Vertebral Body Infarction as a Confirmatory Sign of Spinal Cord Ischemic Stroke
Report of Three Cases and Review of the Literature

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Background — Acute spontaneous spinal cord syndromes often remain etiologically ambiguous despite extensive diagnostic efforts. In the previous literature five cases are described with acute spinal cord syndromes interpreted as spinal cord ischemic strokes because of association with vertebral body infarctions on MRI.

Case Descriptions — Three cases are presented, and the literature is reviewed. In addition to an extensive diagnostic battery including an initial MRI without pathological signs, follow-up MRI at different time intervals from the onset of symptoms showed T2 hyperintense signals in vertebral bodies. Patient 1, who had plaques in the abdominal aorta, had suffered a thoracolumbar spinal infarction; this and a concomitant infarction of the left portion of T-12 could be demonstrated on follow-up MRI on day 12. Patient 2, who had incomplete transverse spinal artery syndrome below T-3, had an abnormal signal at the T-2 level of the spinal cord on follow-up MRI on day 5; this was one segment above infarction of the dorsal area of T-3, corresponding to the ascending course of the medullary artery. The spinal cord of patient 3, who had a posterior spinal artery syndrome below T-11, was unremarkable on follow-up MRI on day 14, but a T2 hyperintense signal was noted in the dorsal area of T-10.

Conclusions — Vertebral body infarction represents the only confirmatory sign for the otherwise exclusionary diagnostic procedure for spinal cord ischemic stroke and must be searched for on follow-up MRI as a key to correct diagnosis. (Stroke. 1998;29:239-243.)

Key Words: magnetic resonance imaging ■ spinal cord stroke ■ vertebral body infarction

In comparison to cerebral infarcts, those of the spinal cord are infrequent or remain undiagnosed as “acute myelopathies of unknown etiology.” Acute nontraumatic complete or partial transverse myelopathies as well as classic vascular spinal cord syndromes are etiologically ambiguous and require well-established diagnostic procedures. Radiological methods such as roentgenography of the spine, CT, MRI, or myelography rule out the numerous causes of spinal cord compression due to extramedullary or intramedullary pathologies or spinal vascular malformations; a CSF examination may prove but not exclude an inflammatory/demyelinative cause. If extensive exclusionary diagnostic efforts are inconclusive, the diagnostic dilemma remains between vascular myelopathy or myelitis with normal CSF. Additional signs such as plaques, aneurysms, or dissection of the aorta or a generalized vascular disease involving other organs as well may favor but not prove the existence of a vascular lesion. However, despite an extensive diagnostic procedure an etiological diagnosis is often impossible (in up to 59% of cases in a recent study), at least at the time of presentation of the disease.

Since 1991 five cases have been published regarding the vascular nature of an acute spinal cord syndrome confirmed by MRI of a corresponding vertebral body infarction. The first report emphasized the significance of this sign: “It is difficult to conceive of a single pathological process other than a vascular event that can affect acutely both the spinal cord and the spinal column.” Therefore, we consider it important to report three additional cases since this vertebral body infarction represents a confirmatory sign for the otherwise exclusionary diagnostic procedure for presumed spinal cord infarctions.

Case 1

In the morning a 66-year-old man suddenly felt burning pain in his left leg followed by a weakness of both legs that progressed over 15 minutes and an inability to walk. A heavy smoker of 20 to 30 cigarettes daily, the subject had suffered for years from intermittent claudication, recently with a walking distance of approximately 200 m. Thirty years ago he had a partial gastrectomy because of gastric ulcer.

On admission the subject had a flaccid paralysis of the left leg, paresis of the left abdominal musculature, and a uniform paresis of the right leg graded 2/5 on the MRC scale. The anal sphincter was flaccid, tendon reflexes were abolished at the legs, Babinski’s response was absent, and cutaneous abdominal reflexes were elicited in the upper segment only. There was loss of pain and temperature sensation below L-3 on the left and below S-1 on the right side, with slightly diminished vibration sense but a normal sense of touch and position. He was unable to urinate, with residual urine of 500 mL. We noted blood pressure of 170/
90 mm Hg on both sides, regular heart rate of 88 beats per minute, no palpation of the pulses of the arteries of the left foot, and auscultation with bruit at the right femoral artery. Laboratory investigations were without significant results.

The x-ray film of the thoracic and lumbar spine showed degenerative alterations, and the x-ray film of the chest showed sclerosis of the aorta. Abdominal sonography ruled out an aneurysm or dissection of the abdominal aorta, but significant atheromatous plaques were noted. MRI of the thoracic and lumbar spine ruled out a compression of the cauda or spinal cord, and the medullary signals showed no significant abnormality.

After an infarction of the great anterior medullary artery (or, in another nomenclature, the great “radicular” artery of Adamkiewicz [AA]) was diagnosed, the patient was given 48 mg IV dexamethasone and placed on dexamethasone 8 mg TID (later reduced and discontinued) for 12 days, subcutaneous heparin, and intensive physiotherapy.

Follow-up-MRI 12 days after admission showed a longitudinal T2 hyperintense signal from the conus to the lower thoracic level and an evident T2 hyperintense bone marrow signal in the left portion of T-12 (Fig 1).

The patient was placed on ticlopidine BID. He improved rapidly within the first days and gradually thereafter. Four weeks later he still had a left accentuated paraparesis with incontinence of bowel and bladder, but he was able to walk with a walker and was transferred to a rehabilitation center.

Case 2

A 51-year-old otherwise healthy woman noticed sudden numbness of both feet the evening before admission. This sensory disturbance had extended to the navel until midnight. Until the time of admission to our hospital the subject experienced progressive weakness in both legs and an inability to urinate.

On admission, approximately 10 hours after the onset of symptoms, the patient had a flaccid asymmetrical paraparesis with uniform paresis of the right leg graded 3/5 on the MRC scale; the left leg had proximal paresis graded 4/5 and normal distal power graded 5/5. Tendon reflexes were exaggerated at the legs, Babinski’s sign on both sides was positive, and the sensory level beginning at T-3 revealed dysesthesia and hyperesthesia to T-2. Below this level we noted a marked hypalgesia, normal function of vibration sense and posture, diminished rectal tone, and residual urine of 900 mL.

Results were negative for x-ray of the whole spine and chest; abdominal sonography; ECG; transesophageal ultrasound of the heart, aortic arch, and descending aorta; lumbar myelography including the cervical level; MRI of cervical and thoracic spine; CSF including a search for oligoclonal bands; all laboratory data including a serological test for syphilis; immunoelectrophoresis; C3 and C4 complement; antinuclear antibodies, double-chain DNA antibodies, extended coagulation screening, cardiolipin antibodies, C-reactive protein, angiotensin-converting enzyme, lycosyn, and visual and acoustic evoked potentials. Only the somatosensory evoked potentials of the tibial nerve showed reduced cortical amplitudes with normal latencies.

Because acute myelitis was suspected, the patient was placed on 8 mg dexamethasone TID, 7500 U SC heparin BID, and intensive physiotherapy.

Follow-up MRI performed on the fourth hospital day (Fig 2A) showed T2 hyperintense signals in the dorsal area of T-3 and a suspicious medullary signal at T-2. In an additional

Figure 1. Case 1. MRI (1.0 T) of the thoracolumbar region on day 12 (TR, 5000 ms; TE, 90 ms; echo train length,15). A, Sagittal images show increased signal in the central region of the spinal cord extending from conus to lower thoracic cord (arrowhead), corresponding to spinal infarction. B, Left parasagittal image shows abnormal bone marrow signal in the left part of T-12, corresponding to partial vertebral body infarction. C, Axial image shows the location of the vertebral body infarction in the region supplied by left anterior central arteries (see Fig 4). The hyperintense signal in the spinal cord corresponds to spinal infarction. Additionally, there are thrombotic plaques in the abdominal aorta (arrowhead).
follow-up MRI 1 day later (Fig 2B and 2C), there was a definite intramedullary hyperintense signal in T2-weighted images on the T-2 level, which was one segment above the abnormal vertebral body signal. On the basis of these MRI data, we diagnosed spinal cord ischemia and placed her on 300 mg/d acetylsalicylic acid. She did not improve significantly and was transferred to a rehabilitation center.

On a clinical follow-up examination 3 months later, the patient suffered from slight proximal paresis of the right leg graded 4+1/5, tendon reflexes were exaggerated with positive Babinski’s sign, and there was dissociated sensory loss with impaired pain and temperature below T-7. The patient had sufficient bladder function only when treated with distigmin bromide, and she had a nondisabling spastic gait. Repeated supplementary tests (MRI, electrophysiology, and CSF) were refused by the patient.

Case 3

A 49-year-old healthy woman awakened during the night with a sudden but transient back pain. She noticed an odd numbness and slight weakness of her left leg but returned to sleep. In the course of the following day she noticed a painful burning in both legs and an instability of gait. Approximately 20 hours after onset of symptoms she was admitted to a neurological department. Myelography and initial CSF data were unremarkable. An MRI on the third hospital day showed a possible abnormal signal in the thoracic spinal cord. An incidental finding was a calcified structure behind the body of T-9 without impression of the subarachnoid space or enhancement of gadolinium, possibly a calcified disk protrusion or a small meningioma (Fig 3). The patient was transferred to our neurological department.

On admission she had a slight paresis of the left leg with a uniform weakness graded 4/5 on the MRC scale; exaggerated tendon reflexes of the legs without Babinski’s sign; a dysesthesia below T-11; markedly diminished sense of vibration, posture, and cutaneous localization with well-preserved sensation of pain and temperature; and sensory ataxia. Her bladder and bowel were intact.

Laboratory data (same battery as in case 2) and CSF examination, visual and acoustic evoked potentials, and x-ray film of the chest were all within normal limits; abdominal sonography ruled out pathologies of the abdominal aorta. Tibial somatosensory evoked potentials showed normal lumbar and cortical potentials by right-sided stimulation and regular lumbar but significantly reduced cortical potentials by left-sided stimulation. MRI of the brain was normal.

The patient was treated with 1000 mg IV prednisone on admission; this dosage was reduced and was discontinued within 12 days. Physiotherapy was performed daily.

Follow-up MRI of the spine on day 14 showed a T2 hyperintense signal in the dorsal region of T-10 with an unremarkable signaling of the spinal cord (Fig 3B). The patient improved slowly and was transferred to a rehabilitation center.

We received a report concerning the patient’s status 3 months later, describing a still abnormal gait as a nondisabling sensory ataxia and a slight dysesthesia approximately below T-11, exaggerated tendon reflexes in the legs with left-sided accentuation, and ankle clonus without Babinski’s phenomenon.

Discussion

A vascular event was evident in case 1 after we excluded a compressive cause and considered the history of arterial occlusive disease of the legs in this patient. The abnormal MRI signal in the thoracolumbar cord 12 days after onset of symptoms corresponds to the supply region of the AA. This vessel usually is the largest and most constant of the anterior medullary arteries, although it is generally but incorrectly referred to as the great radicular artery.9,10 These anterior medullary arteries originate unilaterally from the spinal branches of the paired segmental arteries branching off the aorta and contribute blood to the anterior spinal artery in a
variable number of 6 to 10 posterior medullary arteries feed into the posterior spinal artery. The AA reaches the cord at a low thoracic or upper lumbar level, lengthening the course of a spinal branch usually derived from a left segmental artery. 

In contrast to this well-described arterial supply of the spinal cord, the neurological literature makes few references to the vascular supply of the vertebral column. From each of the segmental arteries or the regional equivalents, the ventral and lateral parts of the vertebral body receive nutritional vessels termed anterior central branches; the dorsal part of the vertebral body is supplied by the posterior central branches of the spinal branch (Fig 4). The postlaminar and prelaminar branches supplying the vertebral arch are not considered in this respect.

The abnormal bone marrow signal of T-12 in case 1 (Fig 1) corresponds to the supply region of the anterior central branches of the left segmental artery. Thus, the following pathogenesis concerning the thoracolumbar infarct of case 1 may be accepted: the large atheromatous plaques in the abdominal aorta (Fig 1C) cause an occluding microatheroma or embolization of the segmental artery, resulting in ischemia in the region of the anterior central branches and the AA. Apparently there was sufficient collateralization from the posterior spinal artery and lumbar arteries, resulting in a predominating anterior spinal artery syndrome of the thoracolumbar cord. The comparatively good outcome of this patient is in accordance with cases reported in the literature.

The patient in case 2 had no risk factors, signs, or symptoms of a vascular disease. After exclusion of a compressive cord lesion, myelitis was first considered despite normal CSF levels. Follow-up MRI at the level suspected from the clinical examination showed an abnormal bone marrow signal in the dorsal part of T-3. This was one segment below the medullary signal (Fig 2B and 2C), thus confirming a vascular origin of the spinal cord syndrome. The segmental difference between the lesion in the vertebral body and the spinal cord is a consequence of the ascending course of the medullary artery following the nerve roots with increasing obliquity from cranial to caudal levels. The posterior central branches, supplying the dorsal parts of the vertebral body, derive from the spinal branch artery at the level of the intervertebral foramen (Fig 4).

Case 3 describes a posterior spinal artery syndrome. At first there was no possibility of distinguishing between a myelitic and a vascular lesion on the basis of the abnormal data of the clinical and electrophysiological examinations. Follow-up MRI of the spine 14 days later revealed a T2 hyperintense signal in the dorsal part of T-10 (Fig 3B). We consider this unequivocal sign corresponding to the sensory level to be a strong argument for a vascular lesion.

We are able to demonstrate the vascular nature of the spinal cord syndrome in our last case only bearing in mind the
**Review of Patients With Vertebral Body Infarctions Confirming Spinal Vascular Syndrome**

<table>
<thead>
<tr>
<th>Author</th>
<th>Age/Sex</th>
<th>Neurological Symptoms</th>
<th>Possible Cause/Concomitant Disease</th>
<th>Interval to MRI From Onset</th>
<th>Location of Spinal Cord Signal</th>
<th>Vertebral Body Infarctions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mikulis et al⁴⁵</td>
<td>61/F</td>
<td>Paraplegia below T-12</td>
<td>Fibrocartilaginous embolus</td>
<td>5 d</td>
<td>Conus</td>
<td>None</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>12 d</td>
<td>Conus</td>
<td>T-11</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>21 d</td>
<td>T-8 to conus</td>
<td>T-11</td>
</tr>
<tr>
<td>Yuh et al⁶</td>
<td>76/M</td>
<td>Not described</td>
<td>Aortoiliac bypass</td>
<td>8 h</td>
<td>T-11 to L-1</td>
<td>T-12 and L-1</td>
</tr>
<tr>
<td></td>
<td>64/M</td>
<td>Not described</td>
<td>de Bakey III</td>
<td>48 h</td>
<td>Thoracolumbar</td>
<td>Multiple lower thoracic vertebrae</td>
</tr>
<tr>
<td>Haddad et al⁷</td>
<td>69/M</td>
<td>Not described</td>
<td>Aortofemoral bypass</td>
<td>10 h</td>
<td>Unable to evaluate</td>
<td>L-4</td>
</tr>
<tr>
<td></td>
<td>58/M</td>
<td>Paraplegia below T-9</td>
<td>Ethanol injection of 7th a. 9th intercostal nerve for pain relief</td>
<td>18 h</td>
<td>None</td>
<td>T-8</td>
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<td>7 d</td>
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<td></td>
<td>4 wk</td>
<td>T-8 level</td>
<td>T-8</td>
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<td>Present study</td>
<td>66/M</td>
<td>Thoracolumbar infarct, anterior spinal artery syndrome</td>
<td>Intermittent claudication, aortic plaques</td>
<td>Hours after admission</td>
<td>Unable to evaluate</td>
<td>None</td>
</tr>
<tr>
<td></td>
<td>51/F</td>
<td>Incomplete sensorimotor spinal syndrome below T-3</td>
<td>None</td>
<td>12 d</td>
<td>Conus to lower thoracic cord</td>
<td>T-12</td>
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<td></td>
<td>Hours after admission</td>
<td>None</td>
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</tr>
<tr>
<td></td>
<td>49/F</td>
<td>Posterior spinal artery syndrome below T-11</td>
<td>None</td>
<td>4 d</td>
<td>T-2 suspicious</td>
<td>T-3</td>
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<td>5 d</td>
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<td>T-3</td>
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<td></td>
<td></td>
<td></td>
<td>14 d</td>
<td>None</td>
<td>T-10</td>
</tr>
</tbody>
</table>

Conditions of cases 1 and 2 and of the five cases described in the literature (Table). Comparable to our case 1, all patients of Yuh et al⁶ had significant aortic diseases. The patient of Haddad et al⁷ had a histologically proven fibrocartilaginous embolus. This is a mechanism of spinal cord infarction that is probably not rare but has never been diagnosed on clinical grounds. ⁸ It may be considered a cause particularly in our case 3, who had a calcified thoracic disk protrusion or small meningioma in the vicinity of the infarcted vertebra.

Of course, the abnormal MRI findings of vertebral bodies described are not specific for infarction. They are only an indication of increased water content of the osseous tissue and may also be seen in fractures, metastases, or infections of the vertebral bodies. But aside from limitation to the vascular supply regions in our cases, an initial normal MRI could be demonstrated. Although the time course of the abnormal bone marrow signal in MRI indicating vertebral body infarction⁹ is not known, it is a strong argument for spinal ischemia if there is an evolution of signal abnormalities in a vertebral body associated with spinal cord syndromes at an appropriate level.⁹ To establish the time course of this bone marrow signal in vertebral body infarction and perhaps the additional value of gadolinium enhancement, further studies are required. Lack of this information may be responsible for the limited number of patients reported and the unawareness to search for this sign. However, a spinal cord infarction may not always be accompanied by vertebral body infarction, because arterial occlusion may be located distal to those vessels supplying the vertebral body or because of the good collateral blood supply of the vertebra. Nevertheless, we reported these three cases to promote awareness of this additional sign, which may be the key to the correct diagnosis in enigmatic acute spinal cord syndromes.

**References**


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