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## Posterior Spinal Artery Infarct

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**Summary:** Acute medullary syndrome developed in a patient in whom glue had been inadvertently injected into the right posterior radiculomedullary artery during endovascular occlusion of a spinal dural arteriovenous fistula at T-11. MR imaging 40 days after the procedure showed signal changes and contrast enhancement in the posterior and right lateral column at T10–11. Circumscribed signal changes in the same areas without contrast enhancement were seen 4 months later. MR imaging was able to show this posterior spinal artery infarct.

Spinal cord infarcts are uncommon lesions that can occur in the territories of the anterior or posterior spinal arteries, or both (1). Infarcts in the territories of posterior spinal arteries are rare; Kaneki et al (2) found fewer than 30 cases reported in the literature. We describe the acute and follow-up magnetic resonance (MR) imaging findings of posterior spinal infarct.

### Case Report

A 73-year-old man had a 6-month history of progressive paraparesis and paresthesias of the left lower extremity and saddle region. MR imaging showed diffuse central signal change in the thoracolumbar spinal cord and retromedullary punctate areas of signal void (Fig 1A–C). After intravenous administration of gadopentetate dimeglumine, the affected spinal cord and serpiginous retromedullary structures enhanced moderately. Abnormal retromedullary vessels were confirmed by phase-contrast MR angiography. Spinal selective arteriography showed a dural arteriovenous fistula fed by the right T-11 intercostal artery (Fig 1D). Endovascular treatment was performed after superselective catheterization of the right T-11 intercostal artery with injection of 1 mL of histoacryl-iodized oil 25% mixture. This obliterated the fistula and its intradural draining vein, but also a posterior radiculomedullary artery arising from the same vascular pedicle proximal to the fistula (Fig 1E and F), which had not been seen in the selective study. A few hours later, the patient experienced a transverse medullary syndrome. Neurologic examination showed ataxia and abnormal gait; severe lower limb weakness, more pronounced on the right; disappearance of the ankle reflexes and reduction of the right patellar reflex; hypopallesthesia and reduced proprioception of the lower extremities; and thermal and pain hypoesthesia, more pronounced on the right at the T-12 level. MR imaging 40 days after the procedure showed disappearance of the diffuse central signal changes. Circumscribed areas of signal change, which enhanced after contrast administration, were observed in the posterior columns at T-11 (Fig 1G) and in the right posterior column, posterior gray horn, and posterior

portion of the lateral column at T-10. No perimedullary vessels were seen at MR imaging or MR angiography. Four months after embolization, the patient could walk with a cane, and neurologic examination showed a deficit of vibratory sensation and of position sense to lower limbs. MR imaging showed sharply defined signal changes at T10–11 (Fig 1H) involving the posterior columns (Fig 1I) and the posterior portion of the right lateral column (Fig 1J). The lesion did not enhance after contrast administration. No perimedullary vessels were seen at MR imaging or MR angiography.

### Discussion

According to Gillilan (3), the paired posterior spinal arteries lying in the posterolateral sulci of the spinal cord are fed by 10 to 20 posterior radiculomedullary arteries. These vessels are smaller than the seven to 10 anterior radiculomedullary arteries (3) and, because of their size, are difficult to see on angiograms, except in the thoracolumbar regions, where they are larger (4, 5). In the adult, the posterior spinal arteries do not coalesce to form a continuous arterial axis on the surface of the cord, as their anterior counterparts do (5). In some instances, the posterior spinal arteries of both sides are supplied by a single radiculomedullary vessel (5). Although interpersonal differences may be observed in the arterial arrangements of the extraspinal and intraspinal cord (5), the arterial territories fed by the posterior spinal arteries usually encompass the posterior column, posterior horns, and posterolateral portion of the lateral column (5, 6).

Posterior spinal artery infarcts are rare (2); their causes include syphilitic arteritis (7, 8), cholesterol emboli from atheromatous aortic plaques (9), intrathecal injection of phenol (10), vertebral artery dissection (6), and plasmocytoma (11); but in many cases, their pathogenesis remains unknown (2). The majority of posterior spinal artery infarcts occur at the thoracolumbar level (2, 7–9) but they have been observed at the thoracic (10–12) and cervical (6, 13, 14) levels, too. Longitudinal extension of these infarcts averages two vertebral segments, but their span can range from one (2) to six (12) segments. In the transverse plane, the more extensive lesions involve the posterior portion of the lateral column, the posterior column, and the posterior gray horns, either bilaterally or unilaterally. However, selective lesions of the posterior gray horn and lateral (2) and posterior (7, 8) column have been described. The histologic features and evolution of spinal cord infarcts resemble those of cerebral infarcts (15).

Clinical features are variable (9), and include loss of vibratory sensation and proprioception due to damage of the posterior column, suspended global anesthesia and segmental deep tendon areflexia due to posterior horn involvement, and paresis below the level at which the posterior portion of the

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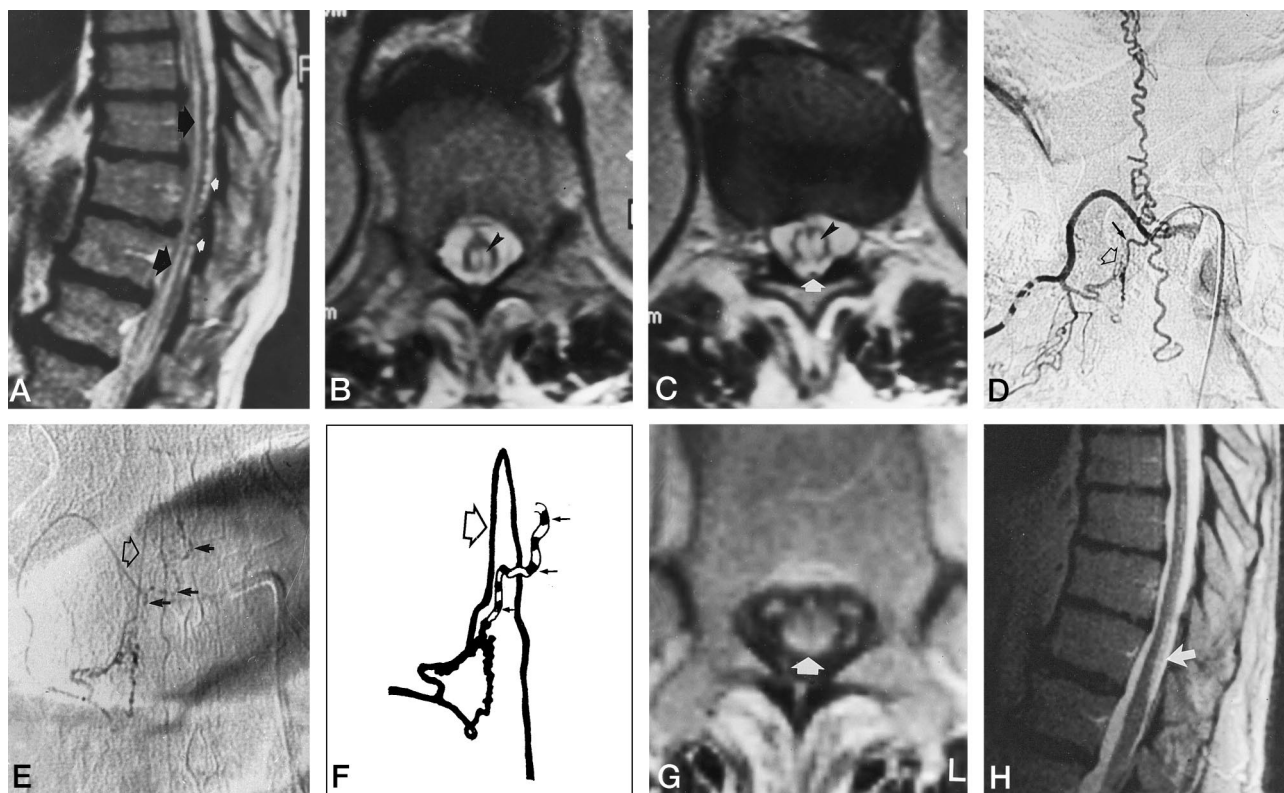


FIG 1.

A 73-year-old man with progressive paraparesis and paresthesias of the left lower extremity and saddle region.

A, Sagittal T2 weighted (3000/120/10 [repetition time/echo time/excitations]) fast spin-echo MR image shows diffuse central high signal intensity in the thoracolumbar spinal cord (*black arrows*) and retromedullary punctate areas of signal void (*white arrows*).

B, Axial T2-weighted (5800/120/8) fast spin-echo image at T-11 shows central signal change (*arrowhead*).

C, Axial T2-weighted (5800/120/8) fast spin-echo image at T11-12 shows central signal change (*arrowhead*) and a punctate retromedullary area of signal void (*white arrow*).

D, Spinal arteriography (anteroposterior projection) with selective catheterization of the right T-11 intercostal artery shows a dural arteriovenous fistula (*open arrow*) and its ascending sinus intradural draining vein (*closed arrow*) joining dilated perimedullary veins.

E, Digital radiograph after superselective injection of 1 mL of histoacryl-iodized oil 25% mixture in right T-11 intercostal artery shows radiopaque material filling the feeding artery and the fistula but also a posterior medullary artery with the typical hairpin shape (*open arrow*) arising from the same vascular pedicle proximal to the fistula. Particulate radiopaque material is also present in the intradural draining vein (*closed arrows*).

F, Diagram of E. *Black lines* indicate arteries and dural arteriovenous fistula; *open arrow*, right posterior medullary artery; *hatched areas*, intradural draining vein containing particulate radiopaque material (*closed arrows*).

G, Axial T1-weighted (540/25/4) spin-echo image after intravenous administration of 0.1 mmol/kg of gadopentetate dimeglumine obtained 40 days after the procedure shows moderate contrast enhancement in the posterior columns (*arrow*) at T-11.

H, Sagittal T2-weighted (4500/120/6) fast spin-echo image obtained 4 months after endovascular therapy shows sharply defined elongated high signal intensity of the posterior spinal cord (*arrow*) at T10-11.

I and J, Axial T2-weighted (5000/120/6) fast spin-echo images show sharply defined signal changes (*arrows*) involving the posterior columns at T-11 (I) and the right posterior column and posterior portion of the right lateral column at T-10 (J).

lateral column containing the crossed corticospinal tract is affected. In our patient, the posterior spinal artery infarct was caused by inadvertent glue injection into a posterior radiculomedullary artery during superselective embolization of a dural arteriovenous fistula.

The MR signal and enhancement features of spinal cord infarct are nonspecific, and a similar distribution of signal changes in the lateral and posterior column and the posterior gray horn can be observed in other acute diseases of the spinal cord, including infectious myelitis (16), myelitis due to collagen diseases (17), and multiple sclerosis (18). Hence, a diagnosis of

posterior spinal cord infarct based on MR imaging findings alone may be impossible. Correlation with clinical history and findings, blood and CSF laboratory data, brain MR imaging, and, eventually, follow-up MR imaging of the spinal cord lesion is necessary to establish the correct diagnosis.

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