Case Report

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Non-traumatic Spinal Cord Compression Presenting with Concomitant Spinal and Neurogenic Shock: A Case Report

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Abstract

Background: Spinal shock is typically associated with high-energy trauma, while non-traumatic causes are exceptionally rare.

Case: We report the case of an 85-year-old male who presented with sudden-onset back pain and lower extremity weakness; hemodynamic collapse developed during emergency department follow-up. Thoracic computed tomography (CT) revealed acute spinal cord compression secondary to a T3 vertebral hemangioma and osteoporotic collapse. The patient developed neurogenic shock with bradycardia, hypotension, and respiratory failure in the emergency department. Immediate resuscitation with atropine, intravenous fluids, and dopamine infusion achieved stabilization. Thoracic computed tomography revealed a T3 vertebral hemangioma with osteoporotic collapse causing acute spinal canal narrowing. High-dose corticosteroid therapy was administered, followed by urgent neurosurgical intervention.

Conclusion: This case highlights the importance of recognizing non-traumatic spinal shock in elderly patients with acute paraparesis and hypotension, even without a history of trauma. Early diagnosis and multidisciplinary management are essential to prevent irreversible neurological deficits.

Keywords: Spinal shock, Paraparesis, Vertebral hemangioma, Osteoporosis, Non-traumatic compression.

Introduction

Spinal shock refers to a transient loss of all neurological activity below the level of spinal cord injury, characterized by areflexia, flaccid paralysis, and autonomic dysfunction (1). It is most often observed following high-energy trauma. However, non-traumatic spinal shock remains an extremely rare clinical entity and is frequently associated with diagnostic delays and poor outcomes.

Vertebral hemangiomas occur in approximately 10–12% of the population, yet fewer than 1% become symptomatic. Only a small subset cause acute spinal cord compression. When combined with osteoporosis, the risk of pathological fracture and sudden canal narrowing increases significantly (2).

We present an unusual case of acute paraparesis and progressive spinal shock secondary to spontaneous vertebral collapse due to coexisting osteoporosis and hemangioma in a patient without a history of trauma.

Case Presentation

An 85-year-old man with a medical history of hypertension, atrial fibrillation, early Alzheimer's disease, and osteoporosis presented to a primary care center with complaints of sudden-onset back pain, bilateral lower extremity numbness, and rapidly progressing weakness. He denied any trauma. To clarify the symptoms and rule out preliminary diagnoses, the patient was referred for brain CT, diffusion MRI, and spinal CT scans. On arrival, his vital signs were: blood pressure 130/80 mmHg, pulse 60 beats/min (atrial fibrillation), and oxygen saturation 92% on room air. Neurological examination revealed bilateral lower limb muscle strength of 1/5, positive Babinski reflexes, and marked tenderness over the thoracic spine. Abdominal examination showed right costovertebral angle tenderness. Laboratory results showed a white blood cell count of 10.94×10^9/L, hemoglobin 13.5 g/dL, plate-

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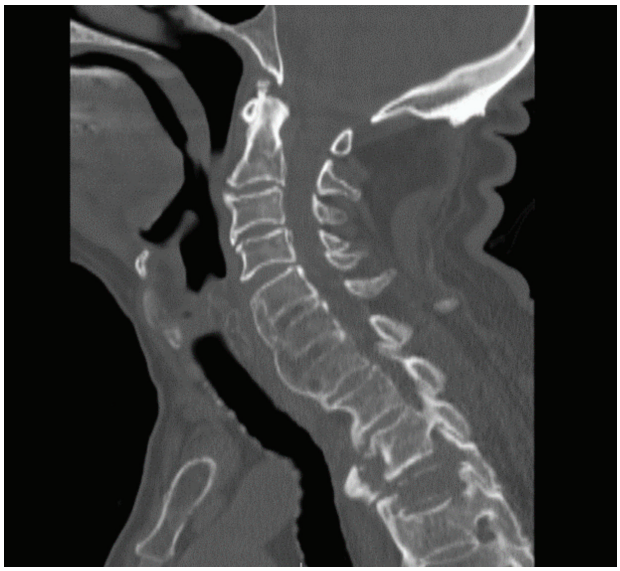

Figure 1. Sagittal contrast-enhanced thoracic computed tomography (CT) scan demonstrates diffuse hypodensity and collapse of the T3 vertebra associated with acute spinal cord compression. The arrow indicates the level of vertebral collapse.

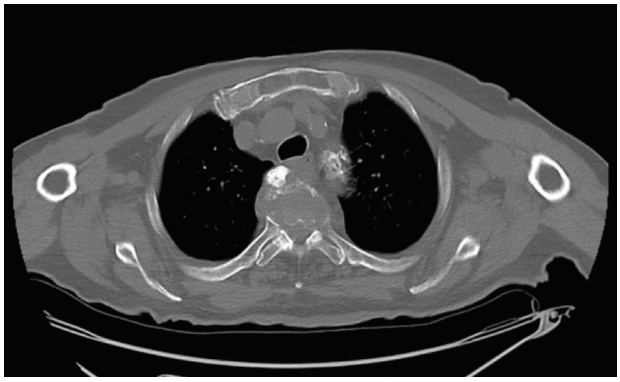

Figure 2. Axial contrast-enhanced thoracic CT image at the T3 level reveals significant spinal canal narrowing due to vertebral body collapse with associated hemangioma. The arrow indicates the direction of compression; the star indicates the vertebral hemangioma.

lets 229×10^9/L, with neutrophilia (86.4%) and relative lymphopenia (9.7%). Coagulation parameters were within near-normal limits (PT 15.8 s, INR 1.16, aPTT 23 s). Urinalysis showed specific gravity 1.009, mild proteinuria (1+), and microscopic hematuria (3+ erythrocytes per high power field). Initial differential diagnoses included Guillain–Barré syndrome, transverse myelitis, and non-traumatic spinal cord compression. Shortly after imaging, the patient developed acute confusion, severe bradycardia (30 beats/min), profound hypotension (50/20 mmHg), and respiratory failure, consistent with neurogenic shock. Immediate resuscitation was initiated with 100% oxygen (10 L/min), bag-valve-mask ventilation, intravenous atropine (1 mg), isotonic fluid resuscitation (1000 mL IV), and dopamine infusion (10 mcg/kg/min). Hemodynamic stability was restored within minutes. Neuroimaging revealed no acute intracranial pathology. Thoracic CT demonstrated a hypodense appearance and collapse of the T3 vertebral body, with findings compatible with hemangioma and osteoporosis causing acute spinal cord compression (Figure 1 and 2). Following neurosurgical consultation, the patient received high-dose corticosteroid therapy with intravenous methylprednisolone (30 mg/kg bolus over 15 minutes, followed by 5.4 mg/kg/h for 23 hours) and was urgently taken for decompressive surgery. MRI could not be performed prior to surgery due to the patient's rapidly deteriorating neurological and hemodynamic status requiring emergency intervention. Following emergency decompressive surgery, the patient was admitted to the intensive care unit due to persistent hemodynamic instability and requirement for mechanical ventilatory support. Despite initial stabilization, the patient developed nosocomial pneumonia and sepsis during the intensive care follow-up. Unfortunately, the patient succumbed to multi-organ failure on postoperative day 34. No significant neurological improvement was observed prior to demise (motor strength remained 1/5 in lower extremities). Written informed consent was obtained from the patient's next of kin for publication of this case and accompanying images.

Discussion

Non-traumatic spinal shock is a rare condition with few cases documented in literature. Vertebral hemangiomas are benign vascular lesions often found incidentally, but in rare cases, they may become aggressive and lead to pathological fracture, epidural extension, or acute cord compression. In this patient, the coexistence of hemangioma and osteoporosis likely triggered sudden vertebral collapse and acute canal narrowing at T3 (2). Neurogenic shock, which often accompanies high spinal cord injury, results from disruption of sympathetic outflow from the T1–L2 spinal segments. This loss of sympathetic tone leads to vasodilation, hypotension, and bradycardia. Spinal shock, on the other hand, represents a complete loss of sensory, motor, and autonomic function below the injury level. Differentiating between spinal and neurogenic shock is critical, as both can coexist and require specific management (3). In the elderly, non-traumatic spinal shock may mimic cerebrovascular events or demyelinating diseases, leading to delayed diagnosis. Early CT/MRI imaging is crucial for identifying compressive lesions (4). Literature emphasizes that prompt administration of corticosteroids, aggressive hemodynamic support, and timely neurosurgical decompression can prevent irreversible neurological damage and improve outcomes (5). Although high-dose methylprednisolone (30 mg/kg bolus followed by 5.4 mg/kg/h for 23 hours) is primarily recommended

for traumatic spinal cord injury based on the National Acute Spinal Cord Injury Study (NASCIS) protocols, its use in non-traumatic acute spinal cord compression remains debated. In the present case, high-dose methylprednisolone was administered due to acute neurological deterioration and suspected spinal cord compression in the emergency setting, aiming to reduce vasogenic edema and inflammatory cascade prior to definitive surgical decompression. Similar approaches have been reported in selected non-traumatic compression cases where urgent anti-edema and neuroprotective therapy was considered beneficial as a bridge to emergency surgery (1,2).

Reported cases of non-traumatic spinal shock secondary to vertebral hemangioma are exceedingly rare (6). Shankar et al. described a similar presentation in a patient without trauma, where sudden collapse of the vertebral body caused acute cord compression and neurogenic shock. In our case, the combination of osteoporosis and hemangioma likely precipitated this catastrophic event. This highlights the need to consider non-traumatic etiologies when elderly patients present with unexplained paraparesis and shock.

Limitations

This is a single-patient report; broader studies and pooled analyses are needed to define the epidemiology, diagnostic approach, and optimal treatment protocols for non-traumatic spinal shock in elderly populations. Additionally, the absence of preoperative MRI due to the emergency nature of the case represents a limitation in fully characterizing the extent of spinal cord compression.

Conclusion

Non-traumatic spinal shock should be considered in elderly patients presenting with acute paraparesis, bradycardia, and hypotension, even in the absence of trauma or malignancy. Rapid diagnosis, early hemodynamic stabilization, and urgent surgical consultation are vital to improving survival and neurological recovery. Delayed diagnosis may result in irreversible neurological damage and increased mortality. This underscores the importance of early recognition in emergency settings.

Ethical Declarations

Informed Consent: Written informed consent was obtained from the patient's next of kin for publication of this case and accompanying images.

Conflict of Interest: The authors declare no conflicts of interest.

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