Bilateral Chronic Subdural Hematomas Associated with Intracranial Hypotension

—Case Report—

Hiroshi NAKAJIMA, Tatsuo SAKAI*, Nobuhiko AOKI*,
and Kintomo TAKAKURA

Department of Neurosurgery, Neurological Institute, Tokyo Women's Medical College, Tokyo; *Department of Neurosurgery, Tokyo Metropolitan Ohkubo Hospital, Tokyo

Abstract

A 56-year-old female presented with chronic subdural hematoma due to spontaneous (primary) intracranial hypotension (SIH). She was admitted in a deep coma. Computed tomography showed bilateral chronic subdural hematomas with tight basal cisterns. Percutaneous aspiration of the chronic subdural hematoma with the twist drill revealed negative pressure. The neurological symptoms rapidly improved postoperatively, but the chronic subdural hematoma recurred three times, requiring further aspiration procedures. Patients with SIH may develop subdural hematoma requiring surgical treatment.

Key words: spontaneous intracranial hypotension, chronic subdural hematoma, computed tomography

Introduction

Intracranial hypotension sometimes occurs secondary to a shunt operation for hydrocephalus, cerebrospinal fluid (CSF) leakage due to head trauma, or lumbar puncture. Spontaneous (primary) intracranial hypotension (SIH) is relatively rare, and consists of CSF pressure below 70 mmH₂O associated with headache, nausea, vomiting, and vertigo.^{11,12)} Patients undergoing lumbar puncture often manifest such symptoms, which are relieved by recumbent posture and aggravated by the upright position. SIH is easily curable, since recovery is complete and recurrence is rare.³⁾

We describe a patient with bilateral chronic subdural hematomas thought to be caused by SIH, in whom computed tomography (CT) showed tight basal cisterns and preherniation appearance, and the chronic subdural hematoma recurred three times.

Case Report

A 56-year-old female complained of headache, when she was dancing and shaking her head on April 29, 1993. Her headache gradually aggravated and she sometimes had nausea and vomiting. She became drowsy on May 11, 1993. She became comatose the next day. On admission, she had isocoric pupils and decerebrate posture with pain stimulation. CT showed bilateral subdural hematomas with tight basal cisterns (Fig. 1).

Percutaneous aspiration of the left subdural hematoma was performed with the twist drill. Her intracranial pressure was negative, and the aspirated hematoma receded spontaneously. The hematoma cavity was replaced with oxygen and then irrigated with saline.¹⁾ Postoperative CT showed residual oxygen in the hematoma cavity, the partially visualized basal cisterns, and the enlarged lateral ventricles (Fig. 2). She immediately became alert.

Three days after the operation, she noted headache. Repeat CT showed enlargement of the right chronic subdural hematoma. Percutaneous aspiration of the right chronic subdural hematoma was performed and again revealed negative intracranial

Fig. 1 Preoperative CT scans showing bilateral chronic subdural hematomas (*left*) and tight basal cisterns (*right*).

Fig. 2 Postoperative CT scans showing enlargement of the lateral ventricles, regardless of the mass effect of the residual air in the left frontal (*left*) and subarachnoid spaces, and the partially visualized ambient cisterns (*right*).

pressure. Further percutaneous drainage for recurrence of the right chronic subdural hematoma was required due to symptoms of hallucination, confusion, headache, and vomiting on June 1, and deep coma and urinary and rectal incontinence on June 14. All percutaneous aspirations revealed negative or low intracranial pressure (below 70 mmH₂O). Postoperatively, she took as much bed rest as possible because the recurrences of chronic subdural hematomas were thought to be aggravated by the upright position. She has had no symptom since and CT showed no recurrence of the chronic subdural hematoma 1 year later.

Serial examination of her blood, including laboratory biochemical findings and blood coagulation factors, showed normal findings and cerebral angiography found no abnormal vascularity.

Discussion

The etiology of SIH is unknown. Posture-dependent headache following minor trauma such as a fall on the buttocks may be due to a lumbar nerve sleeve tear with subsequent leakage of CSF.⁹⁾ In recent years, radioisotope cisternography with SIH has showed that the CSF flow is normal at the level of the spinal cord, but is suggestive of hyperabsorption or leakage of CSF just below the craniocervical junction.¹⁴⁾ Radioisotope cisternography has also revealed a CSF leak along the spinal axis and rapid accumulation of radioisotope in the bladder. Therefore, CSF leakage from spinal meningeal defects might be the most common cause of SIH.

CT shows that SIH is associated with extremely small ventricles and tight basal cistern.^{4,8)} Gadolinium-enhanced magnetic resonance imaging shows diffuse meningeal enhancement.⁷⁾ Such diffuse meningeal enhancement may result from the dural venous dilatation that accompanies reduced CSF volume due to the Monroe-Kellie doctrine.^{5,6)}

The symptoms of SIH frequently resolve spontaneously without specific therapy.⁷⁾ However, intramuscular dexamethasone treatment has achieved rapid resolution of the symptoms of SIH.⁸⁾ Therefore, the initial therapeutic approach is suggested to be conservative therapy with bed rest. Epidural blood patch has also rapidly resolved SIH caused by CSF leakage from a thoracolumbar arachnoid cyst.⁶⁾ Therefore, if radioisotope cisternography reveals CSF leakage, epidural blood patch should be considered for rapid improvement of the symptoms.

Chronic subdural hematoma may be caused by intracranial hypotension due to a shunting operation for hydrocephalus. The incidence of the chronic subdural hematoma was 1% in patients undergoing lumbo-peritoneal shunt and 1.6% in patients with ventriculo-peritoneal shunt.²⁾

Only a few patients with chronic subdural hematoma due to SIH have required surgery.^{10,13)} Percutaneous aspiration of the chronic subdural hematoma was followed by replacement by oxygen in our patient.¹⁾ CT after removal of the chronic subdural hematoma showed the subarachnoid space, especially in the ambient cistern, was only partially visualized and the lateral ventricles were enlarged, but the subdural mass volume caused by residual oxygen increased postoperatively. This paradoxical phenomenon is difficult to explain. The saline used to irrigate the hematoma cavity may have infiltrated into the subarachnoid space and increased the volume of the subarachnoid space and ventricles. The recurrence of the chronic subdural hematoma in our patient may

have been due to persistent SIH caused by CSF leakage. Radioisotope cisternography may be necessary to identify the origin of CSF leakage in such a case.

SIH is not always an easily controlled clinical condition, and the possibility of the complication of chronic subdural hematoma should be considered.

References

- 1) Aoki N: Percutaneous subdural tapping for the treatment of chronic subdural haematoma in adults. *Neurol Res* 9: 19-23, 1987
- 2) Aoki N: Lumboperitoneal shunt: Clinical applications, complications, and comparison with ventriculoperitoneal shunt. *Neurosurgery* 26: 998-1004, 1990
- 3) Bell WE, Joynt RJ, Sahs AL: Low spinal fluid pressure syndromes. *Neurology* 10: 512-521, 1960
- 4) Buchler P, Kubina FG: Spontane (essentielle) Aliquorrhoe. *Nervenarzt* 52: 361-363, 1981
- 5) Cushing H: *Studies in Intracranial Physiology and Surgery*. London, Oxford University Press, 1926, pp 19-23
- 6) Fishman RA, Dillon WP: Dural enhancement and cerebral displacement secondary to intracranial hypotension. *Neurology* 43: 609-611, 1993
- 7) Hochman MS, Naidich TP, Kobetz SA, Maitin AF: Spontaneous intracranial hypotension with pachymeningeal enhancement on MRI. *Neurology* 42: 1628-1630, 1992
- 8) Murros K, Fogelholm R: Spontaneous intracranial hypotension with slit ventricles. Short report. *J Neurol Neurosurg Psychiatr* 46: 1149-1151, 1983
- 9) Nosik WA: Intracranial hypotension secondary to lumbar nerve sleeve tear. *JAMA* 157: 1110-1111, 1955
- 10) Rando TA, Fishman RA: Spontaneous intracranial hypotension. Report of two cases and review of the literature. *Neurology* 42: 481-487, 1992
- 11) Schaltenbrand G: Neure Anschauen zur Pathophysiologie der Liquorzirkulation. *Zentralbl Neurochir* 3: 290-300, 1938
- 12) Schaltenbrand G: Die akute Aliquorrhoe. *Verh Deutsch Ges Inn Med* 52: 473-481, 1940
- 13) Sipe JC, Zyroff J, Waltz TA: Primary intracranial hypotension and bilateral isodense subdural hematomas. *Neurology* 31: 334-337, 1981
- 14) Weber WE, Heidendal GA, Krom MC: Primary intracranial hypotension and abnormal radionuclide cisternography. Report of a case and a review of the literature. *Clin Neurol Neurosurg* 93: 55-60, 1991

Address reprint requests to: H. Nakajima, M.D., Department of Neurosurgery, Neurological Institute, Tokyo Women's Medical College, 8-1 Kawada-cho, Shinjuku-ku, Tokyo 162, Japan.