Cerebellar Hemorrhage after Posterior Lumbar Decompression and Interbody Fusion Complicated by Dural Tear - A Case Report -

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Cerebellar Hemorrhage after Posterior Lumbar Decompression and Interbody Fusion Complicated by Dural Tear - A Case Report -

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Study Design: A case report.
Objectives: To report a rare case of remote cerebellar hemorrhage (RCH), which was a complication after posterior decompression and lumbar interbody fusion (PLIF).
Summary of Literature Review: Remote cerebellar hemorrhage (RCH) after spinal surgery is a rare complication, and its cause is known to be due to a loss of cerebral spinal fluid (CSF) through the dural tear. Most of the literature has disclosed that early diagnosis and treatment of RCH is very important in the patient with suspicious symptoms.
Materials and Methods: A 57-year-old woman had posterior lumbar decompression and interbody fusion for the severe spinal stenosis at L4-5. During surgery, an accidental dural tear with CSF leakage occurred. The torn dura was sutured. Postoperatively, she developed nausea and a severe headache. Hypotension developed at postoperative 2 hours. A brain CT showed RCH. The patient was conservatively managed with clamping of the wound drainage.
Results: The nausea and severe headache were controlled and normal blood pressure could be maintained without dopamine therapy at postoperative day 2. The patient was discharged without any neurological deficit, and her consciousness was clear at postoperative 2 weeks.
Conclusions: Persistent postoperative nausea, headache, and hypotension after repair of the torn dura may suggest that the treating surgeons pay careful attention due to the possibility of RCH, even though the amount of CSF leakage is small.

Key Words: Cerebellar Hemorrhage, CSF, Dura, tear, Lumbar vertebrae

INTRODUCTION

Remote cerebellar hemorrhage (RCH) is a rare condition which can complicate after supratentorial surgery. Also RCH was reported less frequently after spinal surgery than that of post-cranial surgery. Recently it has been often reported(Table 1).1-3 In South Korea, only five articles about spinal surgery-related RCH were reported by neurosurgeons1,4 and anesthesiologists. Although the pathophysiology of RCH has not been clarified, all cases of RCH after spinal surgery had history of a leakage of cerebrospinal fluid (CSF) or dural tear during operation.

We experienced a case of RCH after posterior decompression and lumbar interbody fusion (PLIF) at L4–5 level. Dural tear with CSF leakage was repaired. Early diagnosis and management could prevent disastrous outcome. The current case is reported with a review of related literatures.

CASE REPORT

A 57-year-old woman with spinal stenosis complained back pain and radiating pain to both legs. She had a neurological claudication and could bear only about five-minute walking. She had no history of diabetes mellitus, hypertension and
anticoagulant therapy. On simple radiogram grade I degenerative spondylolisthesis was found, and on magnetic resonance imaging (MRI) severe spinal stenosis with hypertrophy of ligamentum flavum and facet joint arthropathy at L4–5 level was observed (Fig. 1). She had posterior decompression and lumbar interbody fusion of L4-5 segment (Fig. 2).

During the operation, dura was torn accidently. There was CSF leakage through about 5mm longitudinal dural tear. The tear was sutured with non-absorbable suture. Blood pressure was maintained during surgery. Estimated blood loss was 700cc for which a 320cc packed RBC was transfused. Immediate postoperatively there was no neurological change and vital sign. At postoperative 2 hours, hypotension (blood pressure 80/40 mmHg), headache and nausea developed. Hydration with normal saline 500cc was carried out and a packed RBC was transfused, but hypotension persisted for which dopamine was

- **Table 1. The reported cases of remote cerebellar hemorrhage complicated after lumbar spine surgery**

<table>
<thead>
<tr>
<th>Case</th>
<th>Author (Year)</th>
<th>Pathology</th>
<th>Dura tear</th>
<th>Clinical problem</th>
<th>Treatment</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Andrews et al. (1995)</td>
<td>Scoliosis</td>
<td>Occult dural tear</td>
<td>Deterioration in mental status</td>
<td>EVD</td>
<td>Quadraparetic</td>
</tr>
<tr>
<td>2</td>
<td>Gobel et al. (1999)</td>
<td>Postdiscectomy syndrome</td>
<td>Tear</td>
<td>Headache, Lost consciousness</td>
<td>Decompression+EVD</td>
<td>Recovery</td>
</tr>
<tr>
<td>3</td>
<td>Gobel et al. (1999)</td>
<td>Spondylolisthesis</td>
<td>Tear</td>
<td>Headache, Lost consciousness</td>
<td>EVD</td>
<td>Residual disability</td>
</tr>
<tr>
<td>4</td>
<td>Friedman et al. (2002)</td>
<td>Spondylolisthesis</td>
<td>Occult dural tear</td>
<td>Headache, vomiting, dysarthria, ataxia</td>
<td>Dura repair</td>
<td>Residual dysarthria</td>
</tr>
<tr>
<td>5</td>
<td>Thomas et al. (2002)</td>
<td>Schwannoma</td>
<td>Intradural manipulation</td>
<td>Headache, nausea</td>
<td>Conservative</td>
<td>Recovery</td>
</tr>
<tr>
<td>6</td>
<td>Karaeminogullari et al. (2005)</td>
<td>Spinal stenosis</td>
<td>Tear</td>
<td>Lost consciousness</td>
<td>Decompression</td>
<td>Mild ataxia</td>
</tr>
<tr>
<td>7</td>
<td>Farag et al. (2005)</td>
<td>Spinal stenosis</td>
<td>Occult dural tear</td>
<td>Lost consciousness</td>
<td>Decompression</td>
<td>Recovery</td>
</tr>
<tr>
<td>8</td>
<td>Brockmann et al. (2005)</td>
<td>Spondylolisthesis</td>
<td>Tear</td>
<td>Headache, vomiting, dysarthria</td>
<td>EVD</td>
<td></td>
</tr>
<tr>
<td>9</td>
<td>Chalela et al. (2006)</td>
<td>Spinal stenosis</td>
<td>Tear</td>
<td>Headache, vomiting, dysarthria</td>
<td>Conservative</td>
<td>Recovery</td>
</tr>
<tr>
<td>10</td>
<td>Konya et al. (2006)</td>
<td>Herniated disc + spinal stenosis</td>
<td>Tear</td>
<td>Headache, vomiting, dysarthria</td>
<td>Conservative</td>
<td>Recovery</td>
</tr>
<tr>
<td>11</td>
<td>Calisaneller et al. (2007)</td>
<td>Spondylolisthesis</td>
<td>Tear</td>
<td>Headache, nausea</td>
<td>Conservative</td>
<td>Recovery</td>
</tr>
<tr>
<td>12</td>
<td>Nam et al. (2009)</td>
<td>Herniated disc + spinal stenosis</td>
<td>Tear</td>
<td>Headache, nausea, diminished consciousness</td>
<td>Decompression+EVD</td>
<td></td>
</tr>
<tr>
<td>13</td>
<td>Pallud et al. (2009)</td>
<td>Spondylolisthesis</td>
<td>Occult dural tear</td>
<td>Headache, Lost consciousness</td>
<td>EVD</td>
<td>Recovery</td>
</tr>
<tr>
<td>14</td>
<td>Kim et al. (2010)</td>
<td>Herniated disc + Spondylolisthesis</td>
<td>Tear</td>
<td>Headache, nausea, disoriented, dysarthria</td>
<td>Decompression+EVD</td>
<td>Wheelchair ambulation</td>
</tr>
<tr>
<td>15</td>
<td>Fernandez et al. (2011)</td>
<td>Spinal stenosis</td>
<td>Tear</td>
<td>Headache, vomiting, diminished consciousness</td>
<td>Conservative</td>
<td>Recovery</td>
</tr>
<tr>
<td>16</td>
<td>You et al. (2012)</td>
<td>Herniated disc + Failed back syndrome</td>
<td>Tear</td>
<td>Postural headache, CSF leakage through surgical wound</td>
<td>Dura repair</td>
<td>Recovery</td>
</tr>
<tr>
<td>17</td>
<td>Khalatbari et al. (2012)</td>
<td>Herniated disc</td>
<td>Occult dural tear</td>
<td>Headache, vomiting, lost consciousness</td>
<td>EVD</td>
<td>Recovery</td>
</tr>
<tr>
<td>18</td>
<td>Khalatbari et al. (2012)</td>
<td>Spinal stenosis</td>
<td>Dura tear</td>
<td>Failure of anesthesia recovery</td>
<td>Decompression</td>
<td>Die</td>
</tr>
<tr>
<td>19</td>
<td>Yoo et al. (2013)</td>
<td>Intradural mass</td>
<td>Intradural manipulation</td>
<td>Headache, nausea, vomiting, dizziness nystagmus</td>
<td>Decompression</td>
<td>Recovery</td>
</tr>
<tr>
<td>20</td>
<td>Current case</td>
<td>Spondylolisthesis</td>
<td>Tear</td>
<td>Headache, vomiting</td>
<td>Conservative</td>
<td>Recovery</td>
</tr>
</tbody>
</table>

EVD: extraventricular drainage.
Fig. 1. Sagittal view (A) of preoperative MRI shows spondylolisthesis with dural compression at L4-5 level. Axial view (B) shows severe dural compression due to both facet joint and ligamentum flavum hypertrophy.

Fig. 2. Immediate postoperative AP (A) and Lateral (B) x-rays show posterior decompression and reduced spondylolisthesis after posterior lumbar interbody fusion at L4-5 level.
infused. In spite of normalized blood pressure, headache and nausea aggravated and vomiting developed at postoperative day 1. To look for the cause, brain CT was obtained at postoperative day 1. The brain CT revealed bilateral cerebellar hemorrhages facing the tentorium Fig. 3A). The hemorrhage pattern was considered to be ‘zebra sign’ which was produced by linear hyperdensities of bleeding and relatively hypodense cerebellar folia. Neurosurgical consultation about the cerebellar hemorrhages was obtained. The total suction drainage reached to 510cc at postoperative day 1, and the drainage was stopped immediately. She maintained fortunately normal neurology and consciousness. Amount of the cerebellar hemorrhage was small. Thus, she was managed conservatively. Normal saline and mannitol were infused intravenously while dopamine infusion was tapered.

At postoperative day 2, headache, nausea and vomiting lessened. Blood pressure became stabilized without dopamine infusion. On follow-up brain CT, taken at postoperative day 5 slightly increased cerebellar hemorrhages was observed. But the patient had normal neurology without headache. On postoperative 2 week follow-up CT, cerebellar hemorrhage was absorbed almost completely(Fig. 3B). Consequently the patient could be discharged without any residual symptoms such as headache, unconsciousness and neurological change. At postoperative 1 year follow-up, she had no sequel of cerebellar hemorrhage(Fig. 3C).

**DISCUSSION**

Hemorrhage in the remote region - the cerebellum after spinal surgery is a rare complication which is usually known to have favorable prognosis, and to be the self-limiting one. But sometimes it can be life-threatening and needs the surgery. So, early diagnosis and proper treatment are essential, if there are suspicious symptoms suggesting RCH in the patients who have CSF leakage through accidental dural tear during surgery.

Since Chadduck’s first case report of remote cerebellar hemorrhage in 1981, its cause after supratentorial or spinal surgery had been discussed. As yet, its exact pathophysiology has not been clarified, but most of authors thought that RCH was probably venous in origin and caused by perioperative massive CSF leakage, based on the fact that there was CSF leakage through surgical dural tear in all cases of RCH after spinal surgery. In current case, dural tear also complicated during posterior decompression at L4–5. But there was no massive CSF leakage during operation and torn dura was repaired immediately. Postoperative drained fluid was not transparent, rather bloody. So velocity of CSF leakage was thought to be
more important factor for RCH than leaked amount of spinal fluid in this case.

The most common clinical symptoms of RCH were headache and unclear consciousness. In the current case, headache and hypotension which couldn’t be controlled by hydration and transfusion, and those were the main postoperative clinical features. She had no consciousness change. Postoperative arterial hypertension was reported to be a possible risk factor for RCH. But in the current case, postoperative continuous hypotension could be a contributing factor for RCH. It is assumed that continuous hypotension accompanied with CSF leakage could cause intracranial hypotension resulting in downward cerebellar displacement or “sagging”.

A study reported the prevalence of asymptomatic RCH was 0.8% in the patient who underwent supratentorial craniotomies. Incidence of asymptomatic RCH after spinal surgery may be also higher than that of the suggested one by previous reports. That is the reason why asymptomatic cerebellar hemorrhages are unlikely to be detected. Yoich et al emphasized that early diagnosis is particularly important for the treatment of RCH following spinal surgery. In their report patient with severe headache and nausea without neurological change after T9–10 laminectomy with unintentional dural tear underwent second operation for torn dural repair without brain CT or MRI. The patient’s consciousness gradually deteriorated and transferred to their institution at second postoperative day 2. Subsequently the patient underwent cranial decompression. So even though headache was the only symptom, brain CT or MRI should be taken in the patient who had CSF leakage or dural tear during spinal surgery. In the case in whom headache could not be controlled by analgesics, and also even in whom the only symptom without diminished consciousness brain CT should be taken.

In all previous reports, neurological symptoms developed at least ten hours after surgery. In patient with conscious change, cranial surgery had been needed for RCH. But in the current case, headache developed at postoperative 2 hours without conscious change. By brain CT RCH could be diagnosed earlier. When tentative diagnosis of RCH is made, suction drain should be stopped quickly and the patient should be treated early to prevent aggravation of RCH.

The surgeon must always pay attention that RCH can complicate in the patient who has CSF leakage through dura tear during spinal surgery, even though the amount of CSF leakage is small. It is particularly important to detect RCH early even when the patient had only headache before consciousness change develop.

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요추 후방 감압과 추체간 유합술시 합병한 경막 소뇌 출혈 - 증례 보고 -
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연구계획: 증례 보고
목적: 저자 수술 후 드물게 발생하는 원격부 소뇌 출혈에 대한 한 증례 보고와 문헌고찰

선행문헌의 요약: 저자 수술 중 또는 수술 후 뇌척수액의 누출에 의한 소뇌 출혈은 드물게 보고되었으며, 의심증상 발생시 빠른 진단과 치료가 필요함이 강조되었다.

대상 및 방법: 57세 여자 환자로 제 4-5요추의 심한 척추관 협착증으로 후방 광범위 감압과 추체간 유합술을 시행받았으며, 수술중 경막 천공 및 뇌척수액 누출이 있어 경막 봉합을 시행한 바 있다. 술후 2시간째부터 혈압 저하 및 오심, 두통 증상을 호소하여 활영한 뇌 컴퓨터 단층촬영상 소뇌에 출혈 소견이 보이어 수술부의 배액관을 닫고 보존적 치료를 시행하였다.

결과: 수술 후 배액관을 닫고 보존적 치료를 시행 후 1일째(술후 2일째)부터 혈압 상승제 없이 정상 혈압 유지 및 오심, 두통이 감소하였으며 수술 후 2주째 신경학적 변화 및 의식 변화 없이 퇴원하였다.

결론: 저자 수술 중 또는 수술 후 뇌척수액 누출이 있는 경우, 누출된 양이 적더라도 수술 후 혈압 저하, 오심, 두통이 발생시 원격부 소뇌 출혈을 의심해야 하고 빠른 진단과 치료가 필요하다.

색인 단어: 소뇌 출혈, 뇌척수액, 경막, 파열, 요추
약칭 제목: 후방 추체간 유합술후 소뇌 출혈