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A Rare Case of Subarachnoid Hemorrhage due to Rupture of Isolated Anterior Spinal Artery Aneurysm in a Patient with Polycystic Kidney Disease

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Key Words
Spinal artery aneurysm · Subarachnoid hemorrhage · Renal transplant

Abstract
Only a very few cases of subarachnoid hemorrhage due to isolated anterior spinal artery aneurysms have been reported in the literature. We report a case of subarachnoid hemorrhage due to anterior spinal artery aneurysm rupture in a renal transplant patient at our institution. A 47-year-old male had abrupt onset of left lower extremity weakness with bowel and bladder disturbances which prompted emergent surgical evacuation of the clot and hence immediate diagnostic angiography was not performed. However, follow-up serial intracranial arterial ultrasound studies showed only vasospasm of the basilar artery. Repeat MRI of the thoracic spine showed persistence of subarachnoid blood products, but no larger foci compared to previous imaging. When spinal subarachnoid hemorrhage is present in the appropriate clinical setting, isolated anterior spinal artery aneurysm should be considered as a possible, treatable cause.

Introduction

Isolated anterior spinal artery aneurysms have been rarely mentioned in the literature. Other than trauma, the most common cause of intracranial subarachnoid hemorrhage (SAH) is rupture of a cerebral aneurysm. There have been very few case reports worldwide on isolated spinal artery aneurysm rupture [1]. Although histologically similar to intracranial aneurysms, they are uncommon [2]. We report a case of SAH due to anterior spinal artery aneurysm rupture in a renal transplant patient at our institution.

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Case Presentation

A 47-year-old Hispanic male with end-stage renal disease secondary to autosomal dominant polycystic kidney disease was admitted for renal allograft transplantation.

Past medical history was significant for intracranial aneurysms in the left middle cerebral artery (MCA) and in the right posterior communicating artery (PCA), originally diagnosed 30 months ago on magnetic resonance angiography (MRA) of the head done for history of headaches and family history of intracranial bleeds. He underwent stent-assisted coiling of the left MCA aneurysm in September 2008 and also had a two-stage endovascular repair of the right PCA aneurysm in January 2009. Repeat MRA of the head in June 2009 did not show any evidence of recurrent or residual aneurysm. He was hospitalized in January 2010 for subacute subdural hematomas involving bilateral frontal lobes, right parietal and temporal lobes requiring burr hole evacuation. A computed tomographic (CT) scan of the head without contrast during that hospitalization did not reveal SAH to suggest aneurysm rupture. A repeat CT of the head 2 months later showed stable previously stented aneurysms of left MCA and right PCA.

The patient underwent first deceased donor renal allograft transplantation in October 2010. His immediate postoperative course was uneventful. He was ambulating well with the aid of physical therapy and started to complain of muscle spasm in his neck muscles and also back pain, predominantly on the left. On postoperative day five, he developed urinary retention following removal of the Foley catheter. He was seen by Urology and started on tamsulosin with subsequent reinsertion of the Foley catheter. He also had severe constipation and progressive abdominal distention, which was concerning for an internal hernia. He underwent exploratory laparotomy and was found to have Ogilvie's syndrome and a herniation of the small bowel without incarceration or obstruction. While he was recovering from this procedure he complained of bilateral leg numbness and weakness which was initially intermittent, but progressed to persistent motor weakness of the left leg.

Physical examination revealed his blood pressure to be 190/90 mm/Hg, pulse 80 per minute and regular, with respirations 16 per minute. Examination was significant for a distended abdomen. On neurological examination, the patient was alert, oriented and able to follow all commands. Extraocular movements were full and intact. Cranial nerve evaluation was normal. Motor strength was zero in the left lower extremity associated with decrease in all sensory modalities with a sensory level of T12.

Neurology consult was obtained to evaluate for acute abrupt weakness of the left leg. A magnetic resonance image (MRI) of the thoracic and lumbar spine was ordered which showed diffuse SAH throughout the spinal cord with effaced spinal cord from T7 to T12. Maximum cord deformity was seen at T8–9 level with spinal cord edema. The brain MRI showed SAH in the cerebral sulci, prepontine, premedullary and superior cerebellar cisterns (**fig. 1**, **fig. 2**).

A neurosurgical consultation recommended emergent surgical intervention. The patient was taken to the operating room with evacuation of the intradural clots with T7 to T10 thoracic laminectomies. Follow-up intracranial arterial ultrasound showed normal pulsatility indices with vasospasm of basilar artery. He received several sessions of physical therapy with minimal recovery of left leg weakness and was subsequently discharged to a skilled nursing facility for rehabilitation.

Discussion

The syndrome of SAH due to lesions within the spinal canal is a known entity, but is seen infrequently in clinical practice. Aneurysm of the spinal artery is an exceptionally rare entity that can give rise to SAH with an incidence of less than 1% [3]. Henson and Croft [4] stated that though SAH is an uncommon mode of presentation with spinal angiomas, such angiomas are the commonest cause of spinal SAH. However, in contrast, SAH seems to be common when spinal arteriovenous malformations (AVM) are associated with aneurysms and when spinal aneurysms occur in isolation [5].

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Spinal vascular malformations can be categorized into spinal dural arteriovenous fistulae (AVFs), spinal cord AVMs and spinal cord AVFs. The most common type is the spinal dural AVF [6]. A male predominance of 80–90% has been reported. Patients with polycystic kidney disease have a high incidence of aneurysms, as do patients with family members who have had aneurysms [1].

The nidus of spinal cord AVMs may be intramedullary, pial or partly intra- and partly extramedullary. They occur throughout the length of the spinal cord and are considered congenital. Spinal cord AVFs are rare and have direct fistulous connection between medullary artery, usually anterior spinal artery and coronal venous plexus [6].

Spinal artery aneurysms are usually found with other AVMs or entities that increase hemodynamic stress like aortic coarctation or vasculitides [1]. Solitary aneurysms of spinal arteries lacking associated vascular malformations are extremely rare [6]. Aneurysms form in response to high blood flow within a vessel. This theory also explains the formation of aneurysm in the region of the anterior communicating artery to collateral cerebral circulation [1].

Spinal aneurysms may present as SAH or cord compression. Spinal SAH poses diagnostic problems in contrast to intracranial SAH, especially when the lesions are located low in the lumbar region. Lesions of the thoracic region cause intense back pain, paraspinal muscle spasm, headache, photophobia and stiff neck [3]. The sequence of back pain followed by headache is the hallmark of SAH in the thoracic region [7]. SAH due to spinal vascular malformations may cause pain, paresis, bowel and bladder disturbances and sensory abnormalities [8]. Minor SAH can present predominantly as radicular pain that is often mistaken for a musculoskeletal problem. Presence of headache, nausea and vomiting may be the only clue to spinal SAH [7]. Patients with dural AVFs present with gradual progression of sensory and motor deficits in the lower extremities with symptoms increased by physical activity [1]. Acute hemorrhage is uncommon with these lesions. However, dural AVMs often present with spontaneous hemorrhage and neurological deficits that involve both upper and lower extremities [8]. Spinal cord AVFs present with recurrent episodes of transient lower extremity weakness and symptoms of cord infarction [1].

Current knowledge on the incidence and natural history of spinal artery aneurysms is limited to a number of case reports. In contrast to intracranial aneurysms, the location of spinal artery aneurysms is often unrelated to arterial branching sites [6]. There does not seem to be any predilection for spinal aneurysms to occur at any specific segment of the spinal artery. The entire spinal axis has to be studied to rule out aneurysms [7]. Diagnostic imaging of patients who experience sudden onset of back pain, lower extremity radiculopathy, or both, should be evaluated for a potential source of hemorrhage from an aneurysm, when imaging findings indicate the presence of spinal SAH [1]. Selective spinal angiography seems to be the only way by which these aneurysms can be diagnosed [8]. In many instances, the assumption is that the patient may have had a small aneurysm that ruptured and because of vasospasm, the aneurysm is no longer visible [3]. In a small subset of patients, no intracranial vascular lesions are noted on the angiography and it is conceivable that a fraction of these patients may have a high spinal vascular lesion [3].

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Surgical treatment of any spinal aneurysm should be performed only if the various types of inflammatory and noninflammatory vasculopathies are excluded as underlying etiology [7].

Summary

Only a very few cases of SAH due to isolated anterior spinal artery aneurysms have been reported in the literature. Our patient had abrupt onset of left lower extremity weakness with bowel and bladder disturbances which prompted emergent surgical evacuation of the clot and hence immediate diagnostic angiography was not performed. Whether early diagnosis of a spinal artery aneurysm with imaging studies at the first sign of neurologic manifestation would have had beneficial effects in this patient is a matter of speculation. As many of the reported spinal aneurysms are small and have a fusiform rather than saccular shape, standard surgical treatment (clip placement) is not possible. However, follow-up serial intracranial arterial ultrasound studies showed only vasospasm of the basilar artery. Repeat MRI of the thoracic spine showed persistence of subarachnoid blood products, but no larger foci compared to previous imaging.

In conclusion, when spinal SAH is present in the appropriate clinical setting, isolated anterior spinal artery aneurysm should be considered as a possible, treatable cause.

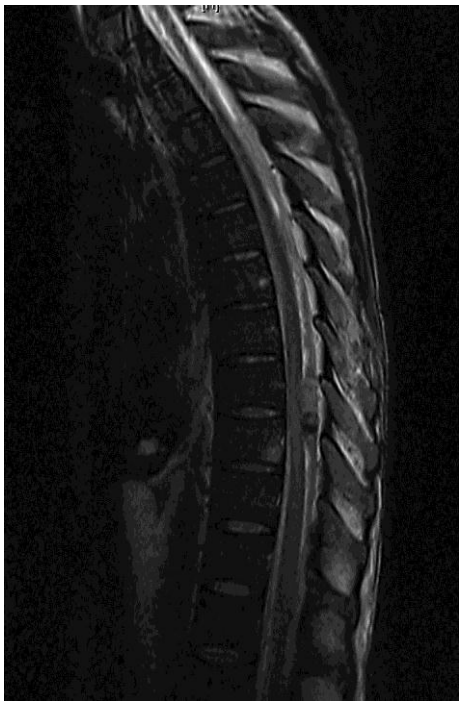

Fig. 1. T_2 sagittal image showing subarachnoid hemorrhage at T8–T9 with spinal cord edema.

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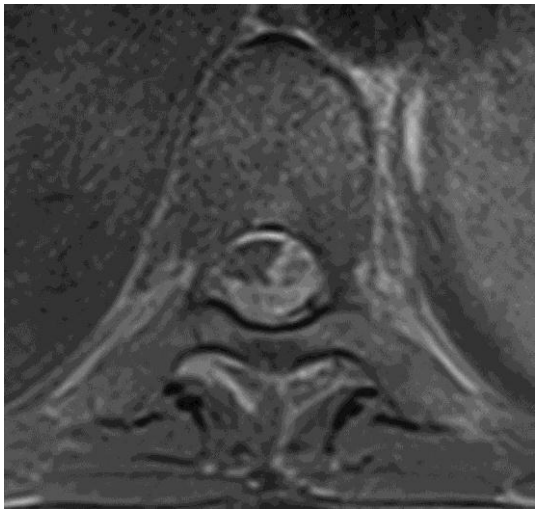

Fig. 2. T_2 axial image showing subarachnoid hemorrhage and spinal cord edema.

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