Spontaneous Resolution of Spinal Epidural Hematoma

A 48-year-old female (H: 172 cm, W: 67 kg, BMI: 22.6 kg/m²) presented to our neurology clinic with severe pain in the thoracic spine region. This was associated with flaccid paralysis, sensory deficit in the T4-T6 dermatomes, as well as bladder and bowel dysfunction. Her past medical history was unremarkable, and she was not taking any medications. The observed deficits proceeded rapidly, despite the lack of obvious disease in the patient’s medical history. On admission, the patient’s neurological status was as follows: she was immobile with normal status of the neck and upper extremity as well as the cranial nerves; she had flaccid paralysis with symmetrical absent reflexes in lower-extremity and pathological reflexes indicating abnormalities in the T4-T6 sensory level, with accompanying bladder and bowel dysfunction.

Laboratory tests were normal. The results of coagulation studies, including prothrombin time, partial thromboplastin time, and bleeding time, were normal. Both macroscopic and microscopic urine analyses were normal. Magnetic resonance imaging revealed a left posterior epidural hematoma extending from the second to the third thoracic segment that deformed the spinal cord (Fig. 1). A consulting neurosurgeon suggested surgical decompression.

Following disappearance of the hematoma, the patient’s initial symptoms gradually improved within 24 hours of their appearance and during the following day. The patient could now move with assistance. Twelve days later, another MRI showed no abnormalities (Fig. 2). Therapy included mannitol, analgesics, and vitamins. The patient experienced a full spontaneous recovery after twelve days. She became mobile without weakness in lower-extremity, and without bladder or bowel dysfunction.

Spontaneous spinal epidural hematoma (SSEH) was first reported in 1869 by Jackson,¹ who described the stereotypic
symptoms in detail: sudden severe spinal pain with radiating pain corresponding to the bleeding spot, followed within a few days by a progressive paralysis in both lower extremities and impairment of bladder and bowel function due to spinal cord compression. Although SSEH is reported to occur in all age groups, it is most frequent after the fourth or fifth decade of life. Some causes of SSEH are related to minor trauma (lumbar puncture, spinal anesthesia); others are spontaneous, but more often SSEH occurs in patients on anticoagulant therapy or those with coagulation abnormalities, individuals with neoplasms at different localization, with vascular malformation, or in those with immune-mediated vasculitis and arterial hypertension. In 40% of cases the cause remains unidentified. Differential diagnosis suggests the possibility of acute transverse myelitis, as well as tumors of the spinal cord. Spinal MRI is essential for early detection of the location of SSEH as well as its compression status. The hematoma appears isointense on T1 sequences. In T2 sequences, acute hematomas appear hyperdense at the periphery with a hypodense center. Nonsurgical therapy may be justified when there is minimal neurological deficit, or if there is evidence of early spontaneous resolution of the hematoma. Alexiadou-Rudolf et al.1 reported that surgery within 12 hours produced favorable functional results in cases that needed early surgical decompression. However, Lawton et al.5 argued that is very difficult to gauge the optimum time for decompressive surgery. Wagner et al. and Rechtine et al.3-6 reported spontaneous resolution of SSEH, as we found in this study as well. Multidisciplinary collaboration, continuous monitoring of the patient, as well as hourly monitoring of neurological deficit, allowing a timely decision about surgery, helped us achieve a favorable outcome for our patient.

References