Arteriovenous Malformation Mimicking Subarachnoid Hemorrhage Complicated by Spinal Cord Compression

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Abbreviations used in this report: Arteriovenous malformation (AVM); subarachnoid hemorrhage (SAH), spinal dural arteriovenous fistula (SDAVF); left upper quadrant (LUQ); magnetic resonance (MR); computed tomography (CT)

ABSTRACT
47-year-old hypertensive male presented with sudden onset of severe neck pain. Physical examination revealed neck stiffness and no neurologic deficit. CT scan of head and neck revealed scant subarachnoid blood. The patient was treated for subarachnoid hemorrhage and was placed under observation due to severe neck pain. Two days after admission, he started complaining of urinary retention and weakness in lower extremities. At that point, hypoesthesia to pinprick at T6-7 level was observed. MRI of the thoracic spine revealed a hematoma at T6-7 level and a type I arteriovenous malformation. The patient underwent hematoma drainage and resection of the AVM. This is an unusual presentation of type I AVM that mimicked an intracranial SAH followed by an acute spinal cord compression. AVM must be considered as a cause of SAH especially if there is a discrepancy between the symptoms and the amount of blood found in the head CT.

INTRODUCTION
Spinal arteriovenous malformations (AVM) are a rare cause of non-traumatic subarachnoid hemorrhage. Up to 10% of the spinal AVMS can present as SAH. They are classified according to their anatomical location and vascular characteristics. The most common type is spinal dural arteriovenous fistula (SDAVF), presenting usually in the fifth decade of life and being most common in men. The usual presentation is progressive myeloradiculopathy with paraparesis, sensory disturbances and sphincter dysfunction.

We present a case of SAH secondary to a ruptured SDAVF that presented with severe abdominal and neck pain complicated with sudden onset of paresis, and with segmental sensory loss and sphincter dysfunction secondary to spinal cord compression.

CASE PRESENTATION
A 47-year-old Latin American man with history of hypertension presented to the emergency room with 4-day history of sudden onset of left upper quadrant (LUQ) abdominal pain. The pain was described as a severe stabbing pain, exacerbated by movement. The pain was associated with nausea and vomiting and it was radiated to the back, lower extremities and neck. The abdominal pain began to recede within 5 hours of its onset, while the neck pain persisted associated with neck stiffness and with a mild constant frontal headache.

On arrival to the emergency room, the patient was noted to have severe nuchal rigidity and cervical pain that limited his body movements. The blood pressure was 222/103 mmHg, and the heart rate of 66 beats/min. There were no cognitive or cranial nerve abnormalities. There was neither focal weakness nor sensory deficit. A computerized tomography (CT) scan of the head without contrast revealed a hyperdensity of the occipital sulci and posterior interhemispheric line due to a mild SAH (Figure 1). A lumbar puncture confirmed the diagnosis of SAH. A magnetic resonance (MR) angiogram of the brain ruled out the presence of any aneurysm or AVM. The patient remained stable for the next 48 hours, but he continued complaining of persistent severe
vere cervical pain with partial relief with morphine. Two days after hospitalization the patient started complaining of inability to urinate and weakness of lower extremities. On physical exam the patient had paraparesis and decreased anal sphincter tone. There was a sensory level of hypoesthesia at T6-T7 level. High dose dexamethasone was given immediately, and an MR imaging of the thoracic spine (Figure 2) showed an intradural hematoma localized in the left posterolateral aspect of the spinal canal producing ventral displacement and compression of the spinal cord at the T6 level and a flow-void phenomena lateral to the spinal cord at the T5 level consistent with a SDAVF.

Figure 2. Intradural hematoma at the level of T6-T7.

A spinal angiogram was performed to confirm the presence of a left DAVF. The patient was taken to the operating room for an emergency laminectomy with hematoma drainage and decompression of the spinal cord.

After the procedure, the patient experienced an improvement of the strength in the lower extremities and of the anal sphincter tone. Residual paresis and sensory deficit persisted.

**DISCUSSION**

Dural arteriovenous fistulas are a rare cause of SAH. We only found 17 cases described in the literature, most of them occurring in the cervical spine (14 cases in cervical spine, 2 cases in the thoracic spine and one case in the lumbar spine). The majority of cases presented with progressive and permanent myelocaudalopathy due to compression by the engorged vessels rather than with SAH, and even more uncommon is the presentation with acute compression of the spinal cord. The case we present constitute a rare presentation of thoracic DAVF.

The diagnosis of SAH was entertained despite the fact that the initial CT scan only exposed a small amount of intracranial blood in the subarachnoid space. However, the sensitivity of the new generation CT scan to detect SAH ranges from 91 to 100% within the first 12h of onset and 81 to 83% if done after 12 hours and continue declining over time. A lumbar puncture is needed to confirm the diagnosis of SAH, especially if the initial CT scan is negative.

This patient presented first with self-limited abdominal pain and persistent neck pain. The severe LUQ abdominal pain radiated to the back could be explained by the rupture of the DAVF at the onset of the symptoms, producing an irritation of the root nerves at the lesion site (T6-7). The persistent cervical pain might be the result of persistent meningeal irritation by the blood in the arachnoids space.

This patient presented with second episode of hemorrhage from the SDAVF, but unlike the first episode where it produced a SAH, this time it formed a subdural hematoma producing acute compression of the spinal cord causing an abrupt onset of paresis. The prompt neurosurgical intervention allowed him to regain some of the neurological deficit and minimize the disastrous complications of this atypical presentation of SDAVF.

The risk of rebleeding in SAH depends on the etiology. For aneurysms the risk is 2.6-4% in the first 24 hours and 6.4% in the next 3 months, in the setting of SAVM, the risk of rebleeding is high. Halbach et al. described in 1987 a series of 5 patients with SAH due to intracranial DAVF. Among these patients, 60% (3 patients) presented one or more episodes of rebleeding within the next few weeks. In one of the cases, there was the formation of a subdural hematoma just like our patient.

It is important to consider extracranial causes of SAH, especially when there is an atypical presentation and the CT scan shows a scant amount of intracranial blood. Patients with spinal AVMs as an extracranial source of SAH must be monitored closely because the risk of rebleeding is high. And the anatomical location of the SDAVF in the spine can lead to severe consequences for the patient.

**REFERENCES**


Arteriovenous Malformation Mimicking Subarachnoid Hemorrhage Complicated by Spinal Cord Compression (Continued)


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