Acute paraplegia after aneurysmal subarachnoid hemorrhage: Case report of a rare complication with a 2-year follow-up

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Abstract. The current overall incidence of subarachnoid hemorrhage (SAH) is ~9/100,000 individuals/year and rupture of an intracranial aneurysm is the main cause of SAH, accounting for ~85% of cases. Only a small number of cases of paraplegia after intracranial aneurysmal SAH have so far been reported and its pathogenesis has remained to be fully elucidated. The present study reports the case of a patient with an aneurysm localized in the medial and inferior lateral wall of the C5 segment of the right internal carotid artery that was treated by coil interventional embolization. The muscle strength of both lower extremities of the patient was grade I and grade 0 before and after the operation, respectively. Lumbar and thoracic magnetic resonance imaging examinations revealed slight hematoma in the subarachnoid space below the L2 level. At two weeks after the operation, the muscle strength of both lower extremities was grade II, while the muscle strength was grade III and grade V at 30 and 60 days after the operation, respectively.

Introduction

Subarachnoid hemorrhage (SAH) is a severe subtype of stroke that affects individuals at an average age of 55 years and leads to the death of several patients (1). Rupture of an intracranial aneurysm is the primary cause of SAH in 85% of patients. Intracranial aneurysm is a common intracranial cerebrovascular disease. The rupture of an intracranial aneurysm leads to SAH, with a high mortality rate of 40% (2). However, 50-80% of patients with aneurysms in their intracranial arteries show no symptoms. After rupture, patients commonly present with headache, dizziness, nausea, vomiting, neck stiffness, and, in severe cases, aphasia, hemiplegia and disturbance of consciousness. There is a small number of reports of acute paraplegia secondary to hemorrhage (3,4). It has also been reported that subarachnoid hemorrhage after spinal anesthesia causes paraplegia (5). The present case report describes the preoperative and postoperative conditions and postoperative recovery of a patient with acute paraplegia following a ruptured aneurysm. The muscle strength of both lower extremities of the patient was grade I prior to the operation and grade 0 after the operation. After active treatment and rehabilitation exercises, the muscle strength of the lower extremities gradually recovered.

Case report

A 40-year-old male patient with a history of hypertension presented to the emergency department of Peking University People's Hospital (Beijing, China) in December 2019 with the complaint of headache for one day, sudden onset of nausea, vomiting and loss of consciousness for 4 h. Neurological examination revealed a Glasgow Coma Scale (GCS) score of 14 (E3V6M5). The patient was a habitual drinker and reported that no immediate family members had experienced any similar cerebrovascular events. The patient had no history of trauma, lumbar puncture or bleeding disorders. The patient was admitted to the emergency department of Peking University People's Hospital (Beijing, China). His blood pressure was controlled through the infusion of urapidil hydrochloride (2-4 ml/h) and his vital signs were stabilized using monitoring equipment and nimodipine administration (2-4 ml/h, 14 days), as described previously (6,7). Cranial computed tomography (CT) and CT angiography (CTA) were performed at the earliest. Brain CT and CTA (Fig. 1A and B) indicated diffuse SAH and an aneurysm in the right internal carotid artery. Thus, a digital subtraction angiography (DSA) examination was scheduled immediately. DSA revealed that the aneurysm was located in the inferior wall of the C5 segment of the right internal carotid artery, with the following characteristics: Aneurysm neck 3 mm and diameter 4x6 mm (Fig. 1C). The patient was placed under general anesthesia 9 h after the bleeding occurred and the intracranial aneurysm was embolized using a coil interventional embolization technique. DSA indicated that the patient's aneurysm was densely packed without contrast retention (Fig. 2A and B). The patient was diagnosed with a right internal carotid artery aneurysm and hypertension.

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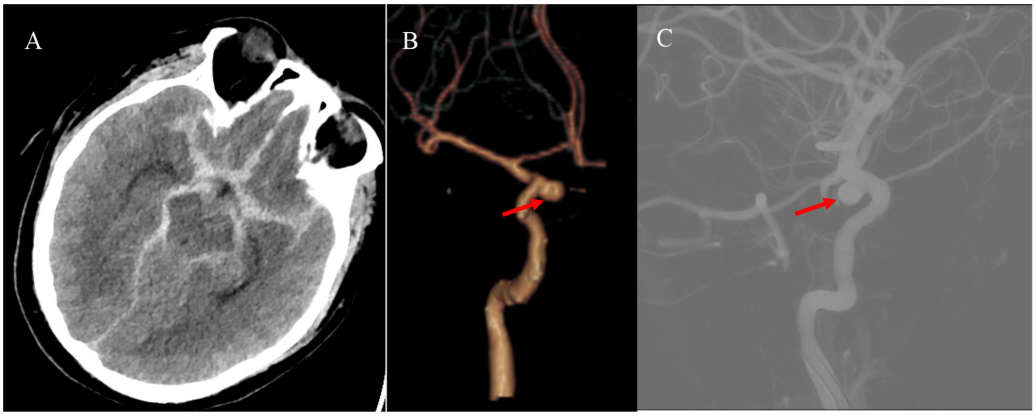

Figure 1. Preoperative imaging examination of the patient. (A) Initial brain CT showing diffuse subarachnoid hemorrhage. (B) Reconstructive CT angiogram and (C) digital subtraction angiography displaying the C5 segment of the right internal carotid artery aneurysm (red arrows). CT, computed tomography.

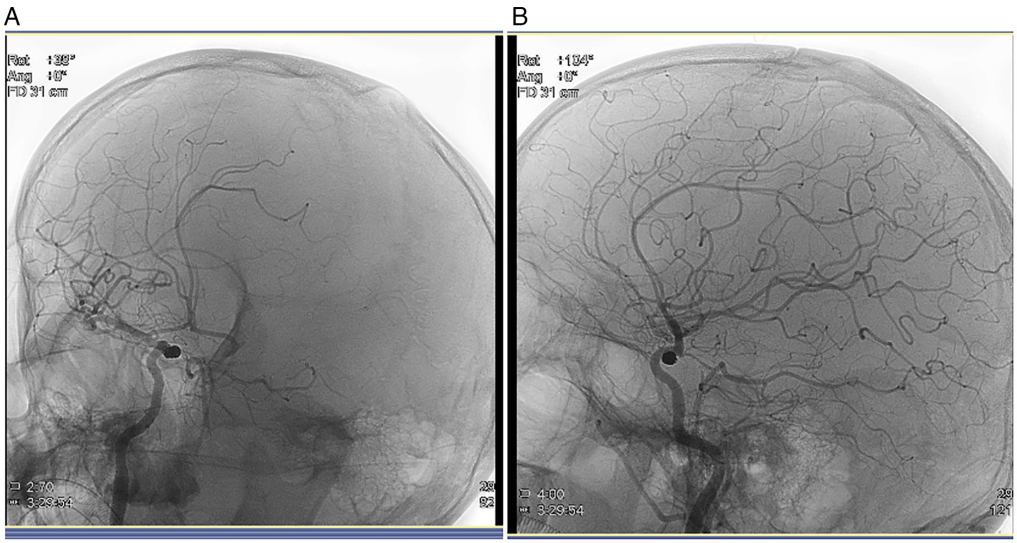

Figure 2. Digital subtraction angiography of the patient after surgery. (A) Skew map of aneurysm after embolization. (B) Side map of aneurysm after embolization. Digital subtraction angiography indicated that the patient's aneurysm was densely packed without contrast medium retention.

After the operation, the patient was transferred to the intensive care unit with tracheal intubation. Following regaining of consciousness and stability of vital signs, the tracheal intubation was removed and the patient was transferred to the general ward on the second day after the operation. The patient was awake and his GCS score was 15 points. The muscle strength assessment was using Lovett's grading approach (8), the muscle strength of both upper limbs of the patient was normal, while the muscle strength of both lower limbs was grade 0, but leg sensation of the patient was normal. No new hemorrhage was detected in the re-examined head CT area on the first day after surgery (Fig. 3). To determine whether the patient's paraplegia was related to the thoracolumbosacral spine, the patient underwent thoracic spine (5 days after the surgery) and lumbosacral spine (4 days after the surgery) magnetic resonance imaging (MRI) examination and lower extremity venous ultrasonography (10 days after the surgery). The thoracic spine MRI scan indicated mild degeneration of the thoracic spine, mild kyphosis of the T4/5 and T5/6 intervertebral discs and mild right kyphosis of the T7/8 intervertebral disc. T3 and T10 vertebral hemangiomas were suspected, as indicated by the

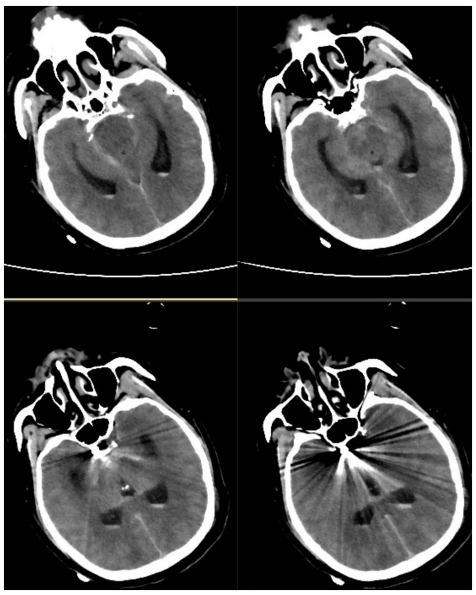

Figure 3. Cranial computed tomography on the first day after surgery. No new hemorrhage was detected in the re-examined head CT area.

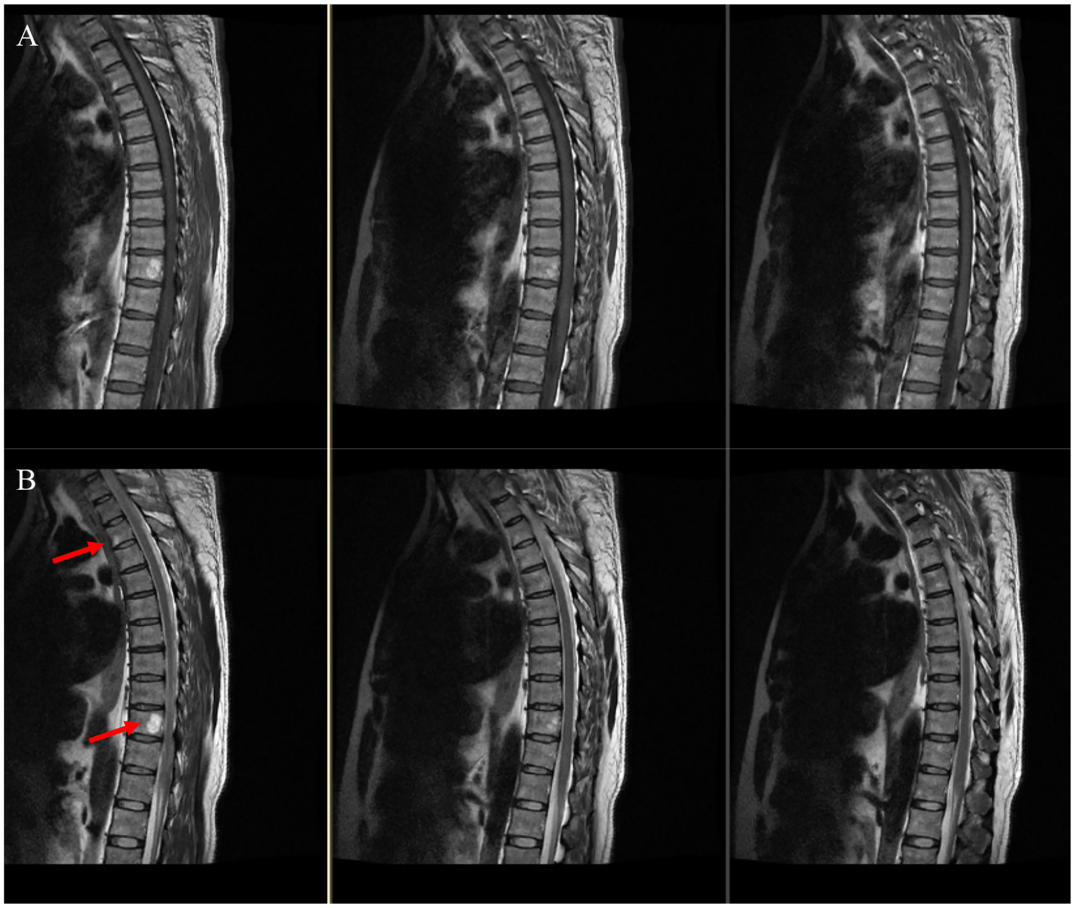

Figure 4. Weighted MRI of the patient's thoracic spine 5 days after the surgery. (A) T1-weighted and (B) T2-weighted MRI images. T3 and T10 vertebral hemangiomas were suspected, as indicated by the red arrow. MRI, magnetic resonance imaging.

red arrow in Fig. 4. The lumbosacral spine MRI scan revealed slight hematoma in the subarachnoid space below the L2 level, as indicated by the red arrow in Fig. 5.

No apparent venous thrombosis was found by lower extremity venous ultrasonography 10 days after the surgery (Fig. 6A and B). To clear the SAH in the patient, lumbar spinal catheter drainage was performed on the first day after the operation. On the fourth day after the operation, the catheter was removed and a lumbar puncture was performed once a day for seven consecutive days to release the cerebrospinal fluid (CSF). The CSF became transparent and clear, while the patient was conscious and responsive. The muscle strength of both lower extremities returned to grade II two weeks after the operation. The patient was discharged three weeks after the operation and transferred to a rehabilitation hospital for further treatment. The patient was treated with cognitive and physical exercise in the rehabilitation hospital. The muscle strength of the patient's lower extremities was grade III one month after the operation and returned to grade V 60 days after the operation. The patient revisited the hospital for re-examination six months after the operation and his nerve function was found to be normal. A two-year follow-up cranial DSA indicated satisfactory embolization of the aneurysm (Fig. 7).

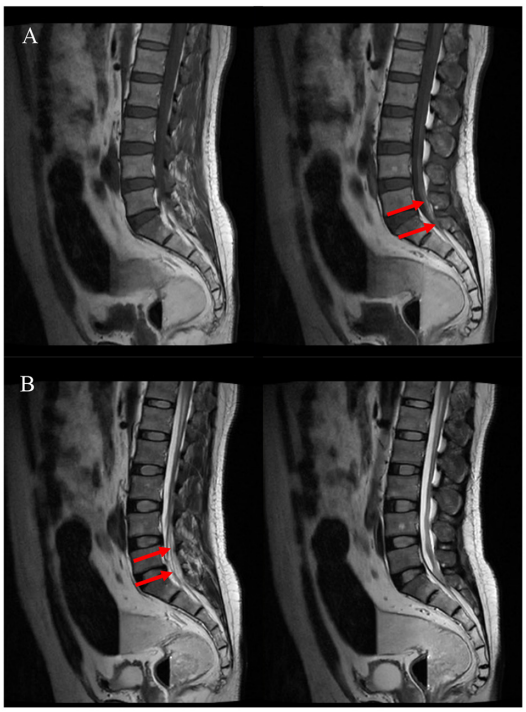

Figure 5. Weighted MRI of the patient's lumbosacral spine 4 days after the surgery. (A) T1-weighted and (B) T2-weighted MRI images. The lumbosacral spine MRI scan indicated a slight hematoma in the subarachnoid space below the L2 level, as indicated by the red arrow. MRI, magnetic resonance imaging.

Discussion

Acute paraplegia secondary to intracranial aneurysmal SAH is a rare complication that, to the best of our knowledge, is

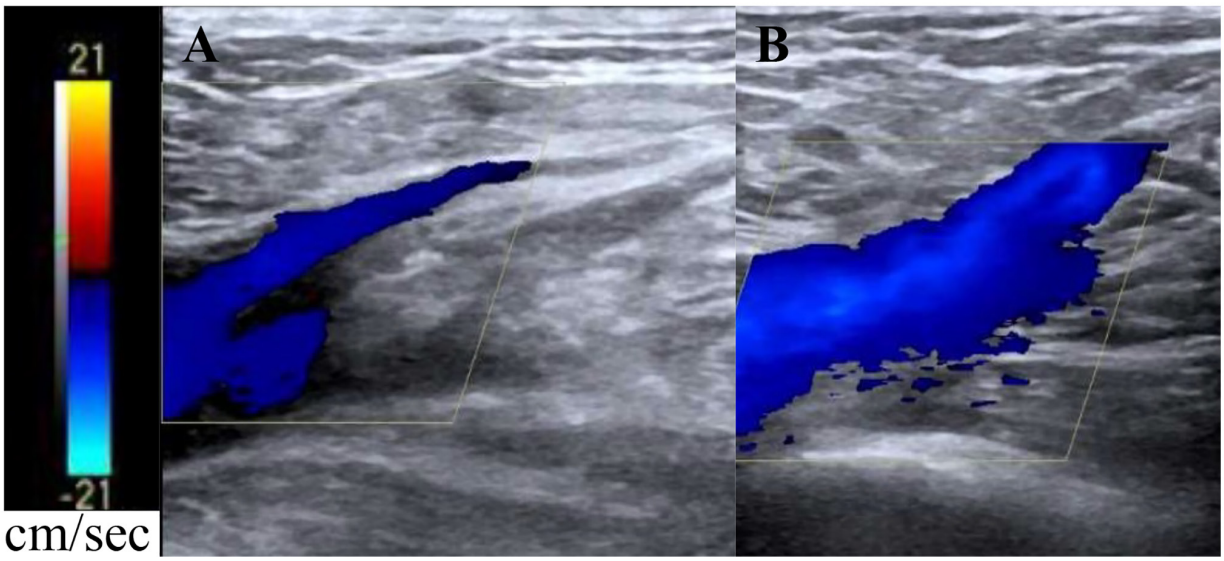

Figure 6. Color Doppler ultrasound of lower limb veins of the patient 10 day after the surgery. (A) Image of the inflow segment of the great saphenous vein into the femoral vein when the patient held his breath. (B) Image of the inflow segment of the great saphenous vein into the femoral vein when the patient was breathing normally. The blood flow in the vascular lumen of lower limbs of the patient was unobstructed and there was no obvious embolism.

scarcely reported in the literature. Its pathogenesis remains unclear and the incidence of paraparesis after the rupture of an aneurysm in the anterior communicating artery is as high as 4.5-9.5% (9-11). Spinal arachnoiditis after aneurysmal SAH may cause paralysis of the lower extremities (12,13). The manifestations of spinal arachnoiditis depend on the extent of involvement and compression of the spinal cord. Common presentations are sensory disturbances with leg pain, progressive weakness and sphincter dysfunction. Klekamp (13) reported that subarachnoid inflammatory disease secondary to SAH was due to blockage of the basal cistern by blood. The accumulation of blood in the subarachnoid space and further hemolysis may lead to meningeal irritation and eventually arachnoiditis (14). Previous studies have reported that the risk of developing spinal arachnoiditis is significantly increased following SAH associated with the rupture of the posterior inferior cerebellar artery or posterior communicating artery aneurysms (15-17). In these aforementioned studies, it was noted that posterior fossa SAH secondary to spontaneous aneurysm rupture may lead to severe spinal arachnoiditis.

Ovali *et al* (17) reported the case of a patient with a ruptured aneurysm in the V4 segment of the right vertebral artery who presented with bilateral buttock and leg numbness, as well as severe back pain, possibly due to the diffusion of hemorrhage into the spinal subarachnoid space. Long-term irritation of the *pia mater* by blood in the subarachnoid space following a ruptured aneurysm may lead to arachnoiditis, but the disease generally has a longer course and persists for a long time (18). Chiang *et al* (4) reported the case of a patient with paraplegia after the rupture of a basilar artery aneurysm and subsequent hemorrhage. After placing a lumbar drainage tube, the patient's paraplegia disappeared immediately.

The patient of the present case report was a middle-aged overweight male and was a habitual drinker. Consequently, atherosclerosis may be the etiology of SAH in this patient (19,20). Furthermore, the patient had a history of hypertension and irregular control of blood pressure may be one of the factors for inducing aneurysm rupture (21,22). In the present case, acute paraplegia developed immediately after the onset of aneurysmal SAH and the possibility of hemorrhage causing spinal vasospasm was considered. To further clarify the reasons for the decreased muscle strength of the patient's lower extremities, a head CT scan and lumbosacral and thoracic MRI scans were performed after the surgery. No lesions related to the hemorrhage were found. After lumbar cistern drainage and multiple lumbar punctures, the blood in the subarachnoid space was cleared and the drug nimodipine was administered immediately after the operation for two weeks to control vasospasm. However, the muscle strength of the patient's lower extremities recovered only to grade II after two weeks. After active neurological rehabilitation, the muscle strength of the patient's lower extremities returned to grade V

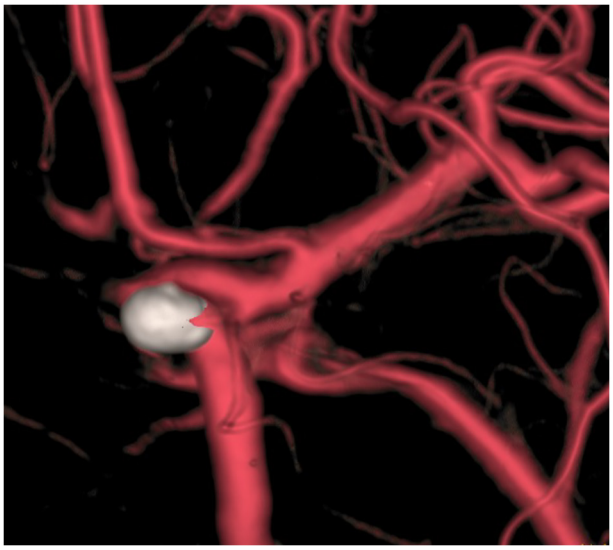

Figure 7. Digital subtraction angiography of the patient two years after the surgery. The image indicated satisfactory embolization of the aneurysm.

60 days after the operation; thus, vasospasm combined with chronic arachnoid inflammation cannot be excluded. The anterior two-thirds and the posterior one-third of the spinal cord are supplied by the anterior and posterior spinal arteries, respectively (23). As the blood supply is the weakest in the transition area supplied by the superior and posterior spinal arteries, the lower thoracic spinal cord is an area with a latent risk of developing vasogenic paraplegia (24). This finding corresponds to the paraplegic condition of the patient of the present study. It may be speculated that his paraplegia may be related to spinal cord edema caused by hypertension and increased intracranial pressure. The symptoms of paraplegia disappeared gradually following the reduction of spinal cord edema after the operation.

It is worth noting that during the inpatient physical examination, the leg circumference of the patient's right calf was found to be significantly smaller than that on the contralateral side. The patient's family recalled that the patient had suffered from polio as a child, but there were no remaining complications. In patients with polio, after injury to the anterior horn cells of the spinal cord, muscle atrophy and weakness occur due to the loss of motor neuron regulation in the muscle (25). To the best of your knowledge, the present study reported on the first case of acute paraplegia after SAH in a patient with a polio history. It may be speculated that in vascular events, patients with polio may be more prone to spasms of spinal vessels. The propensity for ischemic changes in the thoracolumbar region is significantly higher, as it is mainly supplied by a single vessel, namely the Adamkiewicz artery (26,27). A ruptured spinal aneurysm also causes paralysis (28). Of note, the present study has certain limitations: Spinal angiography was not performed to preclude paraplegia due to spinal vascular disease.

Based on the patient's onset characteristics and the postoperative recovery period, it may be considered that the paraplegia of the patient was possibly associated with vasospasm of the spinal arterioles and arachnoid inflammation. This case report concludes that such patients have a high likelihood of recovering from paralysis through the lumbar puncture and active rehabilitation exercises following surgical treatment.

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Availability of data and materials

The datasets used and analyzed during the current study are available from the corresponding author on reasonable request.

Authors' contributions

JO and RL conceived and designed the study. JO and JZ wrote the manuscript. BH, BW and RL reviewed and edited the manuscript. JO and JZ acquired the data. BH, BW and ZL collated and analyzed the data. JO and JZ confirm the authenticity of all the raw data. All authors have read and approved the final manuscript.

Ethics approval and consent to participate

The study was approved by the Ethics Committee of the Peking University People's Hospital (Beijing, China; approval no. 2021PHE039). Written informed consent was obtained from the patient.

Patient consent for publication

The patient provided written informed consent for the publication of the data and the images.

Competing interests

The authors declare that they have no competing interests.

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