Am J Transl Res 2023;15(5):3793-3799
www.ajtr.org /ISSN:1943-8141/AJTR0149566

Case Report

Cerebral stroke mimics associated with spinal vascular disease: two case reports

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Received February 8, 2023; Accepted May 15, 2023; Epub May 15, 2023; Published May 30, 2023

Abstract: Several diseases produce symptoms similar to those of a cerebral stroke, resulting in their misdiagnosis as stroke. Cerebral stroke mimics are common in emergency rooms. We report two cases of cerebral stroke mimics to attract the attention of clinicians, especially emergency room doctors. In one case, a patient with spontaneous spinal epidural hematoma (SSEH) exhibited lower-right limb numbness and weakness. In the other, a patient with spinal cord infarction (SCI) had numbness and weakness of the lower-left limb. Both cases were misdiagnosed as cerebral strokes in the emergency room. One of the patients underwent hematoma removal surgery, and the other received medical treatment for spinal cord infarction. Patients' symptoms improved, but the sequelae remained. Single-limb numbness and weakness are an uncommon initial presentation of spinal vascular disease that can lead to its misdiagnosis. When encountering single-limb numbness and weakness, it is necessary to consider the differential diagnosis of spinal vascular disease, thereby reducing misdiagnosis.

Keywords: Cerebral stroke, spinal vascular disease, spinal epidural hematoma, spinal cord infarction

Introduction

A study of 8,187 patients referred to the emergency department for suspected strokes found that 30% had stroke mimics [1]. Cerebral stroke mimics include several neurological conditions, such as seizures; systemic infections; brain tumors; toxic, metabolic, or vestibular dysfunction; syncope; subdural hematomas; transient global amnesia; dementia; conversion disorders; migraines; and peripheral neuropathies [2]. SSEH is a clinically uncommon disease usually characterized by paraplegia and quadriplegic paralysis accompanied by severe neck or back pain. In recent years, case reports have revealed that SSEH can manifest as hemiplegia, leading to misdiagnosis as cerebral stroke [2-5]. The case of SSEH we report here involved numbness and weakness in the lower-right limb unaccompanied by chest pain. These are rarely the first symptoms in SSEH cases. Similarly, the diagnosis of SCI, another spinal vascular disease, remains a challenge. It is rare for SCI to be misdiagnosed as cerebral infarction, but it is often misdiagnosed as transverse myelitis [6]. SCI develops rapidly, and its symptoms include sudden chest and back pain, rapid progression of lower or quadriplegic limb mobility disorders, and urination and defecation disorders. The presentation of SCI in our reported case, i.e., with numbness and weakness in the lower-left limb, is rare. We report these two cases of spinal vascular disease, which were misdiagnosed as ischemic cerebral strokes, to draw the attention of clinicians to the symptoms of single-limb numbness and weakness, which are common in cerebral stroke cases.

Case reports

A 59-year-old man was admitted to the hospital with symptoms of persistent lower-right limb numbness and weakness. The patient provided written informed consent for the publication of this case report. He had no medical history of hypertension, diabetes, hyperlipidemia, heart or blood system diseases, use of anticoagulants or antiplatelet drugs, or trauma. A neurological examination upon admission showed that the patient's consciousness was clear and that the cranial nerve was normal. The muscle strength score in the limbs was 5/5. The tactile

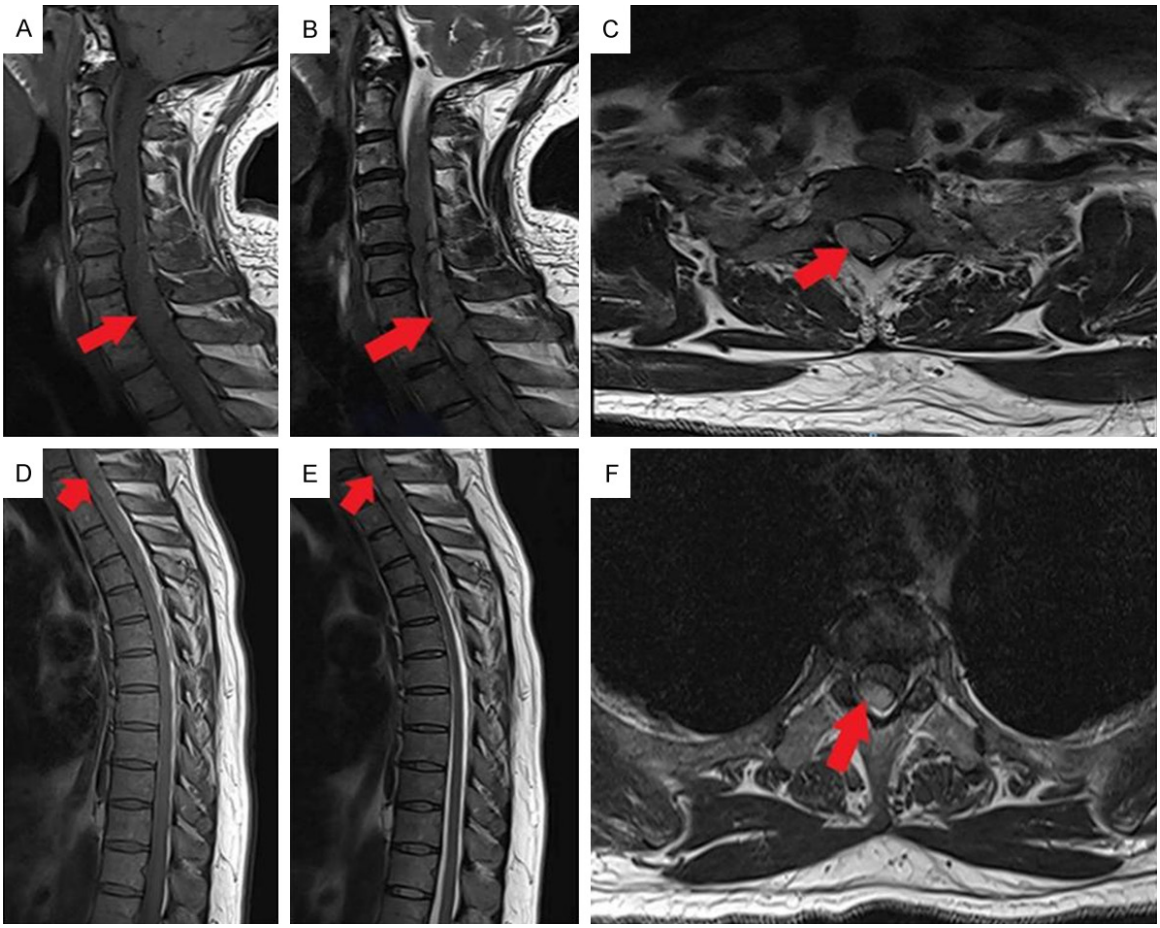

Figure 1. A and D: MRI results (sagittal) of the cervical and thoracic vertebrae show an isointense mass (red arrows) on the T1-weighted images in the vertebral canal at the level of C5-T4. B and E: Show a hyperintense mass (red arrows) on the T2-weighted images in the vertebral canal at the level of C5-T4. C and F: MRI coronal results of the cervical and thoracic vertebrae show a hyperintense mass (red arrows) on the T2-weighted images.

sense in the lower-right extremity was slightly diminished, but the feeling in the other limbs was normal. The bilateral Babinski sign was negative. Computed tomography (CT) found no new cerebral infarction lesions. Routine blood tests and coagulation laboratory tests showed no abnormalities. When the patient finished the head CT scan, he described having difficulty moving the right-lower limb and had lost all sensation in the lower-right extremity. The patient's condition further deteriorated. Weakness in the lower-left extremity was detected, accompanied by numbness. Neurological examination revealed that the patient's muscle strength scores in both lower limbs were 1/5. The sensory deficiency was below the T4 skin watersaving level. The bilateral Babinski sign was positive. Since the patient's bladder was full, urinary retention was considered, and an emergency urethral catheterization was performed immediately.

Based on the patient's symptom development, the emergency doctor determined that the patient's lesion was in the spinal cord. To verify this, the patient was urgently given magnetic resonance imaging (MRI) examinations of the cervical and thoracic vertebrae. The MRI results showed a hyperintense mass on the T2-weighted images and an isointense mass on the T1-weighted images in the vertebral canal at the C5-T4 level (**Figure 1A**, **1B**, **1D** and **1E**). The corresponding region of the spinal cord was compressed (**Figure 1C** and **1F**). The MRI results for the cervical and thoracic vertebrae confirmed that the patient had a huge epidural hematoma. To avoid further deterioration of nerve function due to spinal cord compression, neurosurgery was performed immediately under general anesthesia. Laminectomy and excision of the epidural hematoma were duly carried out. We found no evidence of a tumor or vascular malformation during the operation.

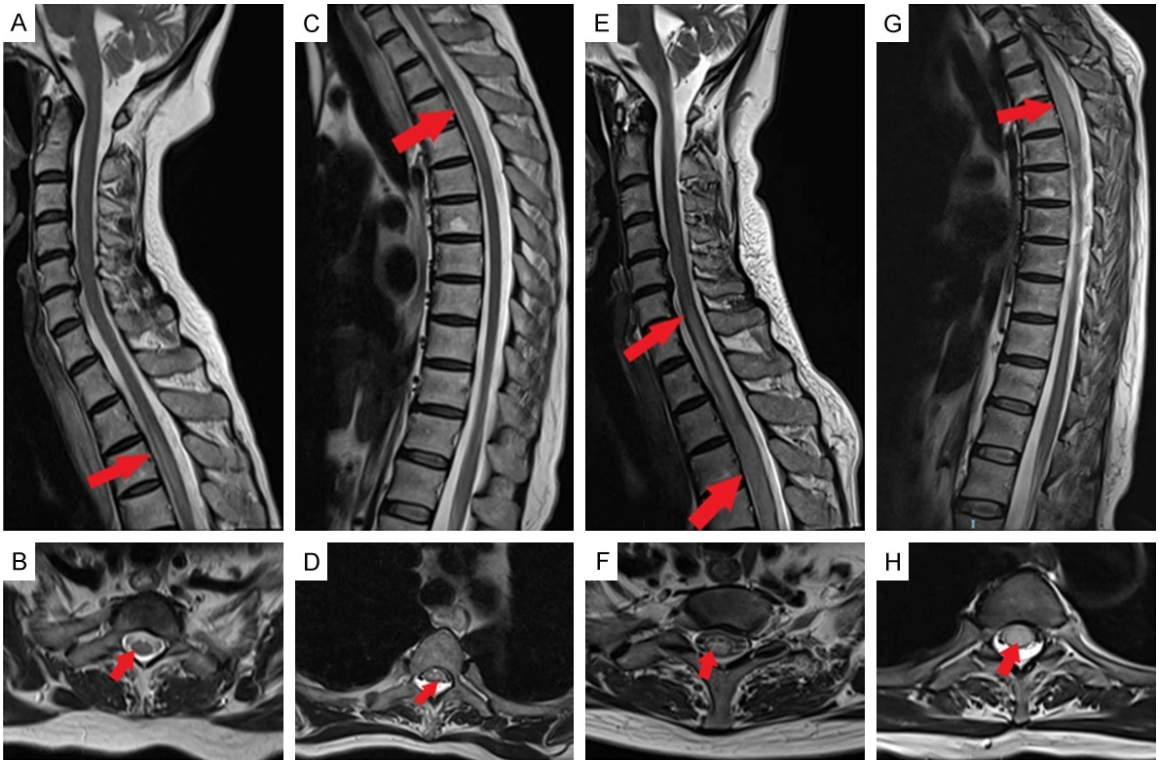

Figure 2. A and C: MRI results (sagittal) of the cervical and thoracic vertebrae show a hyperintense lesion on the T2-weighted image (red arrows). B and D: MRI results (coronal) of the cervical and the thoracic vertebrae show "owl's eye" hyperintense lesions on the T2-weighted images (red arrows). E and G: MRI results (sagittal) of the cervical and thoracic vertebrae show spinal cord swelling and a hyperintense lesion on the T2-weighted image (red arrows). F and H: MRI results (coronal) of the cervical and thoracic vertebrae show a hyperintense lesion and spinal cord swelling on the T2-weighted images (red arrows).

The mass removed during the operation was cultured and showed no cause for concern.

The patient's condition remained stable after the operation. Postoperative MRI examination of the cervical and thoracic vertebrae showed that the spinal cord compression had been relieved. The patient gradually recovered from the neurological deficit and was discharged after 18 days of hospitalization. Six months after the discharge, the patient's neurological deficit had further improved, and he was able to walk by himself and urinate without any hindrance. He also regained sensation in his limbs. Neurological examination showed that the muscle strength scores of the lower-right and lower-left extremities were both 4/5.

A 62-year-old woman with no medical history of disease or risk factors for cardiovascular and cerebrovascular disease reported awaking early in the morning with weakness and a sense of numbness in her lower-left extremity. The patient provided written informed consent for the publication of this case report. The patient was admitted to the emergency department 5 hours later and diagnosed with an acute stroke. Head CT scans showed no abnormalities. Six hours after symptom onset, the patient complained of weakness and numbness in the lower-right extremity. The laboratory results showed no abnormalities in the blood tests. The patient's condition continued to deteriorate. Twelve hours after the onset of symptoms, the patient was unable to move her lower limbs, she lost sensation in her abdomen and lower limbs, and she had difficulty defecating and urinating. A nervous system examination found that the patient was conscious and fluent in speech. The muscle strength of both of her lower extremities was graded as 0/5. Her sensation below the level of the fourth thoracic vertebra disappeared. An MRI of the cervical and thoracic vertebrae revealed a striped lesion presenting with hyperintensity on the T2-weighted images (**Figure 2A-D**). On the T1-weighted

images, the lesion presented with isointensity. On the T2-weighted image (**Figure 2D**), we saw two hyperintense spots in the center of the spinal cord with the typical "owl's eye" appearance of SCI. A lumbar puncture examination showed that the pressure was 120 mm H_2O and the cerebrospinal fluid (CSF) was colorless and transparent. The results of CSF biochemical tests were normal. A test for aquaporin 4 (AQP4) antibody in the CSF was also negative. We concluded that the patient suffered from SCI. The results of thoracic aorta computed tomography angiography (CTA) indicated the presence of arteriosclerosis in the aorta and the partial branch of the artery. The patient was treated with glucocorticoids, vitamins B_1 and B_{12}, and aspirin.

Additional MRIs of the cervical and thoracic vertebrae were performed on the 7th and 14th days after the onset of the disease. On the 7th day, the corresponding part of the spinal cord in the C6-T6 vertebral level segment was found to be significantly swollen. This swelling was dynamic (**Figure 2**) and presented with hyperintensity on the T2-weighted images (**Figure 2E-H**). On the T1-weighted images, the lesion presented with isointensity.

The patient's condition improved on the 11th day after admission. The level of limb sensory disturbance decreased from the T4 vertebra level to the T10 level, and the bilateral Babinski sign was positive. The patient was followed for 6 months after the discharge and regained sensation during defecation. The patient's other conditions remained unchanged.

Discussion

Cerebral stroke mimics are non-vascular diseases, but their neurologic deficit manifestations are quite similar to those of a cerebral stroke. They represent a significant percentage of all acute stroke hospital admissions [9]. The early treatment window for an ischemic cerebral stroke is quite short: intravenous thrombolysis within less than 4.5 hours is an effective treatment for this condition [8]. However, due to this time constraint, many cerebral stroke mimics have been misdiagnosed as ischemic cerebral stroke and treated with intravenous thrombolysis. This may have adverse consequences, such as an increased risk of bleeding [4, 10]. Therefore, expedient identification of cerebral stroke mimics is of great importance. Although some researchers have summarized the classification of cerebral stroke mimics and the associated image findings [2, 9], they did not indicate that spinal vascular disease may also be a common cerebral stroke mimic.

SSEH is an uncommon disease characterized by sudden-onset back pain followed by motor sensory deficits below the site of pain and symptoms of spinal cord compression. Common causes of SSEH include hypertension, vascular malformations, tumors, anticoagulant and antiplatelet therapies, intense exercise, and hematologic diseases [2]. In SSEH, MRI shows T2 hyperintensity with lower apparent diffusion coefficient values [11]. T1-weighted imaging can be used to visualize hemorrhagic lesions and evaluate their age thanks to the temporal changes in hemoglobin degradation products (i.e., iso signal during the acute phase, hyper signal from 3 days to 3 months after the hemorrhagic episode, and hypo signal thereafter) [12]. If the spinal cord edema is severe and subarachnoid obstruction occurs, the protein content of CSF will increase. Peripheral blood tests are usually nonspecific. Many reported cases of SSEH have been misdiagnosed due to its similarity to the hemiplegia observed in strokes [2-5]. However, the initial symptoms of the case we reported were numbness and weakness of the lower-right limb, which are rare, and our patient's symptoms and conditions were not connected to the causes listed above, such as vascular malformations, intense exercise, and hematologic diseases. Neck and back pain accompanied by paraplegia or quadriplegia is a common symptom of SSEH [2, 4]. However, there have been two reported cases of spontaneous cervical epidural hematoma without any back or neck pain: the two patients were Japanese men in their eighties with dementia, and their perceptions and descriptions of the pain were unclear [7]. In this article, we report a case of a patient with SSEH and normal cognitive function. Because the patient did not have any neck or back pain but did experience persistent numbness and weakness in his lower-right limb, he was initially misdiagnosed with an ischemic stroke. Sagittal MRI of the cervical and thoracic vertebrae showed an isointense mass on the T1-weighted images and a hyperintense mass on the T2-weighted images in the vertebral canal at the C5-T4 level (**Figure**

1A, **1B**, **1D** and **1E**). At the onset of the disease, the spinal epidural hematoma may have been small, thus compressing only the right part of the spinal cord, which caused numbness only in the lower-right limb. As the hematoma grew, it significantly compressed the spinal cord, resulting in paraplegia. At present, the treatment of SSEH mainly relies on surgery, but some reported cases with mild symptoms were treated conservatively and achieved a good prognosis [2, 4, 7]. The prognosis of SSEH correlates well with the severity of the disease and the time between onset and treatment [4]. In our reported case, the spinal epidural hematomas were large, and the spinal cord was subjected to a great degree of compression. Rapid diagnosis and surgical intervention resulted in the patient's recovery from neurological deficits.

SCI accounts for 0.3% of all strokes and is less common than cerebral infarctions [13]. The disease usually presents with focal severe tearing pain in the chest and back as the first symptom. There are many causes of SCI, including arterial disease, embolism, blood hypercoagulability, decompression sickness, strenuous exercise, and hemodynamic changes. The most common cause is aortic disease, including aortic rupture, aortic dissection, and atherosclerotic thrombosis [14]. The transverse blood supply of the spinal cord includes the anterior spinal artery that runs along the anterior median groove of the spinal cord and the two posterior spinal arteries that run along the posterior sylvian fissure. The anterior spinal artery supplies the anterior 2/3 of the spinal cord, and the posterior spinal artery supplies the posterior 1/3. The longitudinal blood supply of the spinal cord can be roughly divided into three sections: the upper section, from the first cervical spinal cord segment to the second thoracic cord segment; the middle segment, from the third thoracic cord segment to the eighth thoracic cord segment; and the lower segment, from the ninth thoracic segment to the twelfth thoracic segment, lumbar enlargement, and conus medullaris. The distribution of the anterior spinal artery is most dense in the lumbosacral region, followed by the neck, and the least dense in the chest [15, 16]. Because the diameter of the thoracic segment of the spinal cord is relatively small and the distribution of the central spinal artery in the thoracic segment is the least. Thus the blood supply of this segment is weak, and SCI lesions are more common in the thoracic cord [17]. Interruption of the spinal artery supply causes infarction, which leads to ischemia and necrosis of the spinal cord in the corresponding blood supply area, resulting in neurologic dysfunction of the spinal cord segment [13].

Examining the patient's medical history and conducting a careful physical examination are important steps in the diagnosis of SCI. MRI is the preferred imaging examination for accurate clinical localization and qualitative diagnosis. MRI of SCI in the acute phase shows a thickened spinal cord, a hypo signal on the T1-weighted image, and a hyper signal on the T2-weighted image as well as a "pencil" sign in the sagittal position and an "owl's eye" sign in the coronal position [6]. In some cases of SCI, MRI shows signs of vertebral infarction, the main manifestation of which is abnormal bone marrow signals on T2-weighted imaging in multiple areas near the anterior half of the vertebral body or the endplate and/or the deep medullary portion, which is helpful in SCI diagnosis [16, 18]. Because MRI is not sensitive in the detection of acute SCI, and the SCI lesion is long, SCI must be differentiated from neuromyelitis optica spectrum disorders (NMOSD) using clinical manifestations and laboratory results. CSF tests in patients with SCI are usually normal. By contrast, NMOSD often exhibits a process of recurrence and remission, and approximately 60%-90% of patients have anti-AQP4 antibodies in their CSF [18]. Myelin oligodendroglial cell glycoprotein antibody-related NMOSD occurs in the thoracolumbar segment [19]. Blood tests, such as blood routine, hypersensitive C-reactive protein, vasculitis-related antibodies, and tumor markers, were used to find evidence of infection, autoimmunity, tumor, and vasculitis, in previous cases. These tests are not specific for the diagnosis of SCI but can be used as evidence of etiology. Moreover, CTA can assist in the detection of arterial dissection, aneurysms, vascular malformation, and atherosclerosis, and echocardiography and transcranial Doppler foaming tests can evaluate the presence of cardiogenic emboli. The underlying cause in our reported case was not certain, though we suspected that it might have been related to spinal arteriosclerosis and occlusion. Aortic CTA showed that the patient had multiple atherosclerotic plaques. Based on the diagnostic criteria for SCI, which were

established based on a study and analysis of 133 cases [6], this case is in line with probable SCI, although the initial symptoms-weakness and numbness in the lower-left limb-differed from those typical of SCI. The patient's symptoms reflected acute, non-traumatic myelopathy, and his nadir deficits occurred 12 hours after the onset of symptoms. A study of 133 patients with SCI indicated that some patients' symptoms develop stutteringly [6]. The T2-weighted images showed symmetrical hyperintense lesions, similar to the "owl's eye" sign, with no indication of spinal cord compression. The CSF tests showed no signs of inflammation, which helped us to rule out alternative diagnoses. There is no definite treatment strategy for SCI. Our patient was treated with antiplatelet drugs, vitamins B_1 and B_{12}, and glucocorticoids. A report highlights that SCI patients are more likely to be discharged and show improvements after the initial treatment compared to cerebral infarction patients [20]. The SCI spread to a long segment, and we could observe dynamic changes in the spinal cord edema by MRI (**Figure 2**). The patient's prognosis was therefore poor.

Conclusion

In short, spinal vascular diseases sometimes present as cerebral stroke. To emphasize the occurrence of such rare diagnostic errors, we report two cases of spinal vascular diseases with similar single-limb neurological impairments. Clinicians should consider SSEH or SCI in the differential diagnosis of cerebral stroke.

Disclosure of conflict of interest

None.

Abbreviations

MRI, Magnetic resonance imaging; SSEH, Spontaneous spinal epidural hematoma; SCI, Spinal cord infarction; CTA, Computed tomography angiography; CT, Computed tomography; CSF, Cerebrospinal fluid; AQP4, Aquaporin 4; NMOSD, Neuromyelitis optica spectrum disorders.

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