

Spinal syringomyelia following subarachnoid hemorrhage

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ABSTRACT

Subarachnoid blood has been reported as a cause of chronic spinal arachnoiditis. Although syringomyelia has been thought to be caused by spinal arachnoiditis, reports of syringomyelia following aneurysmal subarachnoid hemorrhage (SAH) are very rare. We describe two patients with syringomyelia associated with chronic spinal arachnoiditis following SAH. From January 2001 to December 2010, 198 patients with aneurysmal SAH were treated at Kinki University School of Medicine. Two of the 198 patients had syringomyelia following aneurysmal SAH; thus the rate of syringomyelia associated with aneurysmal SAH was 1.0%. Patient 1 was a 54-year-old woman who presented with back pain, back numbness and gait disturbance 20 months after SAH. Her MRI revealed syringomyelia of the spinal cord from C2 to T10. She underwent shunting of the syrinx to the subarachnoid space. Patient 2 was a 49-year-old man, who was admitted to the hospital with headache, diplopia, hoarseness, dysphagia and ataxia five months after SAH. MRI revealed syringomyelia from the medulla oblongata to C6, and an enlargement of the lateral and fourth ventricles. After foramen magnum decompression and C1 laminectomy, a fourth ventricle–subarachnoid shunt was placed by insertion of a catheter. Spinal arachnoiditis and spinal syringomyelia are rare but important chronic complications after SAH.

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1. Introduction

Subarachnoid blood has been reported as a cause of chronic spinal arachnoiditis.^{1–4} Although syringomyelia has been proposed as being caused by spinal arachnoiditis, reports of syringomyelia following aneurysmal subarachnoid hemorrhage (SAH) are very rare.⁵ We describe two patients with syringomyelia associated with chronic spinal arachnoiditis following SAH.

2. Case Reports

From January 2001 to December 2010, 198 patients with aneurysmal SAH were treated at Kinki University School of Medicine. Two of 198 patients developed syringomyelia following aneurysmal SAH. Thus, the rate of syringomyelia associated with aneurysmal SAH in our patients was 1.0%.

2.1. Patient 1

A 54-year-old woman was admitted to the hospital with a sudden onset of severe headache. A head CT scan demonstrated diffuse SAH. The SAH was classified as Hunt and Hess grade II, and the CT findings were consistent with Fisher grade 3. The patient underwent three-dimensional-CT angiography, which revealed an aneurysm of the left middle cerebral artery (MCA). The aneurysm was clipped successfully via the left pterional approach. The patient was discharged and returned home with a mild right hemiparesis from vasospasm, with no hydrocephalus.

Twenty months after the SAH, the patient developed back pain, back numbness and gait disturbance. On examination, the patient exhibited a paraparesis that involved the right leg more than the left, and a sensory disturbance from C7 to T10. Deep tendon reflexes were hyperactive in both lower extremities. Her MRI revealed syringomyelia of the spinal cord from C2 to T10 (Fig. 1). After laminectomy and durotomy from C4 to T1, a catheter was carefully inserted as a syringo–subarachnoid shunt through a small

midline myelotomy. At the last examination, five years after surgery, the patient was still experiencing incomplete recovery from back pain, back numbness and gait disturbance. MRI of the spine showed significant reduction of the syringomyelia.

2.2. Patient 2

A 49-year-old man was admitted to the hospital after a sudden-onset consciousness disturbance. A head CT scan demonstrated SAH predominantly in the posterior fossa (Fig. 2). The SAH was classified as Hunt and Hess grade III, and the CT findings were consistent with Fisher grade 3. After external ventricular drainage had been established, the patient underwent four-vessel cerebral angiography, which revealed a left vertebral artery (VA) dissecting aneurysm of the proximal posterior inferior cerebellar artery. Embolization of the left VA was performed by interventional radiology. Later, the patient underwent insertion of a ventriculoperitoneal (VP) shunt. He was discharged home without neurological deficits.

Five months after the SAH, the patient had headache, diplopia, hoarseness, dysphagia and ataxia. MRI revealed syringomyelia from the medulla oblongata to C6, and enlargement of the lateral and fourth ventricles. The VP shunt was changed to a low-pressure system, and the symptoms improved. Eighteen months after the SAH, the patient complained of progressively worsening symptoms. Upon examination, his motor strength was reduced in both upper extremities (manual muscle test [MMT] grade 3/5) and lower extremities (MMT grade 4/5). There were disturbances of cranial nerves VI, IX, and X, bilaterally. Deep tendon reflexes were hyperactive in both upper and lower extremities. MRI revealed syringomyelia of the spinal cord from the medulla oblongata to C6, an enlarged fourth ventricle and an arachnoid cyst in the cisterna magna (Fig. 3). After foramen magnum decompression and C1 laminectomy, a fourth ventricle–subarachnoid shunt was performed by insertion of a catheter. At the last examination, two years after surgery, the patient's symptoms of dysphagia and hoarseness were improved, but he was still experiencing incomplete recovery from diplopia and ataxia. MRI of the spine showed significant reduction of the syringomyelia.

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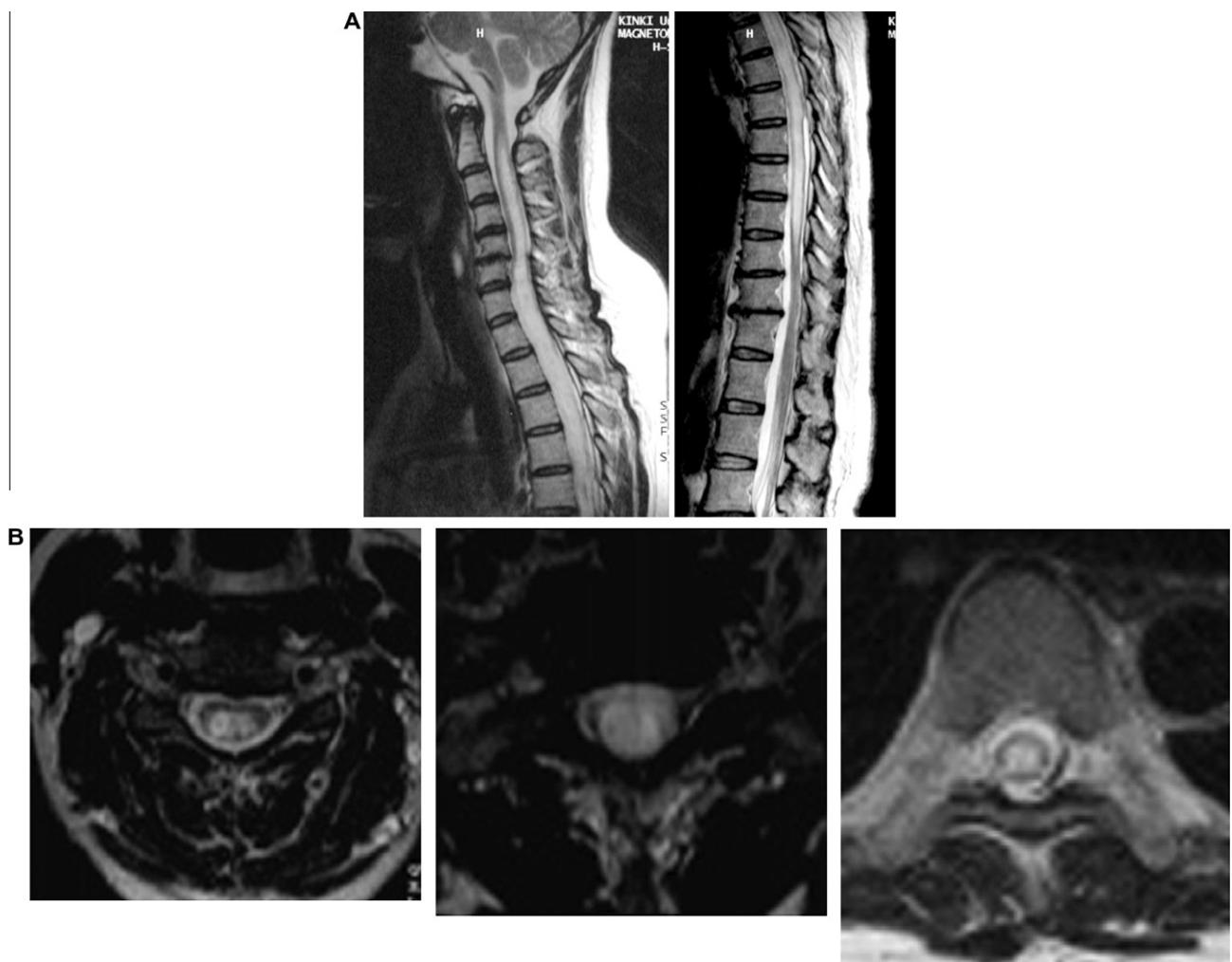


Fig. 1. Patient 1. (A) Sagittal T2-weighted cervicothoracic MRI and (B) axial T2-weighted MRI showing syringomyelia of the spinal cord from C2 to T10.

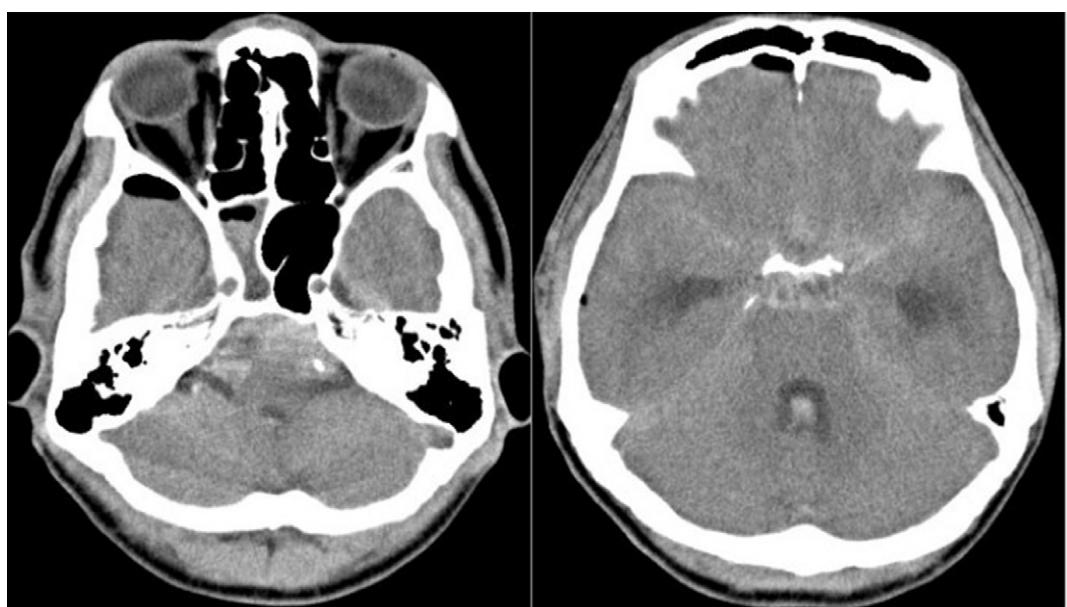


Fig. 2. Patient 2. Axial CT scans on admission showing subarachnoid hemorrhage predominantly in the posterior fossa.

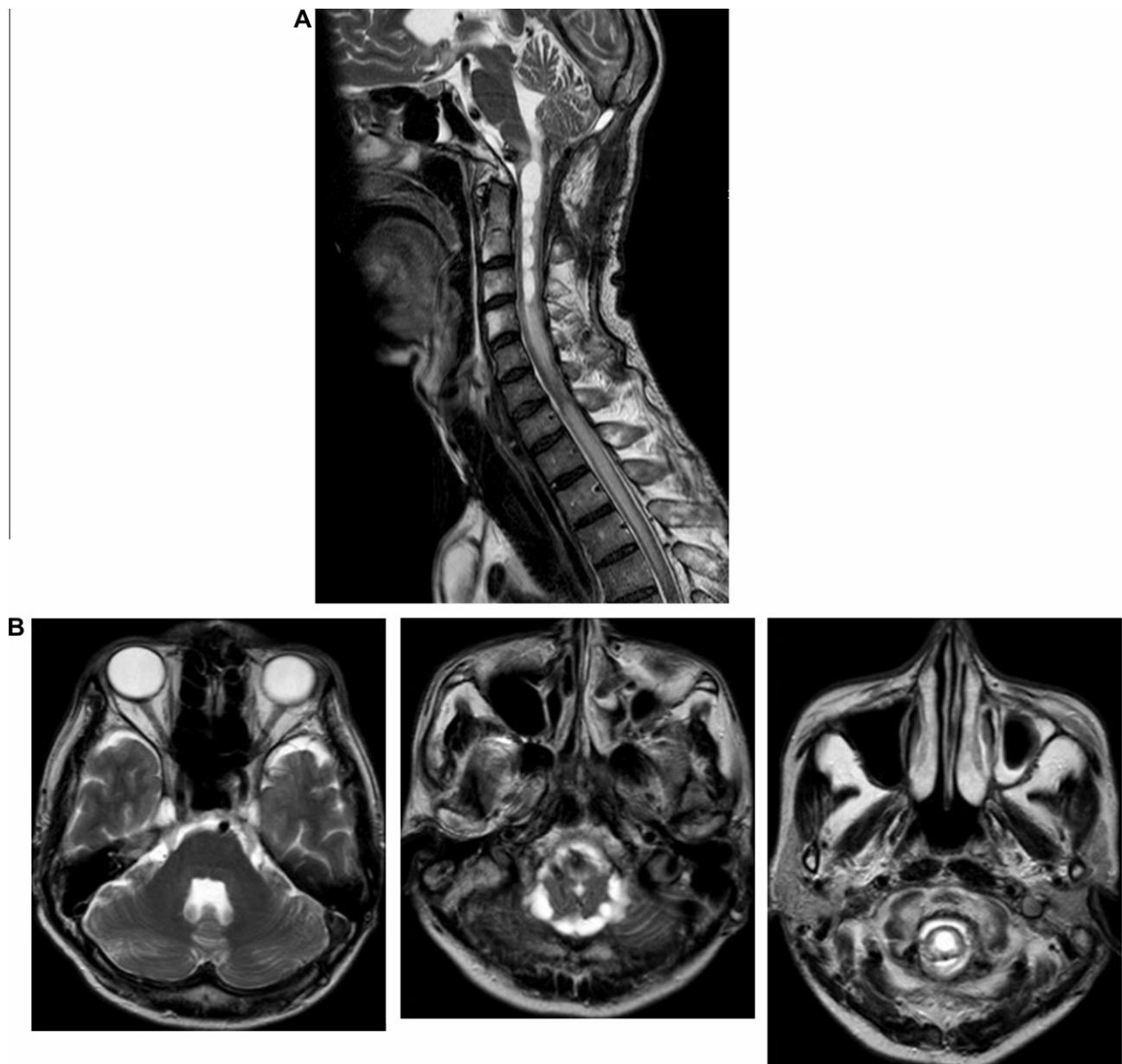


Fig. 3. Patient 2. (A) Sagittal T2-weighted MRI showing syringomyelia of the spinal cord from the medulla oblongata to C6 and (B) axial T2-weighted MRI showing syringomyelia of the spinal cord from the medulla oblongata to C6 (right), enlargement of the fourth ventricle (left) and an arachnoid cyst in the cisterna magna (middle).

3. Discussion

Spinal arachnoiditis or arachnoid scarring may be associated with syringomyelia. However, the pathogenic significance of arachnoid scarring has not been generally acknowledged, although most authors report arachnoid adhesions at the level of the spinal injury in patients with post-traumatic syringomyelia.^{6–9}

Hemolysis of blood from a ruptured aneurysm in the subarachnoid space incites an inflammatory response that chronically irritates the leptomeninges in the post-hemorrhagic period. The fibroproliferative reaction that ensues in the intrathecal compartment may lead to arachnoiditis anywhere in the neuroaxis, with the potential subsequent formation of syringomyelia in some patients.^{10–13} Parker et al.¹⁴ reported a retrospective study of 32 patients treated for syringomyelia associated with non-traumatic arachnoid scarring. Among these 32 patients, three had suffered from SAH. Klekamp et al.⁵ reported a retrospective study of 107 patients treated for syringomyelia associated with arachnoid scar-

ring. In 52 patients, the arachnoid scarring was related to spinal trauma, whereas 55 had no history of trauma and developed arachnoid scarring as a result of an inflammatory reaction. Of the 107 patients, one suffered from an SAH. There is no report of the incidence rate of syringomyelia following aneurysmal SAH. Our results indicate that two of 198 patients (1.0%) had syringomyelia following aneurysmal SAH. Spinal arachnoiditis is generally reported as a complication of SAH in fewer than 1% of patients.^{3,10,14} The complication rate of spinal syringomyelia after SAH is also usually reported as less than 1% of patients. Kok et al.¹⁰ reviewed 14 patients with spinal arachnoiditis after SAH. Ten (four males and six females, of mean age 52 years [range, 22–69 years]) of the 14 were well described, and all displayed a posterior circulation aneurysm. Hydrocephalus was a complication in four patients. The duration from SAH to spinal arachnoiditis was one month to 4.8 months (mean, 9.3 months). Risk factors may include posterior circulation aneurysms, the extent and severity of the hemorrhage, and the need for cerebrospinal fluid (CSF) diversion.¹⁵ Spinal arach-

noiditis after SAH was not exclusively associated with posterior circulation aneurysms. Spinal arachnoiditis after an MCA aneurysm was a complication in our patient 1.

Shunting of the syrinx to the subarachnoid, pleural, or peritoneal space is still widely accepted as the treatment of choice for patients with syringomyelia caused by arachnoiditis.⁵ Shunting of the syrinx to the subarachnoid or peritoneal cavity was associated with a recurrence rate of 60%, whereas microsurgical dissection of the arachnoid scar and decompression of the subarachnoid space had a recurrence rate of 33%, with a mean follow-up period of 28 months.¹⁴ Successful long-term management of the syrinx was associated with basal or focal spinal arachnoid scarring, no history of spinal meningitis, microsurgical dissection of the arachnoid scar and decompression of the subarachnoid space.¹⁴ Cacciola et al.¹⁶ reported 20 patients with syringomyelia refractory to CSF flow restoration who underwent a procedure for placement of a syringopleural shunt. Follow-up MRI revealed syrinx collapse in 17 patients and a marked shrinkage in two. They recommended that a syringopleural shunt should be the syrinx diversion procedure of choice.

4. Conclusions

Two of 198 patients had syringomyelia following aneurysmal SAH. Thus, the rate of occurrence of syringomyelia was 1.0% following aneurysmal SAH. Spinal arachnoiditis and spinal syringomyelia are important chronic complications after SAH.

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Acute infarction in a patient with agenesis of the internal carotid artery and transcaavernous anastomosis

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ABSTRACT

A 70-year-old man presented with left-sided weakness and dysarthria. His brain MRI and magnetic resonance angiography demonstrated acute infarctions in the bilateral middle cerebral artery territories in a setting of agenesis of the right internal carotid artery (ICA) with transcaavernous anastomosis, a rare developmental anomaly. In this setting, atherosclerotic disease in the unilateral common carotid artery or ICA can induce acute infarctions in the bilateral anterior circulation.

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