Spontaneous Acute Subdural Hematoma due to Intracranial Hypotension Secondary to Lumboperitoneal Shunt: A Case Report and Review of the Literature

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ABSTRACT

Objective: To report a rare case with spontaneous intracranial acute subdural hematoma due to overdrainage of cerebrospinal fluid after lumboperitoneal shunting and to review the literature on this topic.

Case report: A 53-year-old lady with spontaneous acute subdural hematoma developing 3 years after lumboperitoneal shunting for treatment of benign intracranial hypertension is reported. She was treated with shunt removal and hematoma evacuation.

Material and Methods: We found 16 cases with intracranial bleeding developing spontaneously or after mild head injury after lumboperitoneal shunting. The characteristics of the patients were recorded, the outcome was given according to the Glasgow Outcome Scale, and a Glasgow Outcome Scale score from 1-3 was accepted as worse outcome. The factors affecting outcome were evaluated.

Results: There were 10 females and 7 males aged 59.7±15.1 years. In most cases, the primary disease treated by lumboperitoneal shunting was hydrocephalus or benign intracranial hypertension. In 12 of the cases, the bleeding happened into the subdural space and in 5 into other compartments (intracerebral or subarachnoid bleeding). Five of the cases died, and 62.5% had a worse outcome. The only factor affecting outcome was the time span from lumboperitoneal shunting to intracranial bleeding. This time was significantly shorter in the patients with worse outcome (3.7 versus 38.6 months).

Conclusions: Lumboperitoneal shunting may cause serious complications such as intracranial bleeding due to overdrainage of cerebrospinal fluid via shunt. Patients with lumboperitoneal shunting must be followed very closely for development of intracranial hypotension especially during the first few months after shunting.

Keywords: acute subdural hematoma, intracranial hemorrhages, intracranial hypotension, lumboperitoneal shunting

ÖZET

Lumboperitoneal şanta bağlı gelişen spontan akut subdural hematom: Olgu sunumu ve literatür taraması

Amaç: Lumboperitoneal şant uygulaması sırasında benign omurilik sıvasının aşıri drenajına bağlı spontan kaťa-façı akut subdural hematom gelişen nadir bir olgunun sunulması ve bu konuda literatür taraması yapılması.


Sonuçlar: Lumboperitoneal şant uygulaması şanttan aşırı omurilik sıvısı drenajına bağlı kaťa-façı kanama gibi ciddi komplikasyonlara neden olabilir. Lumboperitoneal şanti olgar özellikli şant takımlarından sonraiki il birkaŋ ay olmak üzere çok yakından izlenmeli.
**Introduction**

Lumboperitoneal (LP) shunting is frequently used to treat certain disorders such as benign intracranial hypertension and communicating hydrocephalus. It may be performed percutaneously and it is a minimally invasive operation that is simpler than ventriculoperitoneal (VP) shunting because it does not require ventricle catheterization (1). However, some serious complications may be seen after insertion of an LP shunt, too, the most important one being the development of intracranial hypotension due to overdrainage of cerebrospinal fluid (CSF) (2). Actually, the rate of this complication is quite high in patients with LP shunt. In a review on idiopathic intracranial hypertension, Friedman and Jacobson (3) reported a rate of up to 25%. Fortunately, in most cases symptoms and signs of intracranial hypotension due to LP shunt are usually mild. However, this intervention may cause very serious complications such as development of intracranial bleeding. Very few cases with spontaneous intracranial bleeding secondary to LP shunting have been reported in the literature.

We report a woman with development of spontaneous supratentorial acute subdural hematoma (ASDH) due to overdrainage of CSF via LP shunt. In addition, we review the literature for development of intracranial bleeding spontaneously or after mild head injury in patients with LP shunt and we discuss the characteristics of those patients.

**Case Report**

A 53-year-old woman had been admitted with complaint of suddenly and spontaneously developing severe headache 2 days earlier. There was benign intracranial hypertension in her history, and she had been treated with LP shunting in another center 3 years previously (Figure 1). Her neurological examination revealed no findings. A thin ASDH located in the right temporoparietal region, 7 mm in thickness, was seen on brain computerized tomography (CT) (Figure 2). Because of possible intracranial hypotension due to her history of LP shunting, brain magnetic resonance imaging (MRI) with intravenous contrast enhancement was performed and prominent pachymeningeal enhancement and thickening was seen (Figure 3). It was thought that spontaneous ASDH had

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**Figure 1:** Lateral abdominal X-ray showing lumboperitoneal shunt (white arrows).

**Figure 2:** Brain CT showing thin subdural hematoma on the right side.

**Figure 3:** Coronary T1-weighted section of the brain MRI with intravenous gadolinium showing diffuse and thick pachymeningeal enhancement.
Spontaneous acute subdural hematoma after LP shunting

Developed due to overdrainage of CSF via LP shunt. The LP shunt was removed and its entrance point to the lumbar dura was repaired with a piece of muscle and tissue glue. It was a valveless T tube catheter. The severe headache resolved after removal of the shunt. On control CTs, the hematoma increased in size and changed to chronic subdural hematoma (Figure 4). Subsequently, it was drained via a burr hole 10 days after the removal of the LP shunt. Neither subdural hematoma recurred, nor benign intracranial hypertension developed during 2 years’ follow-up (Figure 5).

Material and Methods

Review of the Literature
We reviewed the literature for development of intracranial bleeding spontaneously or after mild head injury in patients with LP shunt. Jones (4) reported 63 cases with spinoperitoneal shunt performed for treatment of communicating hydrocephalus in 1966, and there was one case dying due to subdural hematoma developing after lumbar arachnoid-Fallopian tube shunt. However, this case was not included in our study because the article provided no data about the patient and the shunt. Selman et al. (5) also reported a case with traumatic acute subdural hematoma 48 hours after LP shunting with a distal slit valve shunt system. However, this case was also not included in the present study because of inadequate data given in the article. We found 16 cases with adequate data in the literature (6-14). Age, gender, primary indication for LP shunting, LP shunt type and pressure, time span between LP shunting and development of intracranial bleeding, type of bleeding, presence or absence of head trauma causing intracranial bleeding, treatment modalities, and outcome of the 17 patients (including the 16 cases from the literature and our one) were recorded. Outcomes of the patients were evaluated according to the Glasgow Outcome Scale (GOS) (15); a GOS score of 1-3 was accepted as worse outcome and GOS score of 4-5 as good outcome. We also evaluated the factors affecting outcome of the patients.

Statistical Evaluations
Chi-square or Fisher’s exact tests were used to compare nominal variables according to their subject numbers. F test was used to compare the distribution of the series and Student’s t test was used to compare numerical variables according to the results of the F test. Results were accepted as significant if the p value was <0.05.

Results
All characteristics of the 17 cases are shown in Table 1. There were 10 females and 7 males at 59.7±15.1 (average± standard deviation-SD-, age range 40-88) years of age. The primary disease treated with LP shunting was hydrocephalus in 11 cases, benign intracranial hypertension (BIH) in 5, and development of pseudomeningocele after posterior fossa epidermoid cyst surgery in one.
Intracranial bleeding developed spontaneously in 7 of these cases including our one. In 12 of the cases, the bleeding occurred into the subdural space and in 5 into the other compartments: cerebellar hematoma in 2 cases, subarachnoid hemorrhage (SAH) accompanying intracerebral hematoma (ICH) in 2 cases, and ICH in one case. Most of the cases with ASHD had a history of mild trauma. There were only two cases with ASHD developing spontaneously including our patient. However, there was no history of trauma in any of the 5 cases with bleeding into the other compartments (p=0.0033).

The time span from LP shunting to bleeding was not mentioned for two cases in one article (13), while it was 16.2±25.9 months (average±SD, ranging from a few hours to 7 years) in the others.

In most cases (8 cases), a nonprogrammable medium pressure (MP) valve was used. In the cases with spontaneously developed bleeding, the LP shunt system was valveless in 2 cases, with distal slit valve in 4 cases, and with programmable valve in one case.

### Treatment Modalities

After development of bleeding, only hematoma evacuation was performed in 5 cases, all of whom had ASHD. In one of them, LP shunt ligation was required after a few days because of rebleeding (13). One of these patients was died, and the GOS scores of the others were 3 in two cases and 5 in other two.

Lumboperitoneal shunt ligation or removal was performed in 5 cases. One of them died, and GOS scores of the others were 3 in two cases, 4 in one and 5 in the last one. In 2 of them, including our patient, a chronic subdural hematoma developed at the site of ASHD, and it was evacuated a few days after shunt ligation/removal.

Hematoma evacuation and shunt ligation/removal were performed in the same session in 4 cases. Two of them died. The GOS score was 2 in one case and 5 in the other.

In 2 cases, conservative treatment was applied without any intervention (10,14). One of them died and the GOS score of the other was 5.

### Table 1: Characteristics of cases with LP shunting and intracranial bleeding occurring spontaneously or after mild trauma.

<table>
<thead>
<tr>
<th>No</th>
<th>Study</th>
<th>Age/Gender</th>
<th>Bleeding</th>
<th>Primary disease</th>
<th>Head injury</th>
<th>Time span</th>
<th>Neurological condition</th>
<th>Treatment</th>
<th>Shunt type</th>
<th>CSDH</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Aoki (6)</td>
<td>44/F</td>
<td>ASDH</td>
<td>Pseudo-meningocele</td>
<td>yes</td>
<td>23 days</td>
<td>Comatose</td>
<td>HE</td>
<td>MP valve</td>
<td>yes</td>
<td>GOS 3</td>
</tr>
<tr>
<td>2</td>
<td>Aoki (6)</td>
<td>59/E</td>
<td>ASDH</td>
<td>NPH</td>
<td>yes</td>
<td>22 months</td>
<td>Comatose</td>
<td>HE</td>
<td>MP valve</td>
<td>yes</td>
<td>GOS 5</td>
</tr>
<tr>
<td>3</td>
<td>Aoki (6)</td>
<td>59/F</td>
<td>ASDH</td>
<td>NPH</td>
<td>yes</td>
<td>50 months</td>
<td>Stupor</td>
<td>HE</td>
<td>MP valve</td>
<td>yes</td>
<td>GOS 5</td>
</tr>
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<td>4</td>
<td>Aoki (6)</td>
<td>79/F</td>
<td>ASDH into CSDH</td>
<td>NPH</td>
<td>yes</td>
<td>7 months</td>
<td>Comatose</td>
<td>HE</td>
<td>MP valve</td>
<td>yes</td>
<td>GOS 1</td>
</tr>
<tr>
<td>5</td>
<td>Ayvalik (7)</td>
<td>52/M</td>
<td>ASDH</td>
<td>Cerebellar ICH</td>
<td>BIH</td>
<td>no</td>
<td>1 day</td>
<td>Comatose</td>
<td>Alert</td>
<td>HE/SL</td>
<td>Distal slit valve</td>
</tr>
<tr>
<td>6</td>
<td>Barash (8)</td>
<td>42/M</td>
<td>ASDH</td>
<td>CSDH into CSDH</td>
<td>BIH</td>
<td>no</td>
<td>2 weeks</td>
<td>Comatose</td>
<td>Alert</td>
<td>SL</td>
<td>MP valve</td>
</tr>
<tr>
<td>7</td>
<td>Kamiryo (9)</td>
<td>77/F</td>
<td>ASDH</td>
<td>NPH</td>
<td>yes</td>
<td>10 months</td>
<td>Alert</td>
<td>SL</td>
<td>MP valve</td>
<td>yes</td>
<td>GOS 3</td>
</tr>
<tr>
<td>8</td>
<td>Kamiryo (9)</td>
<td>81/M</td>
<td>ASDH</td>
<td>NPH</td>
<td>yes</td>
<td>7 years</td>
<td>Alert</td>
<td>SL</td>
<td>MP valve</td>
<td>no</td>
<td>GOS 3</td>
</tr>
<tr>
<td>9</td>
<td>Kamiryo (9)</td>
<td>58/F</td>
<td>ASDH</td>
<td>NPH</td>
<td>yes</td>
<td>11 months</td>
<td>Comatose</td>
<td>Alert</td>
<td>SL</td>
<td>MP valve</td>
<td>yes</td>
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<tr>
<td>10</td>
<td>Kamiryo (9)</td>
<td>66/F</td>
<td>ASDH</td>
<td>NPH</td>
<td>yes</td>
<td>1 month</td>
<td>Comatose</td>
<td>Alert</td>
<td>SL</td>
<td>MP valve</td>
<td>no</td>
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<tr>
<td>11</td>
<td>Turkoglu (10)</td>
<td>44/F</td>
<td>Parietal ICH</td>
<td>BIH</td>
<td>no</td>
<td>A few hrs</td>
<td>Comatose</td>
<td>Conservative</td>
<td>Distal slit valve</td>
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<td>GOS 1</td>
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<tr>
<td>12</td>
<td>Suri (11)</td>
<td>42/F</td>
<td>SAH, frontal ICH</td>
<td>BIH</td>
<td>no</td>
<td>40 hrs</td>
<td>Comatose</td>
<td>HE/SL</td>
<td>Distal slit valve</td>
<td>no</td>
<td>GOS 5</td>
</tr>
<tr>
<td>13</td>
<td>Castillo (12)</td>
<td>40/F</td>
<td>ICH, SAH</td>
<td>BIH</td>
<td>no</td>
<td>In a few hrs</td>
<td>NA</td>
<td>Stupor</td>
<td>NA</td>
<td>NA</td>
<td>GOS 3</td>
</tr>
<tr>
<td>14</td>
<td>Hoya (13)</td>
<td>88/F</td>
<td>ASDH</td>
<td>NPH</td>
<td>yes</td>
<td>3 days</td>
<td>NA</td>
<td>Stupor</td>
<td>NA</td>
<td>NA</td>
<td>GOS 3</td>
</tr>
<tr>
<td>15</td>
<td>Hoya (13)</td>
<td>64/M</td>
<td>ASDH</td>
<td>NPH</td>
<td>yes</td>
<td>3 days</td>
<td>NA</td>
<td>Stupor</td>
<td>NA</td>
<td>NA</td>
<td>GOS 3</td>
</tr>
<tr>
<td>16</td>
<td>Er (14)</td>
<td>67/F</td>
<td>Cerebellar ICH</td>
<td>BIH</td>
<td>no</td>
<td>4 days</td>
<td>Alert</td>
<td>Alert</td>
<td>SV first, then HE (chronic)</td>
<td>PV/MP</td>
<td>Valveless</td>
</tr>
<tr>
<td>17</td>
<td>Our patient</td>
<td>53/F</td>
<td>ASDH</td>
<td>BIH</td>
<td>no</td>
<td>3 years</td>
<td>Alert</td>
<td>Alert</td>
<td>SL first, then HE (chronic)</td>
<td>PV/MP</td>
<td>Valveless</td>
</tr>
</tbody>
</table>

Factors Affecting Outcome

Out of 17 cases, 5 cases died (31.2%). The GOS score was 2 in one, 3 in 4, 4 in one and 5 in 5 cases. The outcome of one patient (12) was not mentioned in the article. In total, 10 of the other 16 patients (62.5%) had worse outcome (GOS 1-3).

Age and sex were not statistically significant when comparing patients with worse (GOS 1-3) or good outcome (GOS 4-5) (p=0.87 and p=1, respectively), nor between the dying and surviving patients (p=0.44 and p=1, respectively). Presence or absence of trauma history and bleeding compartment also were not statistically significant comparing patients with worse and good outcome nor between the dying and surviving patients (p=0.6 and p=0.29, respectively, for presence/absence of trauma history, and p=0.6 and p=0.54, respectively, for bleeding compartment). Type of the primary disease treated with LP shunting was also not significantly different in the patients with worse and good outcome and between the dying and surviving patients (p=0.6 and p=0.54, respectively). Seven patients were comatose after bleeding and 3 of them died; however, 2 of 9 patients who were not comatose also died (p=0.59).

On the other hand, the time span from LP shunting to intracranial bleeding was statistically shorter in the cases with worse and good outcome and between the dying and surviving patients (p=0.6 and p=0.54, respectively). Seven patients were comatose after bleeding and 3 of them died; however, 2 of 9 patients who were not comatose also died (p=0.59).

In conclusion, although LP shunting is an easy and smart technique to treat patients with communicating hydrocephalus, unfortunately we could not show such an effect of LP shunt systems had been used in these patients, such as valveless T tube, valves with constant medium pressure, distal slit valves, or programmable valves. Most of the cases had ASDH developing after mild head injury.

Unfortunately, the outcome of these patients was not very good, with 62.5% of them having GOS scores from 1-3. We could not find any factor significantly affecting the outcome of the patient except the time span from LP shunting to intracranial bleeding. Interestingly, the time span was significantly shorter in patients with worse outcome than in patients with good outcome (3.7±4.7 months and 38.6±28.1 months, respectively). This finding was thought to indicate that intracranial hypotension secondary to LP shunting in these patients developed quickly and severely and resulted in more massive bleeding. However, this finding gave us an an important clue for future patients with LP shunting. We recommended that these patients must be very closely followed for symptoms and signs of intracranial hypotension, especially during the first few months after shunting. Warning signs and symptoms of intracranial hypotension such as postural headache, tinnitus, nausea, and vomiting, etc. must be inquired about during outpatient control examinations.

Other factors including age, gender, type of primary disease treated with LP shunting, presence or absence of head injury, neurological condition after bleeding, and bleeding compartment did not significantly affect the outcome of the patients in this small series. It was expected that a valveless shunt system or one with smaller pressure might more frequently cause intracranial hypotension and bleeding, but unfortunately we could not show such an effect of LP shunt type, probably because of the small subject number.

In conclusion, although LP shunting is an easy and smart technique to treat patients with communicating hydrocephalus...
or benign intracranial hypertension, it must be kept in mind that this procedure may cause intracranial bleeding, occurring spontaneously or after mild head injury, due to overdrainage of the CSF. Because mortality and morbidity are quite high in this condition, patients with LP shunting should be very closely followed for warning signs and symptoms of intracranial hypotension, especially during the first a few months.

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References