Syringomyelia and Arachnoid Cysts Associated With Spinal Arachnoiditis Following Subarachnoid Hemorrhage

—Case Report—

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Abstract

A 66-year-old woman with primary Sjogren syndrome developed syringomyelia following two episodes of subarachnoid hemorrhage (SAH) due to the rupture of basilar artery aneurysms. Gait disturbance and abnormal sensation with pain over the foot and abdomen appeared 3 years after the last SAH. Magnetic resonance (MR) imaging revealed a syringomyelia throughout the thoracic cord, from the T2 to T11 levels. In addition, the thoracic cord was compressed by multiple arachnoid cysts in the ventral side of spinal cord. Computed tomography myelography revealed complete block of cerebrospinal fluid (CSF) flow at the T7 level. Surgery for microlysis of the adhesions and restoration of the CSF flow pathway was performed. Postoperatively, leg motor function slowly improved and she could walk unaided. However, abdominal paresthesia was persisted. Postoperative MR imaging revealed diminished size of the syrinxes. We should recognize syringomyelia and arachnoid cysts due to adhesive arachnoiditis as a late complication of SAH. Microlysis of the adhesions focusing on the lesion thought to be the cause of the symptoms is one of the choices to treat massive syringomyelia and arachnoid cysts associated with arachnoiditis following SAH.

Key words: syringomyelia, arachnoid cyst, subarachnoid hemorrhage, adhesive arachnoiditis
Introduction

Syringomyelia and arachnoid cysts associated with arachnoiditis are commonly caused by trauma, infection, and surgery of the spinal cord, but are rare complications of subarachnoid hemorrhage (SAH). Severe cases with extensive adhesive arachnoiditis are very difficult to treat. Here, we present a severe case which was successfully treated with a simple surgical strategy.

Case Report

A 46-year-old female patient presented with SAH caused by rupture of a basilar artery and posterior inferior cerebellar artery aneurysm. The patient was treated with craniotomy and clipping of the basilar artery aneurysm. She presented with dry eyes, dry mouth, Raynaud’s phenomenon, erythema annulare, and polyarthritis of the hands at age 50 years. Lacrimal and salivary hyposecretion was confirmed by physiological examination. As a result, she was diagnosed with primary Sjögren’s syndrome. The patient developed SAH caused by the rupture of the basilar tip aneurysm at age 63 years. She was treated by coil embolization followed by lumbar drainage for a few days. There were no signs of hydrocephalus.

The patient presented at our institution at age 66 years with a 4-month history of gait disturbance and numbness in the lower extremities. On admission, the manual muscle test in the lower extremities demonstrated antigravity strength proximally and 4/5th of antigravity strength distally. However, tone was increased in the bilateral lower extremities. Sensory examination demonstrated pinprick hypesthesis at T10 on the left and T6 on the right, and abnormal sensation with pain on the foot. The patient also complained of gradual worsening of bladder function, with occasional episodes of urinary incontinence. Preoperative examination included lumbar puncture, which

Fig. 1 Photograph of the viscous and xanthochromic cerebrospinal fluid (CSF) obtained from the lumbar puncture. CSF protein, glucose, adenosine deaminase, and cell count were within normal limits and CSF Gram stain was negative.

Fig. 2 A: Sagittal T2-weighted magnetic resonance (MR) image revealing deformation of the spinal cord and a syrinx cavity extending from the T2 to T11 levels, and multiple arachnoid cysts on the ventral side of spinal cord (arrowheads). B: Sagittal computed tomography (CT) myelogram revealing a complete block of cerebrospinal fluid flow at the T7 level (arrow). C: Axial T2-weighted MR image revealing a cyst compressing the spinal cord from the ventral side at the T5-6 level. D: Axial CT myelogram revealing that the spinal cord was displaced laterally due to arachnoid adhesion and the syrinx was filled with contrast medium (arrow) at the T10 level.

Fig. 3 A: Intraoperative photograph after opening the dura showing thickened arachnoid membrane (arrow) around the spinal cord (SC). B: Intraoperative photograph showing the arachnoid adhesions were carefully dissected, and the spinal cord (SC) and nerve roots (NR) were untethered (arrow). C: Sagittal T2-weighted magnetic resonance image revealing diminished size of the syrinxes.
Syringomyelia and arachnoid cysts are rare complications of tissue. The dura mater was sutured with 4-0 of intense adhesion of the arachnoid membrane to the subarachnoid space lower than the T7 level because confirmed the connection between the intact and lesioned subarachnoid space above the T2 level when the subarachnoid space. CSF flowed out from the intact open the isolated subarachnoid space to the intact upper of adhesive arachnoiditis was at the T2 level, so we could retraction of the spinal cord. A cyst located on the ventral arachnoid membrane which could be confirmed without nal cord as far as possible, and dissected only the from the T2 to T7 levels. We avoided manipulating the spinal cord as far as possible, and dissected only the arachnoid membrane which could be confirmed without retraction of the spinal cord. A cyst located on the ventral side of the spinal cord at the T5-6 level was punctured, causing CSF to gush out under high pressure. The top level of adhesive arachnoiditis was at the T2 level, so we could open the isolated subarachnoid space to the intact upper subarachnoid space. CSF flowed out from the intact subarachnoid space above the T2 level when the arachnoid membrane was dissected. However, we confirmed the connection between the intact and lesioned subarachnoid spaces at the T2 level. We could not define intact subarachnoid space lower than the T7 level because of intense adhesion of the arachnoid membrane to the surrounding tissue. The dura mater was sutured with 4-0 Nurolon® (Johnson & Johnson Medical Ltd., Livingston, West Lothian, UK). Her urinary incontinence and spastic gait were improved postoperatively and MR imaging demonstrated reduction in the size of the syringes (Fig. 3C). Three years after the surgery, the patient could walk unabaided and there was no evidence of the recurrence. However, abdominal paresthesia persisted.

**Discussion**

Syringomyelia and arachnoid cysts are rare complications of SAH, with a few reports about this clinical condition.

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<table>
<thead>
<tr>
<th>Author (Year)</th>
<th>Aneurysm location</th>
<th>Treatment for SAH</th>
<th>Interval after SAH</th>
<th>Level of adhesive arachnoiditis</th>
<th>Spinal lesion</th>
<th>Treatment for spinal lesion</th>
<th>Presurgical symptoms/outcome</th>
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<tbody>
<tr>
<td>Taguchi et al. (1996)</td>
<td>unknown</td>
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<td>9 mos</td>
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<td>unknown</td>
<td>laminectomy and microlysis of adhesion</td>
<td>paraplegia with sensory/ motor deficit</td>
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<td></td>
<td>PICA</td>
<td>clipping</td>
<td>4 yrs</td>
<td>T7–T11</td>
<td>arachnoid cyst</td>
<td>laminectomy, syringostomy, and CSF drainage</td>
<td>paraparesis and paresthesia/ persisted</td>
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<tr>
<td></td>
<td>VA-PICA</td>
<td>clipping and duraplasty with fibrin glue</td>
<td>4 mos</td>
<td>T6-T8</td>
<td>arachnoid cyst at T3-T6, syringomyelia at T8</td>
<td>laminectomy and microlysis of adhesion</td>
<td>pain sensation/ persisting; leg spasticity/ persisted</td>
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<td>Lorenzana-Honrado et al. (1996)</td>
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<td>clipping</td>
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<td>cyst-peritoneal shunt</td>
<td>lower extremity weakness, urinary retention/ improved</td>
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<td>ICA-PcomA</td>
<td>craniotomy</td>
<td>5 wks</td>
<td>T7</td>
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<td>laminectomy</td>
<td>paraparesis/ improved; urinary incontinence/ persisted</td>
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<td>syringomyelia at C6-T3</td>
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<td>1st: not improved; 2nd: paraparesis was slightly improved</td>
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<td>Tumialan et al. (2005)</td>
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<td>unknown</td>
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<td>laminectomy and microlysis of adhesion, cyst-peritoneal shunt</td>
<td>paraparesis/ improved</td>
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<td>Nakata et al. (2006)</td>
<td>VA-PICA</td>
<td>ligation of VA and OA-PICA anastomosis</td>
<td>6 mos</td>
<td>T3-T4</td>
<td>presyrinx state at T3</td>
<td>syringo-subarachnoid shunt</td>
<td>paraparesis/ persisted; paresthesia/ improved</td>
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<td>Eneling et al. (2012)</td>
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<td>18 mos</td>
<td>T3-T5</td>
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<td>at least T4-T9</td>
<td>syringomyelia at C1-T11</td>
<td>laminectomy and microlysis of adhesion</td>
<td>tetraparesis, urinary incontinence, and sensibility deficit/ persisted</td>
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nerve roots resulted in the improvement of paresthesia with pain in this case. Our strategy might be reasonable as treatment for severe cases with syringomyelia and multiple arachnoid cysts due to adhesive arachnoiditis. A previous case was successfully treated with expansive laminoplasty and duraplasty using a Gore-Tex membrane. We agree with this strategy, because the essential aim of the treatment is normalization of CSF flow and one of the key points for good outcome is to avoid the recurrence of adhesive arachnoiditis. However, we performed duraplasty without Gore-Tex to avoid surgical site infection due to the usage of steroids for Sjögren’s syndrome. If the syrinx size increases in follow-up examinations, we plan to implement the shunt procedure. However, discussion regarding the best treatment method for syringomyelia and arachnoid cysts associated with arachnoiditis is still underway. The treatment strategy should be decided on the basis of the extent of syringomyelia and adhesive arachnoiditis. In addition, clarification of the symptom to be improved and the lesion thought to cause the symptom are most important. For severe cases as in the present patient, the surgical treatment should be focused on the lesion which contributes to motor function, especially arachnoid cysts in this case. Extensive arachnoid scarring may be strongly associated with poor clinical outcomes. Needless to say, early-stage diagnosis is most important for a good clinical outcome.

In conclusion, syringomyelia and arachnoiditis should be recognized as late complications of SAH. Severe cases with extensive arachnoiditis can be treated with laminectomy and microlysis of the adhesion. Our surgical strategy focusing on the lesion thought to cause the symptom, especially motor weakness, may be useful for severe cases.

References


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