered. He gradually started to develop FOG and bradykinesia 6 months before admission. Amantadine was administered following the diagnosis of Parkinson's disease, but the patient did not improve. The FOG became slightly aggravated several days prior to admission. Neurological examination on admission revealed generalized bradykinesia and FOG. There were no signs of dementia and no urinary incontinence. MRI revealed multiple ischemic changes in the periventricular white matter and bilateral basal ganglia, as well as diffuse cerebral atrophy with ventriculomegaly (Fig. 1A). Levodopa was administered following the diagnosis of vascular parkinsonism, but did not improve the condition of the patient.

LP and CSF drainage were performed to rule out normal-pressure hydrocephalus (NPH). The opening pressure was 13.5 cmH_2O, and 30 mL of CSF was drained. The patient's gait did not improve, and LP and CSF drainage were again performed 3 days later. The opening pressure at that time was 7.7 cmH_2O, and a further 30 mL of CSF was drained. After the second drainage, the patient's gait improved slightly, and he was pleased with the result. However, because the improvement was not marked, ventriculoperitoneal shunting was not performed and he was discharged.

Five months after discharge, the patient revisited the hospital with aggravated FOG. He was taking aspirin (100 mg daily) and ramipril (5 mg daily). His platelet count and coagulation panel at admission were normal. Levodopa was tried again, but without benefit. Brain computed tomography (CT) revealed slight progression of ventriculomegaly and no signs of intracranial hemorrhage (Fig. 1B). CSF drainage was repeated once more because his gait had improved slightly after CSF drainage on the first admission and aggravation of FOG due to progression of hydrocephalus could not be com-

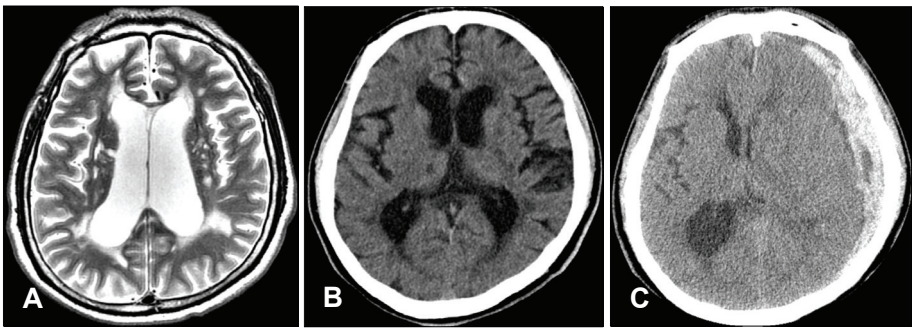

Fig. 1. Brain MRI and CT (A) Brain MRI performed on the first admission revealing multiple ischemic changes in the periventricular white matter, bilateral basal ganglia, and pons, and diffuse cerebral atrophy with ventriculomegaly. (B) A brain CT scan taken before LP during the second admission shows no signs of hemorrhage. (C) A brain CT scan taken 5 days after LP, showing left frontotemporoparietal SDH with midline shifting. LP: lumbar puncture, SDH: subdural hematoma.

pletely ruled out. A 22-gauge Tuohy needle was used in the lateral decubitus position. The opening pressure during this procedure was 7 cmH_2O, and 25 mL of CSF was drained. The patient's gait did not improve after this drainage.

Two days after drainage, he began to develop mild posterior neck pain and an occipital headache accompanied by nausea. The headache was tightening, nonthrobbing, and partially relieved by recumbency. A post-LP headache was suspected. He received conservative treatment but showed no improvement. On the 3rd day post-LP, his headache slightly worsened, but was still mild. No focal neurological abnormality was found on examination. Although his headache was not aggravated on the 4th day, on the 5th day he was found in the bathroom stuporous and with right hemiplegia. An immediate brain CT revealed left frontotemporoparietal SDH with midline shifting (Fig. 1C). Within a few hours, his right pupil became fully dilated and unresponsive to light. An emergency craniotomy and evacuation of the hematoma were performed, and he recovered to his pre-SDH condition.

Discussion

SDH is a rare but well-known complication of LP performed for spinal anesthesia[2] and diagnostic LP performed for suspected meningitis.[3] However, a massive SDH after LP and CSF drainage, which were performed as part of the diagnostic evaluation of our patient with FOG, has not been described previously.

The pathophysiological mechanism of post-LP SDH may involve low intracranial pressure (ICP) as a result of LP and subsequent congestion, dilatation, and tearing of the subdural vein.[4] This could explain the occurrence of SDH in patients with ventriculoperitoneal shunts[4] and in those with spontaneous CSF hypovolemia,[5] where the ICP is chronically low. Considering these findings, it is surprising that most of the reported cases of post-LP SDH were associated with LP performed for spinal anesthesia, in which the loss of CSF is negligible, rather than with diagnostic LP for CSF analysis, in which the loss of CSF is greater and the risk of consequent intracranial hypotension is higher. In LP and CSF drainage for the diagnosis of NPH, as in our patient, the loss of CSF is by substantially greater than for diagnostic LP for CSF analysis. It is conceivable that patients undergoing LP and CSF drainage are at an increased risk of post-LP SDH.

It was initially thought that the headache in our patient was a common post-LP headache because it developed 2 days after LP, which is when post-LP headaches usually occur,[1] and because of the absence of focal or lateralized neurologic abnormality, which would have suggested the presence of an intracranial space-occupying lesion. Furthermore, the patient's headache improved with recumbency, although it did not completely disappear. All of these features contributed to the delay in diagnosis. The preexisting gait disturbance and parkinsonism observed in our patient, which are occasionally initial symptoms of SDH, might have hindered the early detection of an SDH. This case shows that SDH should not be excluded from the differential diagnosis of headache following LP, especially when there has been a loss of CSF.

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