Arachnoid cyst with associated arachnoiditis developing after subarachnoid hemorrhage

Case report

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The authors report the case of a 53-year-old woman in whom a T1–T2 spinal arachnoid cyst with associated arachnoiditis developed, compressing the thoracic spinal cord 1 year after the patient had suffered a Hunt and Hess Grade IV subarachnoid hemorrhage (SAH). Development of spinal arachnoiditis with or without an arachnoid cyst is a rare complication of aneurysmal SAH. Risk factors may include posterior circulation aneurysms, the extent and severity of the hemorrhage, and the need for cerebrospinal fluid diversion. Surgical drainage, shunt placement, or cyst excision, when possible, is the mainstay of treatment.

KEY WORDS • spinal arachnoid cyst • spinal arachnoiditis • posterior inferior cerebellar artery aneurysm • subarachnoid hemorrhage

Hemolysis of blood from a ruptured aneurysm in the subarachnoid space incites an inflammatory response that chronically irritates the leptomeninges in the posthemorrhage period. The fibroproliferative reaction that ensues in the intrathecal compartment may lead to arachnoiditis anywhere in the neuroaxis, with the potential subsequent formation of an arachnoid cyst in some patients. Spinal arachnoid cysts and arachnoiditis are exceptionally rare complications observed after SAH. Nevertheless, it is important to recognize these entities because the patient’s symptoms may be misinterpreted as resulting from shunt malfunction or needing CSF diversion. The formation of arachnoiditis in conjunction with an arachnoid cyst may represent a broad clinical spectrum dependent on the extent and location of compression of the spinal cord. The true prevalence of arachnoiditis with or without arachnoid cysts after SAH is not presently known because some patients may have subclinical manifestations of these entities. Fourteen cases have previously been reported in the literature. We describe the case of a 53-year-old woman in whom arachnoiditis and an associated arachnoid cyst developed, causing spinal cord compression after the rupture of a posterior circulation aneurysm. Surgical decompression of the arachnoid cyst markedly improved the patient’s symptoms.

Case Report

History. This 53-year-old woman with a medical history significant for polycythemia vera presented to another hospital with severe headache, nausea, and vomiting. An unenhanced CT scan of her head demonstrated diffuse SAH. On arrival at our institution, the patient’s condition acutely deteriorated and she became unresponsive, requiring intubation and mechanical ventilation. An unenhanced CT scan of the head demonstrated evidence of repeated hemorrhage with SAH extending into the fourth ventricle. The CT scan was suggestive of a posterior circulation aneurysm and associated hydrocephalus. The SAH was assigned Hunt and Hess Grade IV and the CT findings were consistent with a Fisher Grade 3. After external ventricular drainage had been established, the patient underwent four-vessel cerebral angiography, which revealed a 3.3 × 3.5 × 3-mm fusiform aneurysm of the proximal left PICA. The aneurysm was not amenable to coil embolization and, therefore, a left suboccipital craniotomy and transcervical approach for clip ligation of the lesion was performed. The dura mater was closed with bovine pericardium and Tisseal. The patient remained in the hospital for a prolonged time postoperatively because her recuperation was complicated by vasoconstriction requiring chemical and mechanical angioplasty. She also required prolonged ventricular drainage. At no time

Abbreviations used in this paper: CSF = cerebrospinal fluid; CT = computerized tomography; MR = magnetic resonance; PICA = posterior inferior cerebellar artery; SAH = subarachnoid hemorrhage.
Arachnoid cyst after subarachnoid hemorrhage during her entire hospital course did she undergo placement of a lumbar drain. She was discharged to a rehabilitation facility 30 days after admission.

The patient returned 3 months later with persistent nausea, vomiting, and gait disturbances. She was found to have hydrocephalus and underwent placement of a ventriculoperitoneal shunt. By 4 months after her initial hemorrhage, the patient had made a full recovery. She was living independently and was fully ambulatory.

Examination. The patient returned 1 year after her initial hemorrhage complaining of progressively worsening gait. She was hyperreflexic in the lower extremities with 4/5 strength in all muscle groups in the right leg and 4–5 strength in the left leg. She exhibited decreased proprioception in the left lower extremity. No sensory level dissociation could be appreciated. She had no upper extremity involvement and normal bowel and urinary function. Her ventriculoperitoneal shunt was found to be fully functional and a complete metabolic workup yielded normal findings.

An MR image of the cervical and thoracic spine demonstrated an intradural extramedullary collection of CSF extending along the ventral surface of the cervical and thoracic spinal cord from C-7 to T-2 before turning dorsolaterally to the left at T-3 (Fig. 1). There was a series of isolated loculations of CSF in the subarachnoid space at T-3. Diffuse enhancement of the spinal cord was also observed, which was consistent with the presence of arachnoiditis (Fig. 2).

Operation. The T1–2 levels were marked using fluoroscopy and a linear incision was made over these levels. Standard laminectomies of T1–2 were performed with a left lateral extension to the pedicles, thus enabling the surgeons to reach the ventral component of the lesion. With the aid of the operating microscope, the dura was opened. Immediately obvious was the extent of arachnoid scarring in this area—a sign consistent with arachnoiditis. There were multiple loculated arachnoid cysts, which extended ventrally from the dorsal aspect of the cord, causing an obvious mass effect on the spinal cord. With the aid of somatosensory evoked potential monitoring, the dentate ligaments were divided and the spinal cord was mobilized. Dense fibrous adhesions prevented total excision, but fenestration of the arachnoid cyst in several areas was possible. The largest loculation, ventral to the T-2 vertebral body and eccentric to the left, was treated by creating a shunt into the subarachnoid space. After the arachnoid cysts had been fenestrated and a shunt placed, the entire spinal cord relaxed markedly into its normal position. The dura was reapproximated in a watertight fashion with Tisseal and Durarepair, and the wound was closed in the usual multilayered fashion.

Postoperative Course. Immediately after surgery the patient was found to have normal reflexes and improved muscle strength. On walking, she noted resolution of her gait and proprioceptive disturbances. After an uncomplicated hospital course, she was discharged home. Postoperative imaging demonstrated decompression of the arachnoid cyst (Fig. 3). Seven months postoperatively, the patient demonstrates marked improvement in her gait disturbance and muscle strength.

Discussion

Arachnoid cyst formation is a rare yet potentially devastating complication of SAH.\(^3,12\) Despite the continually growing number of patients successfully treated every year for aneurysmal SAH, both surgically and endovascularly, there has not been a parallel rise in the number of SAH-induced spinal arachnoid cysts or arachnoiditis. In fact, the literature remains sparse regarding this disease entity in patients in whom SAH has occurred, with only 15 patients with spinal arachnoiditis described in the literature, six of whom also had associated arachnoid cysts.\(^2,10,13\)

Typically, acquired arachnoid cysts arise in the context of trauma, infection, and surgery.\(^4,7\) It is thought that these cysts are formed from discrete pockets of CSF that have been loculated within the arachnoid membranes. Locations may include intradural, extradural, perineural, intracranial, or intraspinal areas.\(^10\) Cyst enlargement results from the continuous production of CSF within these loculated arachnoid membranes.\(^4\) This may result in the displacement of surrounding structures, thereby producing symptoms. The formation of these cysts in patients who have suffered aneurysmal SAH is poorly understood.\(^9\)

If subarachnoid blood is the cause of spinal arachnoiditis with arachnoid cyst formation, one would expect a higher prevalence of patients exhibiting symptoms of this disease process. This observation has prompted investigators to propose alternate causes for the spinal arachnoiditis seen in

![Image of MRI scans](attachment:103x150 to 559x275.png)

**FIG. 1.** Magnetic resonance images of the thoracic spine revealing an arachnoid cyst. Left: Sagittal T\(_1\)-weighted image without administration of Gd demonstrating a large ventral arachnoid cyst (arrow). Center: Sagittal T\(_2\)-weighted image depicting dorsal loculated arachnoid cysts (small arrow) and areas of arachnoiditis (large arrowhead). Right: Axial T\(_2\)-weighted image demonstrating that the ventral component of the arachnoid cyst is causing a mass effect on the spinal cord at the level of the T-2 vertebral body.
their patients with SAH. Taguchi and colleagues\textsuperscript{16} discussed a patient who underwent surgical clip ligation for a ruptured PICA aneurysm and was found 6 months after surgery to have arachnoiditis with an associated arachnoid cyst, possibly caused by fibrin glue. Operative findings of granulation tissue and residual bone chips were confirmed histologically. Although this may be a plausible explanation, Marcoux and associates\textsuperscript{13} describe the formation of an intracranial arachnoid cyst 18 months after coil embolization of a ruptured anterior communicating artery aneurysm. Obviously no fibrin was used in this patient. Treatment of this patient subsequently involved surgery in which the cyst was fenestrated along with the lamina terminalis. These authors wonder whether concomitant opening of subarachnoid spaces during surgery would have prevented the formation of the cyst in their patient by facilitating drainage of blood.

Lorenzana-Honrado, et al.,\textsuperscript{12} reported the histological examination of an arachnoid cyst in a patient who experienced a Hunt and Hess Grade III SAH, which was treated by surgical clip ligation of a left PICA aneurysm. The patient returned to the hospital with lower extremity myelopathy and urinary retention. A histological study of this cyst revealed lysed erythrocytes and leukocytic infiltrate. The patient’s symptoms resolved after implantation of a cystoperitoneal shunt.

At our institution, we have treated more than 1000 patients with SAH during the last decade; a significant number of these patients harbored posterior circulation aneurysms. Nevertheless, the patient we describe in this paper remains the only one we have identified as suffering from symptoms of an arachnoid cyst. Nonetheless, the adage “absence of proof is not proof of absence” applies here. It is a well-established fact that hemolysis of blood in the subarachnoid space begins an ill-defined cascade of events contained within the thecal compartment, which may lead to vasoconstriction and a fibroproliferative reaction and ultimately to arachnoiditis.\textsuperscript{8,14,15} Furthermore, the anesthesia literature\textsuperscript{1,2} provides ample examples of arachnoiditis forming after blood patches in patients who have had epidural anesthesia.\textsuperscript{12} With the amount of blood that circulates into the intrathecal cranial compartment after a ruptured aneurysm, as demonstrated by Klimo and colleagues\textsuperscript{8} a similar inflammatory response may occur in the intrathecal spinal compartment in the patient who has SAH. This may be especially true in the critically ill patient in whom CSF tends to pool in dependent areas of the thecal sac, specifically the thoracic spine.\textsuperscript{9} The fact that subclinical elements of arachnoiditis or arachnoid cysts may be present in many patients with SAH who never come to the attention of the clinician may account for the limited number of reported cases.\textsuperscript{3} Furthermore, most patients harboring arachnoid cysts have no symptoms.\textsuperscript{11}

The review by Kok and associates\textsuperscript{9} identifies certain risk factors in patients who have had SAH-induced arachnoiditis with or without an arachnoid cyst. Patients in whom posterior circulation aneurysms have resulted in severe hemorrhages that required CSF diversion and prolonged intensive care courses appear to be at an increased risk. It is in these patients that spinal arachnoiditis, with or without an associated cyst, should be considered as a possible cause for para-

![Image](https://example.com/image1)

**Fig. 1.** Sagittal Gd-enhanced T\textsubscript{1}, weighted MR image depicting diffuse leptomeningeal enhancement (arrows) consistent with the presence of arachnoiditis.

![Image](https://example.com/image2)

**Fig. 2.** Sagittal Gd-enhanced T\textsubscript{1}, weighted MR image depicting diffuse leptomeningeal enhancement (arrows) consistent with the presence of arachnoiditis.

![Image](https://example.com/image3)

**Fig. 3.** Postoperative MR images of the thoracic spine demonstrating resolution of the arachnoid cyst. Left: Sagittal T\textsubscript{2}, weighted image displaying resolution of the ventral component of the arachnoid cyst. Right: Axial T\textsubscript{2}, weighted image depicting resolution of the mass effect on the spinal cord at the level of the T-2 vertebral body.

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paresis, upper motor neuron signs, and myelopathy. Surgical decompression in the form of complete excision when possible, fenestration, or placement of a shunt is the treatment of choice when there is a mass effect on the spinal cord.7,10–12,17

Conclusions

Although rare, formation of an arachnoid cyst is a potentially devasting complication of SAH. In patients with a history of SAH, especially those with posterior circulation aneurysms, the possibility of an arachnoid cyst should be considered as a cause of progressive gait dysfunction and myelopathy. Surgical decompression is indicated when there is mass effect on the spinal cord.

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References


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