Spontaneous Spinal Subarachnoid Hemorrhage with Development of an Arachnoid Cyst—A Case Report and Review of the Literature

Julia Velz[1,2], Jorn Fierstra[1,2], Luca Regli[1,2], Menno R. Germans[1,2]

Key words
- Review of the literature
- Spinal subarachnoid hemorrhage
- Thoracic arachnoid cyst

Abbreviations and Acronyms
MRI: Magnetic resonance imaging
SAC: Spinal arachnoid cyst
SAH: Subarachnoid hemorrhage

From the [1]Department of Neurosurgery, Clinical Neuroscience Center, University Hospital Zurich; and [2]University of Zurich, Zurich, Switzerland

To whom correspondence should be addressed: Menno R. Germans, M.D., Ph.D. [E-mail: menno.germans@usz.ch]

Citation: World Neurosurg. (2018) 119:374-380. https://doi.org/10.1016/j.wneu.2018.08.108

Journal homepage: www.WORLDNEUROSURGERY.org

Available online: www.sciencedirect.com

BACKGROUND: **Spontaneous spinal subarachnoid hemorrhage (SAH) is extremely rare and occurs in <1% of all cases of SAH. To the best of our knowledge, only 1 case of spontaneous spinal SAH with later development of a symptomatic spinal arachnoid cyst (SAC) has been described in the literature to date. The objective of the present study was to report the challenging clinical management of SAC based on a literature review.**

CASE DESCRIPTION: **We report the case of a 51-year-old woman with acute onset of back pain, neck pain, and headaches with an angiogram-negative supratentorial SAH. Further magnetic resonance imaging screening of the spine revealed additional subarachnoid blood at the level of the thoracic spine, without evidence of vascular malformations. Several weeks after the hemorrhage, the patient developed progressive numbness in her trunk and lower extremities and weakness in her lower extremities. A follow-up magnetic resonance imaging study revealed a large arachnoid cyst at level T4—T7 with spinal cord compression. The patient underwent left hemilaminectomy at T4—T6 and fenestration of the SAC. In a second surgery, right-sided hemilaminectomy at T7 was performed with complete marsupialization of the larger cyst and placement of an intradural shunt. At the 6-month clinical follow-up examination, she showed improvement of her clinical symptoms.**

CONCLUSIONS: **Treatment of secondary SAC is challenging, and surgery of the SAC with or without placement of a shunt is a possible treatment option.**

INTRODUCTION

In 1943, Nelson[1] described the first patient with a symptomatic spinal arachnoid cyst (SAC) 4 years after subarachnoid hemorrhage (SAH). Chronic inflammation in the pia-arachnoid space leading to arachnoiditis is widely believed to be the cause of SACs.[2] Arachnoiditis can vary from mild arachnoidal thickening to extensive adhesions of the pia-arachnoid space and formation of a SAC.[2] Although formation of SACs secondary to inflammatory reactions related to meningitis, trauma, and intradural surgery has been commonly described, the formation of SACs after SAH is rare.[2,3] SACs display a rare cause of spinal cord compression and should be considered when patients develop new spinal cord deficits after SAH.[4,5] Spontaneous spinal SAH is extremely rare and occurs in <1% of all cases of SAH.[6] To the best of our knowledge, only 1 case of spontaneous spinal SAH with later development of a symptomatic SAC has been described in the reported data to date.[4]

The rare incidence of spinal SAH with subacute hemorrhage in a SAC and the challenging clinical management prompted us to report the present case and discuss the current management of SAC based on a literature review.

CASE DESCRIPTION

A 51-year-old woman was admitted to the Department of Neurosurgery, University of Zurich, with an episode of sudden-onset severe back and neck pain accompanied by headaches 1 week earlier. The patient provided written informed consent for the report of her case. The patient had a known ankylosing spondylitis without other significant medical history. On neurological examination, no functional deficits were detected. The computed tomography images at presentation showed some SAH at the left occipital lobe and in the pontomedullary cistern (Figure 1A). The diagnosis of SAH was confirmed by positive findings from a lumbar puncture. The cranial magnetic resonance imaging (MRI) examination showed bilateral hemosiderin deposits in the parieto-occipital sulcus (Figure 1B). Time-of-flight magnetic resonance angiography showed that the vessels of the anterior circulation, in particular, the anterior cerebral artery in the A1 and A2 segments had caliber irregularities, highly suspicious of vasospasms (Figure 1C). Time of flight angiography and digital subtraction angiography revealed no evidence of vascular malformations, in particular, no intracranial aneurysm or vasculitis. It was then decided to perform an additional MRI examination of the complete spinal axis, which showed localized subarachnoid blood at the level of T5—T7 (Figure 2). The mass effect had caused the spinal cord to shift slightly anteriorly without significant compression. Magnetic resonance angiography of the spine revealed no evidence of a vascular malformation. A differential diagnosis of vasculitis (drug-induced systemic lupus

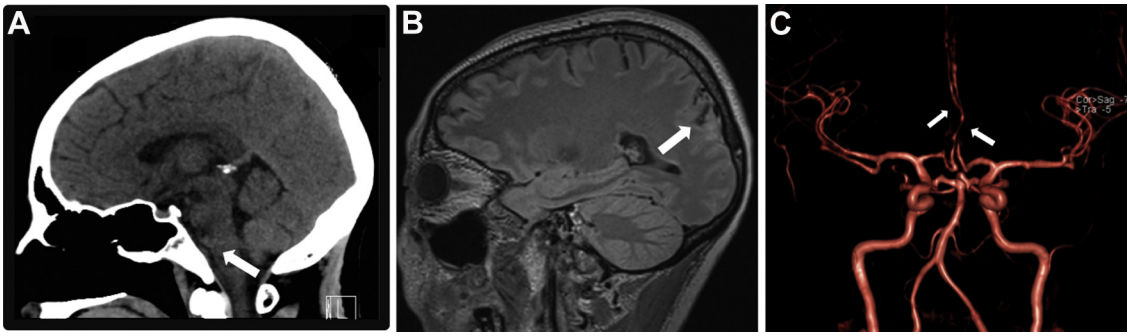

Figure 1. (**A**) Sagittal section of computed tomography scan showing small amounts of subarachnoid hemorrhage in the pontomedullary cistern (*arrow*). (**B**) Sagittal section of fluid-attenuated inversion recovery magnetic resonance imaging study showing blood in the parieto-occipital sulcus (*arrow*). (**C**) Time-of-flight angiogram showing caliber irregularities in the vessels of the anterior cerebral artery, highly suspicious for angiographic vasospasm (*arrow*).

erythematosus due to adalimumab) was considered; however, the serology findings were negative. Conservative treatment in accordance with our institutional SAH protocol was performed without further therapeutic intervention, other than the initial lumbar puncture. At discharge (17 days after hospitalization), the patient showed improvement of her neck and back pain and her headaches. She was discharged to home without any neurological deficits.

Three months later, the patient presented at our outpatient clinic with numbness in her trunk and lower extremities and weakness in her lower extremities without sphincter dysfunction. She reported the development of slowly progressive neurological symptoms since the day of discharge. The neurological examination revealed a strength of 4+ of 5 in both legs using the Medical Research Council grading system, a sensory deficit below T10 with dysesthesia, and increased reflexes in the legs. MRI of the spine revealed large, multiple, and septated arachnoid cysts at the T4–T7 level with a mass effect on the spinal cord. A focal T2-weighted imaging hyperintense signal was seen at T6–T8, indicative of myelopathy (**Figure 3**). The progressive neurological deterioration and radiological evidence of myelopathy prompted us to treat the cysts surgically. Left-sided hemilaminectomy at T4–T6 and marsupialization of multiple cysts with cranial fenestration of the larger arachnoid cyst at the level of T7 was performed 4 months after the initial SAH. Postoperatively, the patient showed an initial improvement of her neurological deficits. However, in the subsequent weeks, she developed new neurological symptoms, with weakness of her lower extremities, back pain, and gait difficulties. A repeat MRI examination of the thoracic spine revealed a recurrent SAC at the T4–T7 level with signs of spinal cord compression. Another surgery was elected, and right-sided hemilaminectomy at T7 was performed, with complete marsupialization of the larger cyst and introduction of an intradural shunt crossing the cyst without complications. At the 6-month follow-up examination, the patient showed a slight improvement in her preoperative neurological deficits. The neurological examination revealed a sensory deficit below T10 and muscle strength of 4 of 5 using the Medical Research Council grading system

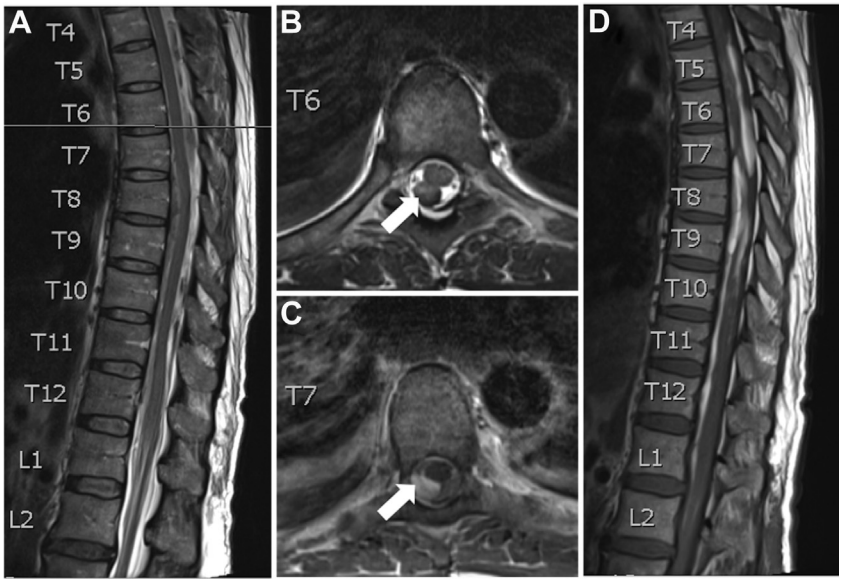

Figure 2. Magnetic resonance images of the midthoracic spine after angiogram-negative subarachnoid hemorrhage (SAH). (**A**) Sagittal and (**B**) axial T2-weighted and (**C**) axial and (**D**) sagittal T1-weighted images. SAH at the T5–T7 level had caused the spinal cord to shift slightly anteriorly and, at the T8-T9 level, slightly posteriorly, without significant compression. *White arrows* indicate the localized clot in the subarachnoid cavity.