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Spinal cord infarction associated with subarachnoid hemorrhage

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Keywords

Delayed ischemia; Subarachnoid hemorrhage; Spinal cord stroke

1. Introduction

Paraplegia is a rare complication of subarachnoid hemorrhage (SAH) [1]. Acute paraparesis is proposed to be due to distortion of pyramidal tracts from hydrocephalus. The delayed occurrence is thought to be related to mass effect from interhemispheric clot or parasagittal ischemic strokes from vasospasm in the anterior cerebral circulation [1]. Spinal imaging in these reports has largely been reported negative. We report the delayed development of flaccid paraplegia from spinal cord stroke in a patient of subarachnoid hemorrhage caused by a ruptured anterior communicating artery aneurysm. This stroke was adjacent to a large collection of blood in the lumbo-sacral subarachnoid space. We discuss the various explanations of these findings including the novel possibility of vasospasm in spinal circulation.

2. Case report

A forty two year old Asian female was transferred to the Medical University of South Carolina hospital with sudden onset of altered mental status and subarachnoid hemorrhage. She did not have any significant previous history, except for a fifteen pack year history of smoking and occasional marijuana consumption. Neurologically she had withdrawal response in her arms and trace movement in her legs. Her Hunt and Hess score was 4; Cranial CT (CCT) of the head demonstrated hydrocephalus with transependymal flow and a modified Fisher grade 4. She immediately underwent external ventricular drain (EVD)

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placement. CT angiogram revealed a 6 mm × 5.3 mm aneurysm of the anterior communicating artery (ACoM). She underwent coil embolization of this aneurysm uneventfully with complete exclusion of the aneurysm from the circulation. She was subsequently started on nimodipine and pravastatin. Her care was managed according to the institutional SAH protocol, which includes permissive hypertension, euvolemia, and daily transcranial Doppler monitoring for vasospasm. Her condition improved, and she began localizing with the right upper extremity though she still withdrew the left upper and bilateral lower extremities. A detailed review of her daily nursing and procedure records does not reveal any periods where her systolic blood pressure was less than 90. She underwent a surveillance CT angiogram and perfusion (CTA/CTP) scan six days after the ictus that did not reveal vasospasm or perfusion defects and demonstrated resolution of hydrocephalus. Nine days after aneurysmal rupture, she was found to have no movement in her legs on painful stimulus. Deep tendon reflexes were absent in both lower extremities and the patient had poor rectal tone. A sensory examination could not be performed secondary to depressed mental status. A repeat CTA/CTP showed radiographic vasospasm in the left anterior circulation and scattered areas of ischemia in bilateral ACA territories. She immediately underwent endovascular treatment of vasospasm with intra-arterial verapamil. Despite an optimal angiographic result, she unfortunately remained paraplegic. Subsequent workup included MRI of the brain as well as the cervical, thoracic and lumbar spine. The MRI brain revealed small, scattered strokes in bilateral cingulate gyri, anterior cerebral circulation, corpus callosum and a small tract hemorrhage from EVD insertion in the right thalamus. MRI of the thoracic and lumbar spine revealed subarachnoid blood in the lumbo-sacral subarachnoid space along with signal abnormality within the spinal cord at the level of the conus extending from T11-L1 (Fig. 1). Multiplanar spin echo diffusion weighted imaging confirmed spinal cord stroke in this region giving the appearance of 'owl's eye sign' (Fig. 2). She underwent further evaluation including CSF studies and transthoracic echocardiography. Repeat MRI after two weeks revealed resolving cord signal abnormality. Four months after the ictus, the patient has slight improvement in mental status but continues to be non-verbal with profound lower extremity weakness rendering her wheelchair bound.

3. Discussion

In this article we present the first case of spinal cord stroke associated with subarachnoid hemorrhage. Paraparesis has been reported in SAH patients with anterior circulation aneurysms with a variable frequency between 0.5 and 12% [1]. This is typically caused by parasagittal strokes due to vasospasm, hydrocephalus and mass effect from interhemispheric clot [1]. Although cranial vasospasm has previously been reported after spinal subdural hemorrhage, this is the first case report of spinal cord stroke leading to flaccid paraplegia in subarachnoid hemorrhage patients.

Spinal cord infarct is associated with prolonged hypotension, traumatic mechanical compression of spinal cord vasculature, thromboembolism, dissecting aortic aneurysms, fibro-cartilaginous embolism, infections, and inflammatory processes [2]. Cervical spinal cord ischemia has previously been described in association with spinal subarachnoid hemorrhage in patients with dissection of the vertebro-basilar system or ruptured vascular malformations of the spinal cord [3]. Disruption of the blood supply due to the involvement of radiculo-medullary arteries is thought to be the underlying mechanism in these cases. There were no periods of prolonged hypotension, nor were any infectious or inflammatory processes identified to explain the spinal cord infarct observed in our patient. Thromboembolic etiology from an aortic atherosclerotic plaque during the endovascular procedure remains a possible explanation for these findings. Although, the delayed onset of paraplegia several days after the procedure make this explanation less likely.

SAH patients often complain of back pain secondary to the presence of blood hemolysis products in the lumbo-sacral subarachnoid space. Similar to headache, the intensity of this pain often correlates with the amount of blood in the lumbo-sacral subarachnoid space. There is anecdotal evidence to link spinal arachnoiditis to subarachnoid hemorrhage [4]. High dose steroids seem to offer some relief from this presumed arachnoiditis. Interestingly, although delayed cerebral ischemia (DCI) is a well-known complication of SAH, spinal cord ischemia has never been reported. The vascular supply of the spinal cord is derived from both anterior and posterior circulations. The anterior and posterior spinal arteries receive inputs from radicular arteries through radiculomedullary and radiculopial branches. The superficial spinal cord is supplied by rami perforantes both from anterior and posterior spinal arteries. The deeper spinal cord is supplied by the anterior radiculomedullary artery via sulco-commissural arteries. A pial network extensively connects the anterior and posterior circulation. It is therefore conceivable that presence of subarachnoid blood may cause micro and macro circulatory failure leading to delayed ischemic deficits. The most likely mechanism is vasospasm in the spinal vasculature, which has been shown to cause secondary injury in experimental animal models of spinal cord injury [5]. Unfortunately, this case report lacks angiographic evidence of vasospasm in the spinal circulation. The decision not to perform an angiogram was made because of the additional risks from the procedure without clear therapeutic benefit. Unfortunately, it was an omission to have not obtained MRA concurrent with a spinal MRI. It is strongly suggested that, in future cases, MRA is obtained in order to determine whether larger vessels are involved with vasospasm.

4. Conclusions

This is the first case report describing the development of stroke in the spinal cord following subarachnoid hemorrhage. In absence of a clear cerebral cause, imaging of the entire spine should always be considered to investigate lower extremity weakness in subarachnoid hemorrhage patients. Further research is needed to investigate whether vasospasm can occur in the spinal circulation.

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Fig. 1. Sagittal T1 (A) and T2-weighted (B) images of the lumbar spine reveal sub-arachnoid hemorrhage in the distal thoracic and lumbar spine (arrows) along with bilateral hyperintense signal changes on T2- and diffusion weighed imaging in the gray matter of the thoraco-lumbar spinal cord.

Fig. 2. Axial images T2-weighted (A), T1-weighted pre (B) and post-gadolinium (C), diffusion-weighted imaging (DWI) (D) and corresponding apparent diffusion coefficient (ADC) map (E) at the level of the thoraco-lumbar spinal cord. There is abnormal swelling, T2 hyperintensity and enhancement of the central gray matter. DWI images demonstrate restricted diffusion of the central gray matter of the distal spinal cord.