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Cutaneous ischemia and vertebral body infarction indicating spinal cord ischemia

Kožní ischemie a infarkt obratlového těla jako příznak míšní ischemie

Dear Editor,

Spinal cord ischemia is a rare condition, whose exact incidence is unknown. It accounts for about 6% of all acute myelopathies and about 1–2% of all vascular diseases of the central nervous system [1]. The clinical presentation and wide range of differential diagnoses further complicate the diagnostic process.

We present a patient with posterior spinal cord infarction with bone infarction and skin changes due to cutaneous ischemia facilitating the diagnosis.

An 82-year-old man with a history of arterial hypertension and type 2 diabetes mellitus was admitted to the hospital with acute weakness of the left lower limb. The night before admission, at around 3 AM, the patient experienced sudden pain in the left lower thoracic region of his back. The next morning, his wife noticed a dark red patch on the skin corresponding to the painful area. At 1 PM, the patient noticed some weakness of the left lower limb for the first time. By 3 PM that day, he was unable to walk properly due to instability.



Fig. 1. Skin changes in dermatomes T8-9 on the left side of the back mimicking infectious etiology.

Obr. 1. Kožní změny v dermatomech T8-9 na levé straně zad napodobující infekční etiologii.

The Editorial Board declares that the manuscript met the ICMJE "uniform requirements" for biomedical papers.

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Upon admission that evening around 6 PM, the patient presented with mild left lower limb paresis along with patellar hyporeflexia, distal areflexia, and a negative Babinski sign bilaterally. His upper extremities showed no obvious pathology and sphincter function was intact. A dark red oval area measuring 15 × 5 cm was found in the T8-9 dermatomes on the left side of his back with no apparent papules, vesicles, or pustules (Fig. 1). Meningeal signs were negative. Hypercholesterolemia was the most prominent laboratory abnormality. Blood tests, including inflammatory and autoimmune markers, were otherwise unremarka-

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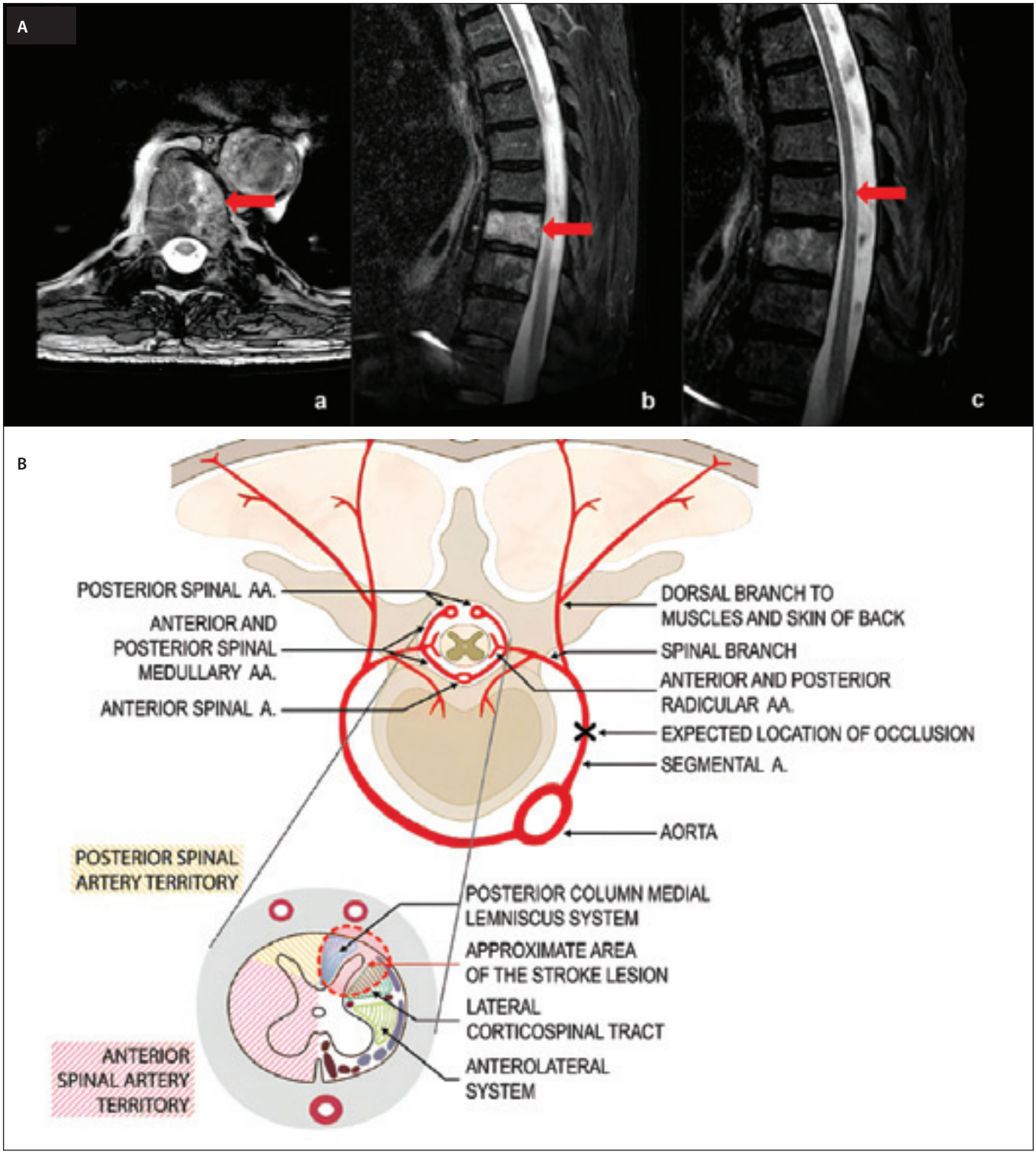


Fig. 2. (A) MRI – newly observed pathological signal (red arrow) in the left half of the vertebral body at the level of T9, indicating changes related to a bone infarct: (a) balanced turbo field-echo (B_TFE) sequence in the transverse plane, (b) STIR sequence in the sagittal plane. Discrete spinal cord inhomogeneities are seen on the STIR sequence at the T8 vertebra level (c). (B) Arterial blood supply to the spinal cord and thoracic vertebrae.

STIR – short time inversion recovery

Obr. 2. (A) MR – nově pozorovaný patologický signál (červená šipka) v levé polovině obratlového těla na úrovni T9, indikující změny související s kostním infarktem: (a) balanced turbo field-echo (B_TFE) v transverzální rovině, (b) STIR v sagitální rovině. Diskrétní nehomogenity míchy jsou vidět na sekvenci STIR na úrovni obratle T8 (c). (B) Tepenné zásobení míchy a hrudních obratlů.

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ble. Serum liver, kidney, and thyroid function tests and vitamin B12 and folate levels were normal.

Cerebrospinal fluid (CSF) examination revealed a slightly elevated total protein level (0.53 g/dL) and slightly elevated glucose level (4.30 mmol/L), with a normal white blood cell count and negative PCR tests for neuroviral panel and Borrelia antibodies.

The next day, the patient developed severe central left lower limb paresis with hyperreflexia and positive Babinski sign on the left leg. Sensory examination revealed impaired tactile discrimination up to the T10 dermatome on the left side with loss of vibration in the distal parts of the lower limbs, more expressed on the left side, and impaired proprioception of the left lower limb. Thermal and pain sensation were normal, and sphincter function was intact.

MRI of the thoracic and lumbar spine performed on the same day did not reveal any explanatory pathology. CT angiography showed aortic dilatation, diffuse atherosclerosis, and an ulcerated plate at the L1 vertebra. CT and MRI of the brain and MRI of the cervical spine were unremarkable. Spinal ischemia was considered the most likely etiology as the case met the diagnostic criteria [2]. The vast majority of published cases have an unremarkable initial MRI, with acute ischemic changes on T2-WI MRI scans becoming visible within hours to days, therefore the MRI was repeated within a week, showing an acute bone infarct involving the left half of the T9 vertebra and discrete post-ischemic cord changes at the T8 level (Fig. 2A) [3–6].

Acetylsalicylic acid and rosuvastatin were added to the patient's medication and he underwent intensive physical therapy. Two weeks later, at the time of discharge, there was a significant improvement in strength with only mild left lower extremity paresis and spontaneous resolution of the skin changes.

In similar cases, spinal cord MRI and CSF examination are the most important investigations to determine the etiology. The latter was performed first because an inflammatory (herpetic) etiology was considered due to the skin changes. Cerebral ischemia in the anterior cerebral artery territory was initially not considered due to flaccid monoparesis of the lower limb accompanied by back pain and skin changes. Negative findings in both CSF and MRI ruled out inflammatory and compressive etiologies. The clinical presentation was most likely related to incomplete unilateral posterior spinal artery infarction [7,8]. Although unilateral motor weakness is considered an atypical symptom of posterior spinal artery infarction, it may be more common than previously thought because clinical symptoms are not limited to posterior column function [7] (Fig. 2B). Ischemia on the left side of the spinal cord at the level of the T8 vertebra detected on follow-up MRI scans supported an ischemic etiology and explained the ipsilateral left central lower limb paresis and ipsilateral posterior column sensory loss with tactile discrimination and proprioception impairment [7]. Impairment of vibratory sensation in both lower limbs was most likely caused by diabetic polyneuropathy.

Previous studies have shown that vertebral bone infarction can support the diagnosis of spinal cord ischemia due to a shared vascular supply [7]. The anterior spinal artery supplies the ventral two-thirds of the spinal cord, while the posterior spinal arteries supply the dorsal third. In the thoracic region, the anterior spinal artery is discontinuous; vascular supply depends on segmental arteries from the aorta. These are the posterior intercostal arteries in the thoracic region. Infarcts of the anterior spinal artery are most common; infarcts of the posterior spinal artery are rare [7,8]. To our knowledge, cutaneous ischemia has not been reported as a supportive finding in spinal cord infarction. The posterior intercostal arteries supply the muscles of the back, vertebral column, spinal cord segments, and overlying skin in the corresponding area. If the posterior intercostal arteries are occluded, we can expect infarction of the spinal cord and the vertebral bones and possibly ischemia of the skin (Fig. 2B). We suspect atherosclerosis of the aorta as the cause of spinal cord ischemia in this case. Based on the degree of ischemia, segmental occlusion of the posterior intercostal artery at the T8-T9 level is likely [7]. The exact incidence of cutaneous ischemia is not well-documented in the medical literature. Cutaneous ischemia itself is a rare condition and can occur due to various underlying causes such as peripheral artery disease, embolism, or vasculitis [9].

In summary, our case report aimed to propose consideration of skin changes alongside bone infarction as possible supportive signs of spinal cord ischemia.

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Conflict of interest

The authors declare they have no potential conflicts of interest concerning drugs, products, or services used in the study.

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