Acute Subdural Hematoma After Lumboperitoneal Shunt Placement in Patients With Normal Pressure Hydrocephalus

—Four Case Reports—

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Abstract

Acute subdural hematoma (SDH) is a rare but disastrous complication after lumboperitoneal shunt placement. Four of 206 adult patients with normal pressure hydrocephalus (1.9%) who underwent lumboperitoneal shunt placement suffered acute SDH following head trauma. The interval between shunt placement and acute SDH was one month to 7 years. Two patients had subdural effusion on computed tomography (CT) at 2- and 6-month follow up. All four patients required assistance in their daily activities before acute SDH onset. The traumatic event was a fall. On admission, CT revealed a large SDH that required surgical removal in two patients, of whom one had manifested subdural effusion after shunt placement. The other two patients had a small SDH. None of the four patients had cerebral contusions. Patients with lumboperitoneal shunts, especially those not capable of independent daily activities, are at risk for acute SDH after even minor head trauma.

Key words: acute subdural hematoma, lumboperitoneal shunt, normal pressure hydrocephalus

Introduction

Lumboperitoneal shunts are widely used for cerebrospinal fluid (CSF) diversion, especially in patients with communicating hydrocephalus.^{3,5)} Lumboperitoneal shunting does not require cerebral cannulation, so the risk of intracranial complications is minimal, and results in fewer clinical complications than the placement of ventriculoatrial or ventriculoperitoneal shunts. Postsurgical complications occur in 30–40% of patients treated by CSF shunt procedures, and the rate of mortality or severe morbidity is 5–15%.⁹⁾ Various complications can occur after lumboperitoneal shunting, including shunt obstruction, infection, radicular pain, chronic subdural hematoma (SDH), and shunt migration.^{3,6)} The most disastrous complication is acute SDH.^{1–3,7)}

We have treated four patients with normal pressure hydrocephalus (NPH) who experienced acute SDH after lumboperitoneal shunt placement.

Case Reports

Computerized record review identified a total of 206 adult patients, 97 males and 109 females aged from 34 to 85 years (mean 65 years), who had undergone lumboperitoneal shunting using a non-programmable pressure valve for NPH at Kumamoto University Hospital and affiliated hospitals between January 1978 and December 2001. The diagnosis of NPH was based on the clinical symptoms and computed tomography (CT) cisternography. The cause of NPH was subarachnoid hemorrhage in 134 (65.0%), idiopathic in 16 (7.8%), intracerebral hemorrhage in four (1.9%), trauma in 19 (9.2%), tumor in nine (4.4%), meningitis in four (1.9%), and other causes in 20 patients (9.7%). By the time of the last follow up (December 31, 2001), four of these 206 patients (1.9%) had suffered acute SDH between one month and 7 years after lumboperitoneal shunting (Table 1).

The two males and two females were aged from 58 to 81 years (mean 71 years) at the onset of acute SDH. They had no coagulation abnormalities or hematologic disorders and had received no

Table 1 Summary of four cases of acute subdural hematoma (SDH) after lumboperitoneal shunt placement

Case No.	Age/ Sex	Cause of NPH	Post-shunt subdural effusion	GOS before onset of acute SDH	Cause of trauma	Period between shunt placement and acute SDH	Consciousness loss within the first hour	Operation	GOS at 3 mos after acute SDH
1	77/F	SAH	yes (6 mos after shunt placement)	SD	fall while moving from wheelchair to bed	10 mos	no	no	SD
2	81/M	SAH	no	MD	fall while walking	7 yrs	no	no	MD
3	58/M	idiopathic NPH	yes (2 mos after shunt placement)	MD	fall while walking	11 mos	no	craniotomy	VS
4	66/F	idiopathic NPH	no	MD	fall while walking	1 mo	yes	craniotomy	D

D: died, GOS: Glasgow Outcome Scale, MD: moderately disabled, NPH: normal pressure hydrocephalus, SAH: subarachnoid hemorrhage, SD: severely disabled, VS: vegetative state.

anticoagulant therapy. The cause of NPH was subarachnoid hemorrhage in Cases 1 and 2 and idiopathic in Cases 3 and 4.

These four patients underwent shunt operations between September 1988 and May 1995, using a one-piece tube with a distal slit valve (opening pressure 5–10 cmH₂O). Programmable pressure valves were not used. Briefly, under general or spinal anesthesia, a lumbar puncture at the L3-4 intervertebral level was performed and a silastic shunt tube was introduced rostrally into the subarachnoid space at a depth of 20 cm from the skin. The catheter was passed subcutaneously around the flank and the other end of the tube was introduced into the peritoneal cavity under direct vision.

CT revealed subdural effusion at the 2- and 6-month follow-up examinations in Cases 3 and 1, respectively. Subdural effusion was defined as a widened extracerebral space appearing as water density on CT. These two patients were monitored without intervention because neither manifested neurological deterioration attributable to the lesion. According to the Glasgow Outcome Scale (GOS), Case 1 was severely disabled, and Cases 2–4 were moderately disabled before the onset of acute SDH.

All four patients suffered head trauma as the result of falls and were taken to the emergency room. Cases 1–3 did not lose consciousness during the hour following the fall, but Case 3 subsequently manifested consciousness disturbance. Case 4, who partially depended on the support of others to walk in daily life, was semicomatose without signs of herniation on admission. Skull radiography revealed no fractures in any of the four patients and CT revealed

no cerebral contusion. However, serial CT later demonstrated a small contusion in Case 3. CT showed a large SDH with marked midline shift in Cases 3 and 4, and a small SDH in Cases 1 and 2 (Fig. 1).

Immediately after the detection of acute SDH, all four patients underwent lumboperitoneal shunt ligation. Cases 3 and 4 manifested progressive deterioration and emergency craniotomy and hematoma evacuation were performed. Case 4 suffered intractable seizure and died. According to the GOS at 3 months post-treatment, Case 2 was moderately disabled, Case 1 was severely disabled, and Case 3 was vegetative state.

Discussion

Four of 206 adult patients with NPH (1.9%) suffered acute SDH at one month to 7 years after treatment by lumboperitoneal shunting using a non-programmable pressure valve. Acute SDH after lumboperitoneal shunt placement is a rare but disastrous complication. One of 130 consecutive patients who underwent lumboperitoneal shunting manifested acute SDH after a severe fall.⁷⁾ Four patients developed acute SDH after minor head trauma, of whom one patient was treated for epidural CSF collection and the other three patients for NPH originating in subarachnoid hemorrhage in two patients and subcortical hemorrhage in one patient.^{2,3)} Two of our patients had subarachnoid hemorrhage as the original disease. Patients with NPH due to subarachnoid hemorrhage are apparently at increased risk for acute SDH after lum-

Fig. 1 Computed tomography scans of four patients before (left column) and after (center column) lumboperitoneal shunt placement and after the onset of acute subdural hematoma (right column). A: Case 1, B: Case 2, C: Case 3, D: Case 4.

boperitoneal shunt placement, but subarachnoid hemorrhage was the prevalent cause of NPH in both ours and the previous study population.^{2,3)}

Two of our patients had a history of post-shunt subdural effusion, which is not rare in patients subjected to a CSF diversion procedure.^{4,5)} Two of three patients with NPH and a lumboperitoneal shunt who developed acute SDH also had post-shunt subdural fluid collection or widening of the subarachnoid space (here defined as subdural effusion).²⁾ The presence of subdural effusion had no apparent effect on the size of the acute SDH in our patients, but we suggest that patients who experience subdural effusion after lumboperitoneal shunt placement are at increased risk for acute SDH following even minor head trauma.

None of our four patients were able to live independently and their compromised status may have worsened the consequences of falling. However, the head trauma preceding acute SDH was probably not severe because Cases 1–3 did not lose consciousness immediately after the fall, although Case 3 suffered consciousness disturbance later.

We suggest that the increase in intracranial pressure following acute SDH was prevented by the shunt bypass. Subsequently, although the shunt was immediately ligated, the hematoma continued to enlarge, leading to a further increase in intracranial pressure which resulted in progressive deterioration. However, neither subdural effusion nor shunt valve pressure affected the size of the subdural hematoma, as one of our patients with subdural effusion had a large SDH requiring craniotomy and evacuation, and the other had a small SDH with mild mass effect. Lumboperitoneal shunting used a medium-pressure valve in all four patients.

It is unclear whether previously reported patients with acute SDH after lumboperitoneal shunt placement would have benefited from shunt ligation, which we consider is preventive in patients with impending herniation. We propose that patients with progressive neurological deterioration should be treated as soon as possible by shunt ligation followed by craniotomy and hematoma evacuation.

The interval between lumboperitoneal shunt placement and acute SDH following head trauma was from one month to 7 years in our patients and up to 4 years in a previous study.²⁾ We cannot conclude that the lumboperitoneal shunt was the direct cause of the acute SDH in our series. However, elderly patients with unstable gait before shunt placement may experience improvement of their

gait disturbance and become more active after lumboperitoneal shunting, thus increasing their chance of suffering a fall. Follow up of patients after lumboperitoneal shunting should consider the possibility of increased risk for acute SDH following even minor head trauma.

The incidence of acute SDH appears to be lower after ventriculoperitoneal than lumboperitoneal shunt placement.³⁾ However, acute SDH is a complication in any procedure that entails CSF diversion.⁸⁾ Future large-scale, comparative studies will determine whether ventriculoperitoneal or lumboperitoneal shunt placement is preferable to prevent acute SDH.

References

- 1) Aoki N: Acute subdural hematoma of arterial origin in a patient with a lumboperitoneal shunt. *Neurol Med Chir (Tokyo)* 27: 60–62, 1987
- 2) Aoki N: Acute subdural hematoma due to minor head trauma in patients with a lumboperitoneal shunt. *Surg Neurol* 29: 22–26, 1988
- 3) Aoki N: Lumboperitoneal shunt: Clinical applications, complications, and comparison with ventriculoperitoneal shunt. *Neurosurgery* 26: 998–1004, 1990
- 4) Black PM: Idiopathic normal-pressure hydrocephalus. Results of shunting in 62 patients. *J Neurosurg* 52: 371–377, 1980
- 5) Chumas PD, Kulkarni AV, Drake JM, Hoffman HJ, Humphreys RP, Rutka JT: Lumboperitoneal shunting: A retrospective study in the pediatric population. *Neurosurgery* 32: 376–383, 1993
- 6) Samuelson S, Long DM, Chou SN: Subdural hematoma as a complication of shunting procedures for normal pressure hydrocephalus. *J Neurosurg* 37: 548–551, 1972
- 7) Selman WR, Spetzler RF, Wilson CB, Grollmus JW: Percutaneous lumboperitoneal shunt: Review of 130 cases. *Neurosurgery* 6: 255–257, 1980
- 8) Tamaki N, Suyama T, Shirakuni T, Matsumoto S: Acute subdural hematoma of arterial origin following ventriculoperitoneal shunting. *Neurol Med Chir (Tokyo)* 27: 643–646, 1987
- 9) Vanneste JA: Three decades of normal pressure hydrocephalus: are we wiser now? *J Neurol Neurosurg Psychiatry* 57: 1021–1025, 1994

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