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Acute Spinal-Cord Ischemia: Evolution of MRI Findings

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Background and Purpose Magnetic resonance (MR) findings in acute spinal-cord ischemia can be summarized as focal cord enlargement and hyperintensities on T2-weighted images and gadolinium enhancement, especially of the central gray matter. However, in analogy with acute brain ischemia, it is to be expected that the findings of MR imaging (MRI) may be normal in the first hours after symptom onset. We evaluated the clinical and MRI findings in a series of patients with acute spinal-cord ischemia, and tested the hypothesis that the development and course of MR abnormalities are predictable.

Methods Five patients with acute spinal-cord ischemia were admitted to our hospital over a 2-year period. Repeated MRI (1.5 T) was performed in all patients. Clinical data were retrieved from the patients' charts.

Results Four women and one man with a median age of 52 years (range, 31-75 years) were admitted. Three patients had anterior spinal artery infarction and two patients had transverse infarctions. All patients underwent spinal MRI within 24 hours; the findings were normal in four of the five patients. After 1-2 days, T2-weighted MRI generally exhibited focal cord enlargement and hyperintensity in all patients, while spinal-cord enhancement appeared after 2-11 days.

Conclusions Acute spinal-cord ischemia may have a typical course on MRI. MRI findings are usually normal in the acute phase, but spinal cord swelling and T2 abnormality are expected after several days, while gadolinium enhancement appears even later after symptom onset. The sensitivity and specificity of MRI can be increased by repeated MRI in patients suspected of acute spinal-cord ischemia.
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Introduction

Spinal-cord infarction is a cause of acute transverse myelopathy (ATM). The most likely diagnosis was found to be spinal-cord infarction in 14% of a large series of patients admitted with acute myelopathy.[1] Spinal-cord infarction is characterized clinically by sudden motor and sensory loss below the level of the spinal-cord injury. Different clinical subtypes have been recognized: anterior and posterior spinal artery syndrome due to radicular artery territory infarction, and central and transverse infarctions due to general spinal-cord hypoperfusion and ischemia.[2] Underlying causes include aortic disease and surgery, atherosclerosis, degenerative spinal disease, systemic hypotension, vertebral artery dissection, coagulopathy, trauma, and cocaine abuse. However, the cause has been reported to be idiopathic in 28-74% of cases.[2-4] The diagnosis of spinal-cord infarction depends on clinical symptoms and on magnetic resonance imaging (MRI) findings. Spinal-cord ischemia typically manifests in MRI as focal cord swelling and 'pencil-like' hyperintensities on T2-weighted images, according to both clinical studies[1,5,6] and textbooks.[7,8] However, as described in several studies, conventional MRI may be normal in some patients, especially in the acute phase.[3,9,10]

The development of magnetic resonance (MR) abnormalities in acute brain ischemia is well documented. Conventional MRI may show no or few abnormalities in the first hours after the onset of cerebral ischemia, and acute ischemia may be visible only on diffusion-weighted images. Brain abnor-

malities appear on T2-weighted images after several hours, and gadolinium enhancement becomes visible after about 3 days in the ischemic area due to damage to the blood-brain barrier. A similar pattern is likely to exist in the spinal cord. We hypothesize that repeated MRI of the spinal cord in spinal-cord ischemia reveals a predictable pattern of abnormalities, which may explain the previously reported 'normal' MR findings. To illustrate this, the findings of one patient with acute spinal-cord ischemia are described in greater detail.

Methods

Five patients with acute spinal cord infarction were admitted over a 2-year period (2005-2007) to the Department of Neurology of our hospital. MRI of the spinal cord was performed on all patients at least once using a 1.5-T Siemens Avanto or Symphony machine. The imaging protocol consisted of sagittal T1-weighted turbo spin-echo (TSE) sequences [echo train length (ETL), 3], with repetition times (TRs) varying from 450 to 700 ms, and an echo time (TE) of 14 ms. Sagittal T2-weighted imaging was performed using a standard proton density and a T2-weighted TSE sequence (ETL, 5) with TRs varying from 2500 to 3200 ms and TEs from 23 to 180 ms, respectively. Axial imaging consisted of T2*-weighted MEDIC imaging using TRs of 1500-1700 ms, depending on the number of axial images, and a TE of 27 ms. Axial T1 imaging consisted of a T1-weighted TSE (ETL, 3) sequence with a TR/TE combination of 600/15 ms. The sagittal and axial T1-weighted sequences were repeated after administration of DTPA-gadolinium.

In three patients (nos. 1, 3, and 5), diffusion-weighted imaging (DWI) of the spinal cord was also performed using a standard axial spin-echo sequence, and an echo-planar imaging sequence was also used for brain imaging. All images were acquired using a pixel size of 0.8-1.0 mm^2. Clinical data were retrieved from the patients' charts, and all MRI scans were reviewed by the same neuroradiologist.

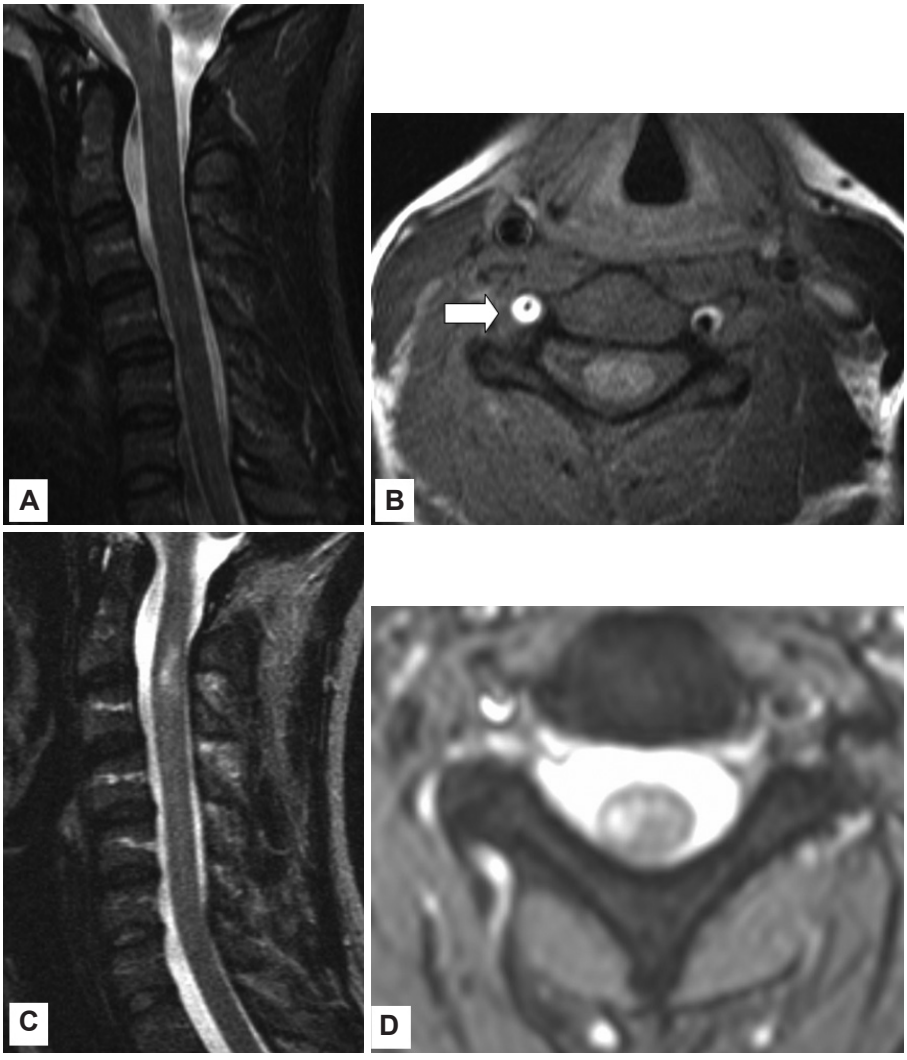

Fig. 1. A: MRI performed 12 hours after symptom onset: 31-year-old female presenting with right-sided Horner syndrome and right-sided loss of strength. Sagittal T2-weighted image showing no cord abnormalities. B: MRI performed 12 hours after symptom onset: axial T1-weighted image with fat suppression showing an abnormally high signal intensity (SI) in most of the cross-sectional area of the right vertebral artery, with a small central flow void (arrow), confirming suspected vertebral artery dissection. C and D: MRI performed 6 days after symptom onset: sagittal (C) and axial (D) T2-weighted images showing abnormal areas of high SI on the right at the C2 level.

Results

Case report

A 31-year-old woman (patient no. 1) with no medical history presented with rapidly progressive right-sided hemiplegia, ipsilateral loss of touch sensation below dermatome level C4-C5, and a right-sided Horner syndrome. She had no cerebellar signs, urine incontinence, or neck or radicular pain. Spinal MRI was performed approximately 12 hours after the onset of symptoms, revealing no abnormalities on T1- and T2-weighted images (Fig. 1A). However, there was an absence of flow voids and an abnormally high intramural signal intensity in the right vertebral artery, suggesting vertebral artery dissection (Fig. 1B). Laboratory investigations including cerebrospinal fluid analysis were normal. A diagnosis of probable acute cervical spinal-cord infarction due to vertebral artery dissection was made based on the imaging findings and clinical symptoms. MRI of the brain revealed no abnormalities. She was treated with antiplatelet therapy. The next day her muscle strength had improved slightly. Repeat MRI performed on day 6 revealed an increased signal in the spinal cord at the C2 level, some cord swelling on T1- and T2-weighted images, and abnormal contrast enhancement of the spinal cord (Fig. 1C-F). Furthermore, DWI revealed a high signal intensity with low apparent diffusion coefficient values at the affected level, further supporting the diagnosis of spinal-cord infarction (Fig. 1G and H). MRI conducted at 6 weeks after symptom onset revealed residual signal abnormalities at the C2 level on T2-weighted images, without contrast enhancement. After 2 months the patient had normal muscle strength and was left with only slight hyperesthesia of the left arm and leg.

Patient characteristics, and clinical findings

The characteristics and clinical data of the five patients (numbers 1-5) evaluated between 2005 and 2007 are listed in Table 1. Four patients were female and one was male. The median age was 52 years (range 31-75). Two patients had a history of cardiovascular disease. All of the patients presented with motor deficit and a spinothalamic sensory deficit, and two also had a lemniscal sensory deficit. Clinically, three of the patients had an anterior spinal-artery infarction and two had a transverse infarction; none of the patients had a posterior spinal-artery infarction. Two patients had an identifiable underlying cause for the spinal-cord ischemia: vertebral artery dis-

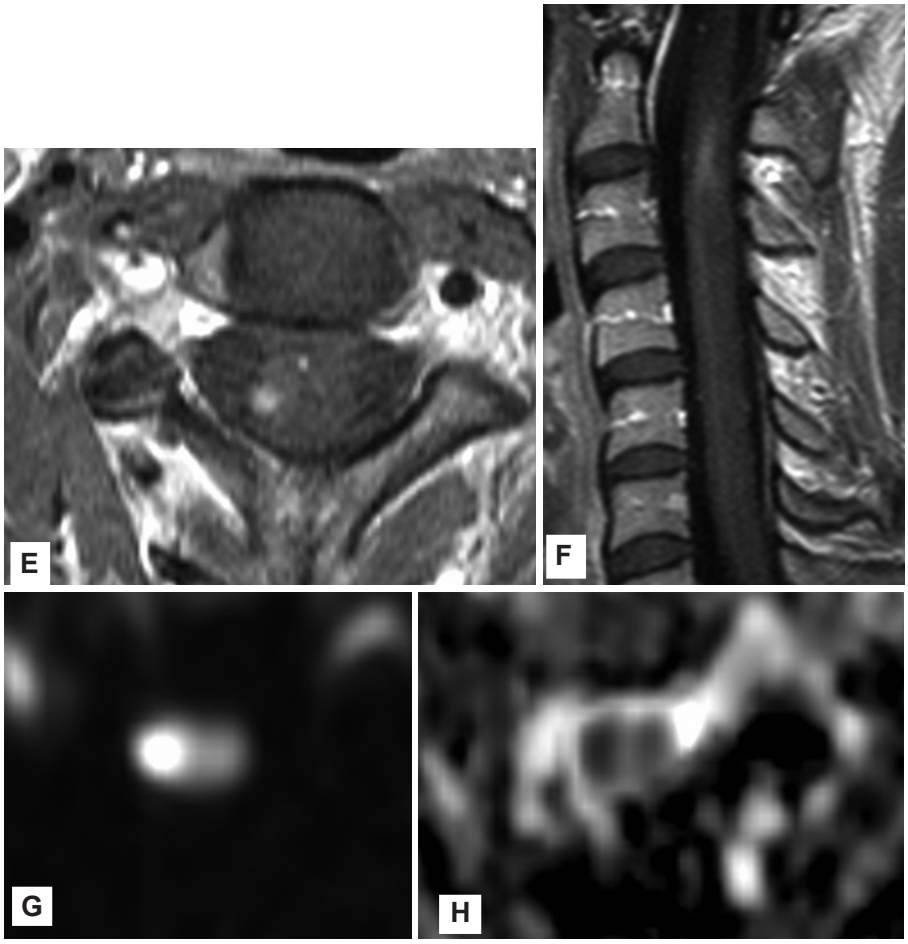

Fig. 1. E and F: MRI performed 6 days after symptom onset: axial (E) and sagittal (F) T1-weighted images after administration of gadolinium, revealing enhancement of the cord at the C2 level. G and H: MRI performed 6 days after symptom onset: axial (G) DWI showing right-sided high signal at the C2 level and axial (H) low apparent diffusion coefficient values showing diffusion restriction, supporting the diagnosis of spinal-cord ischemia. DWI: diffusion-weighted imaging, MRI: magnetic resonance imaging.

Table 1. Patient characteristics and clinical data

Patient	Age	Sex	Cardio-vascular history	Cardio-vascular risk factors	Level of ischemia	Spinal cord syndrome	Cause	Clinical symptoms	Outcome at follow-up (MRS)
1	31	F	-	-	C3-C4	Anterior	Right vertebral artery dissection	Right sided hemiparesis Right sided Horner syndrome SSD Normal reflexes	Normal muscle strength, slight hyperesthesia left arm and leg (1)
2	31	F	-	+	Conus	Transverse	Mild trauma?	Paraparesis legs SSD, LSD UI Areflexia legs Indifferent plantar reflex Absent anal reflex	Paraparesis legs, SSD and LSD legs sphincter abnormalities (4)
3	52	F	-	+	Th10-Th12	Anterior	No cause	Paraparesis legs SSD UI Brisk reflexes legs Extensor plantar reflex	Paraparesis legs, SSD legs, urine incontinence (4)
4	54	M	+	+	Conus	Transverse	Thrombosis aorta	Paraplegia legs SSD, LSD UI, FI Areflexia legs Indifferent plantar reflex	Paraplegia legs, SSD legs, urine and fecal incontinence (4)
5	75	F	+	+	Th9	Anterior	Atherosclerosis aorta?	Paraplegia legs SSD UI, FI Areflexia legs	Paraplegia legs, SSD legs, urine and fecal incontinence (4)

FI: faecal incontinence, LSD: lemniscal sensory deficit, MRS: Modified Rankin Scale, SSD: spinothalamic sensory deficit, UI: urinary incontinence.

Table 2. MR imaging results

Patient	Level of ischemia	Time of scan	Hyper-intensity T2	Cord enlargement	Contrast enhancement	Diffusion restriction (DWI)
1	C3-C4	12 hours	-	-		
		Day 6	+	+	+	+
2	Conus	8 hours	-	-		
		Day 4	+	+	-	
		Day 11	+	+	+	
3	Th10-Th12	12 hours	-	-		
		Day 4	+	+	+	+
4	Conus	14 hours	+	+	-	
		Day 7	+	+	+	
5	Th9	15 hours	-	-		
		Day 2	+	+	+	
		Day 6	+	+	+	+

DWI: diffusion-weighted imaging, MR: magnetic resonance.

section in one patient and acute occlusion of the aorta in the other. In two patients the cause was less obvious: one patient had extensive atherosclerosis of the aorta with occlusion of the lumbar arteries at the affected level, and the other patient had suffered a trauma on the day of symptom onset. No identifiable cause was found at all in the remaining patient. Detailed clinical information obtained at follow-up is given in Table 2, demonstrating the poor prognosis in these patients.

MRI

The MRI results are summarized in Table 2. All patients were first examined by MRI within 24 hours after symptom onset, yielding normal results in four of the five patients. MR abnormalities were seen on T2-weighted images at 14 hours after symptom onset in one patient. In this case a thrombus in the aorta caused ATM with ischemia of the conus and severe neurologic deficit. Given the sudden onset and the observed vascular risk factors, we concluded that the cause was spinal-cord infarction, although MRI had already produced hyperintensities on T2-weighted images. There was no contrast enhancement. All patients received one or more repeat MRI examinations, which revealed a high signal intensity and focal cord enlargement in all patients on T2-weighted images acquired at least 2 days after the onset of symptoms. Gadolinium enhancement was seen on all MR examinations at least 2 days after symptom onset. DWI was performed in three patients (numbers 1, 3, and 5, after 4-6 days) revealing diffusion restriction in the ischemic area, thus confirming the presence of ischemia.

Discussion

The findings of our study suggest that spinal-cord ischemia has a predictable course on repeated MRI. MRI findings are usually normal in the acute phase, whereas spinal-cord edema and T2 abnormalities may be expected after 1-2 days. Gadolinium enhancement appears even later after symptom onset.

The incidence of spinal-cord infarction is low. In our center we saw 5 patients over a 2-year period, compared to 600 stroke patients in the same period. In the setting of ATM, the main reason for performing MRI is to rule out treatable causes such as compression by disc herniation. About half of cases of ATM are caused by idiopathic myelitis,[1,11] which may be the presenting symptom of multiple sclerosis in about 50% of such cases.[1] In the acute phase, acute myelitis always produces a high signal intensity on T2-weighted images, while gadolinium enhancement is seen in about 62-84% of acute cases.[1,11] Since patients with acute ischemic myelopathy may have normal MR images in the acute phase, this may help in differentiating between myelitis and ischemic myelopathy. Other, more uncommon causes of ATM include myelitis related to systemic inflammatory diseases such as systemic lupus erythematosus or Sjögren's disease, and other vascular causes such as arteriovenous malformation or spinal dural arteriovenous fistula. These conditions also show clear MR abnormalities in the acute phase; in our opinion, normal MRI findings in the acute phase could greatly help in differentiating between spinal-cord ischemia of other etiologies. Of course, this only holds true when patients are imaged within several hours (in our patients within 15 hours) after symptom onset, which is dependent upon MR availability.

In a previous series of spinal-cord ischemia cases the MRI findings were generally found to be normal, and this was interpreted as a pitfall in the diagnosis of cord infarction. However, in some other reported cases, repeated MRI revealed MR abnormalities, as in our patients. As reported by Weidauer et al.,[5] early MRI (i.e., within 3 hours) may show no abnormalities, while later on hyperintensities will be seen on T2-weight-

ed images, such as swelling of the spinal cord. We saw in our series the same sequential MR abnormalities as reported by Weidauer et al.[5] Thus, an early normal MRI in a patient suspected of spinal-cord ischemia supports the diagnosis. We therefore recommend repeated imaging in these patients in order to detect the sequential MRI changes seen in our series.

The absence of T2 abnormalities in patients with acute ischemia (both in the brain and spinal cord) may be due to an abnormal increase of T2 due to cytotoxic edema, which takes some time to develop. Another possible reason for the absence of T2 abnormalities in acute spinal-cord ischemia is the presence of a good collateral blood supply in the affected region, causing partial infarcts with slow evolution of MR changes. We believe that although conventional T2 imaging has a low sensitivity for acute ischemic changes, this could actually be beneficial to diagnosing spinal-cord ischemia, because other conditions causing ATM usually show abnormalities in the acute phase. Early detection of spinal-cord infarction may of course be improved by using DWI, which can reveal spinal-cord ischemia in the acute stage when T2-weighted imaging fails to do so.[12]

DWI is the most powerful tool for detecting cytotoxic edema in infarction, but its application in spinal-cord lesions has been limited for technical reasons (susceptibility artifacts due to the movement and small size of the spinal cord), and there are only a few reports of useful DW-MRI sequences in the imaging of spinal-cord ischemia.[6,13,14] DWI was performed in three of our patients (nos. 1, 3, and 5), albeit after up to 4 days after symptom onset, and diffusion restriction was indeed seen, illustrating the potential use of DWI. Nevertheless, in the absence of DWI, our findings show that conventional MR techniques can suggest spinal-cord ischemia based on the typical sequential appearance of abnormalities.

A shortcoming of our study is that we did not image all patients using the same MR protocol and at a standard time after symptom onset, which limits the interpretation of our findings. Future studies should prospectively investigate all ATM patients using the same MR protocol at fixed time points, in order to confirm our hypothesis.

The findings of our study suggest that the development of MR abnormalities in spinal-cord ischemia has a predictable course. Just as in cerebral infarction, the findings of conventional MRI may be normal in the acute phase. Hyperintensities on T2-weighted images and focal cord enlargement are expected after 1-2 days, followed even later by cord enhancement after gadolinium administration. Therefore, the sensitivity and specificity of the diagnostic process could be improved by applying MRI in the acute phase (including administration of gadolinium and if possible with application of DWI), and repeated MRI may confirm the diagnosis in patients suspected of spinal-cord ischemia.

Conflicts of Interest

The authors have no financial conflicts of interest.

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