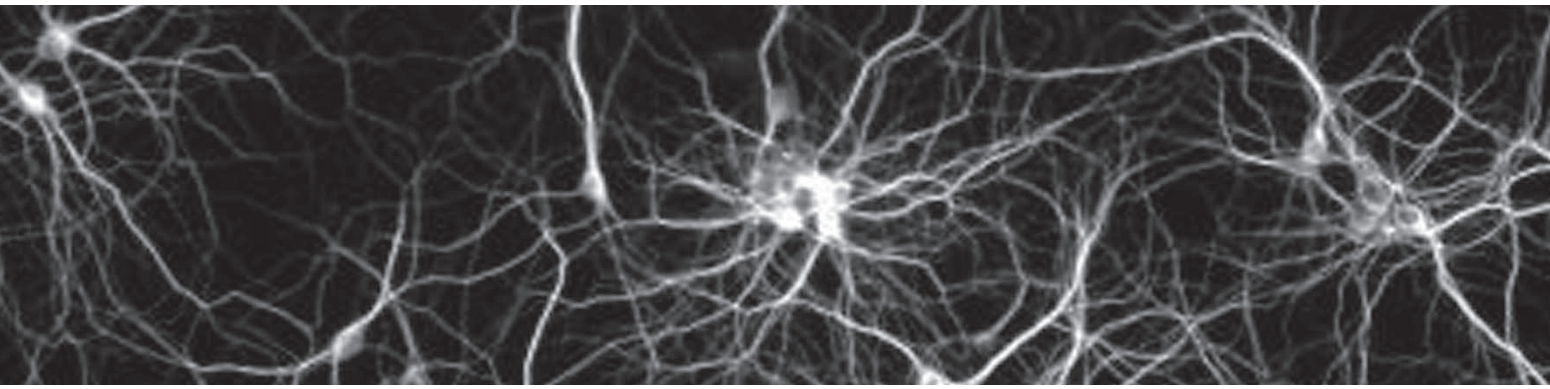

Vertebral body infarction as a sign of spinal cord ischemic stroke

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A 51-year-old male with a history of arterial hypertension and anxiety attacks presented to the emergency room with pain in his lower back and numbness and weakness in his left leg. The symptoms started on the same day upon waking up, and the numbness spread from his leg to the genital area during the day. He reported hesitancy and loss of feeling of his urine passing during micturition and had no erection that morning. His medications included bisoprolol/perindopril 5/5 mg daily and escitalopram 10 mg daily. Neurological examination revealed left leg weakness 4/5, with an extensor left plantar response. There was hypoesthesia of his left leg affecting the dorsum of the foot, lateral and back sides of the calf and thigh, and the genital and perianal area. MRI of the thoracic and lumbosacral spinal cord was unremarkable. A follow-up MRI was performed after three weeks and revealed T2-weighted STIR hyperintensity of the Th10 vertebra with post-contrast enhancement, indicating spinal cord ischemia (Fig. 1). There was a small intramedullary hyperintense signal in T2-weighted images at the Th9 level on the left side corresponding to ischemic sequelae. At that time, the patient experienced partial recovery, his sensation was normal, and there was residual weakness in his left foot with normal micturition and erectile function.

Spinal stroke most commonly occurs in the vascular territory of the arteria radicularis magna, or the artery of Adamkiewicz, the largest radiculomedullary artery (1). It most commonly arises from the intercostal artery between segments Th9 to Th12, being the major contributor to the anterior spinal artery in the lower thoracic and upper lumbar region and providing arterial blood for the spinal cord approximately from the Th8 vertebra to the conus medullaris (1). The anterior part of the spinal cord is especially vulnerable to ischemia as the supplying vessels are end arteries and have no anastomoses (1). Occlusion of the anterior spinal artery can lead to the anterior spinal syndrome, which consists of motor weakness, loss of superficial sensation, and sphincter dysfunction. Infarction of the vertebra that may accompany spinal cord ischemia is explained by the shared vascularization of the vertebral body and the spinal cord (2). The common blood supply usually ceases after adolescence; however, degenerative changes in the vertebra may induce neovascularization that reestablishes the shared blood supply (3). In patients with spinal cord ischemic strokes, initial MRI may

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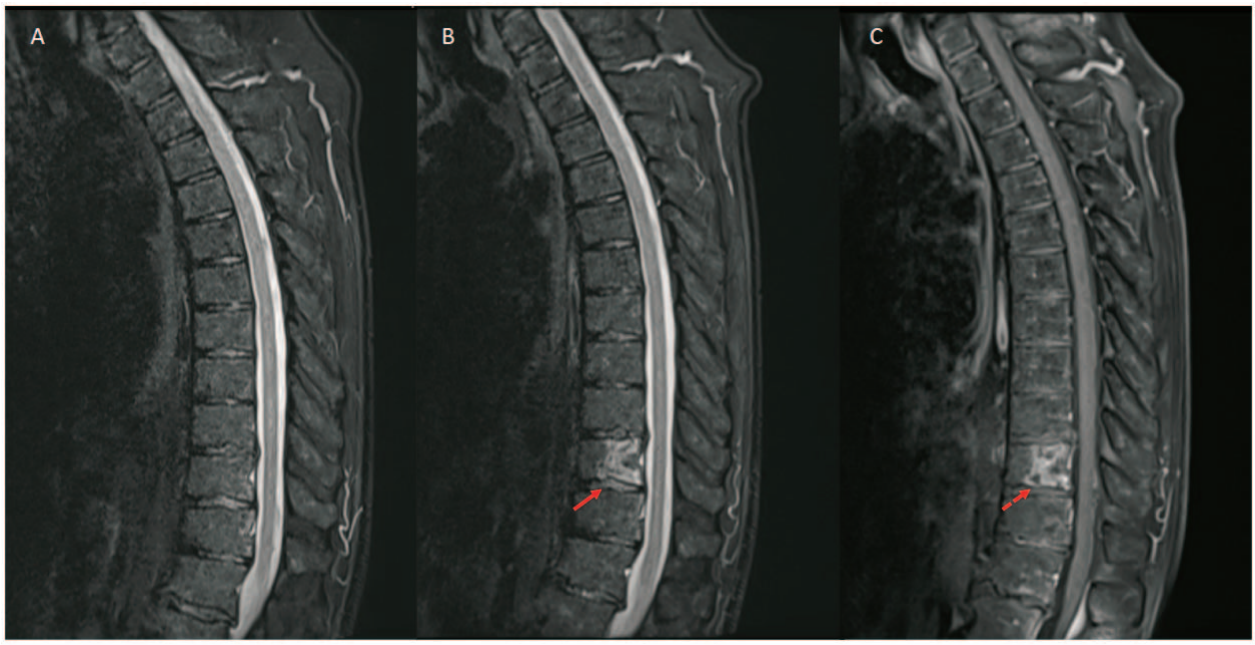

Fig. 1. *MRI of the spinal cord showing initial (a) and follow-up images after three weeks (b, c). A. Sagittal T2 weighted STIR image revealing normal signal of the thoracic vertebra. B. Sagittal T2 weighted STIR image demonstrating hyperintensity of the dorsal part of the Th10 vertebra (red arrow). C. Sagittal T1 weighted post-contrast image demonstrating gadolinium enhancement of the dorsal part of the Th10 vertebra (red dotted arrow). STIR-short-tau inversion recovery.*

not reveal pathological changes. Therefore, infarction of the vertebra, most appropriately demonstrated on STIR sequences, as in the current patient, can represent the only confirmatory sign of spinal cord infarction (4).

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