



Trauma-Induced Neurogenic Shock in a Middle-Aged Man without Spinal Displacement

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Neurogenic shock is an autonomic dysfunction causing abnormal blood distribution without blood loss. Immediate diagnosis is critical, especially in cases lacking apparent spinal displacement. A 59-year-old male involved in a motorcycle accident presented with out-of-hospital cardiac arrest and on initial examination revealed bradycardia, hypotension, and a fracture at the C4 spinous process. Despite the absence of apparent spinal displacement, the patient's severe hypotension and bradycardia, coupled with findings from a magnetic resonance imaging (MRI) that highlighted spinal cord edema at the C3 – C4 level, confirmed suspicions of neurogenic shock. Though hemodynamic stability was temporarily achieved via dopamine infusion, the patient succumbed 3 days after admission. The elevated prehospital mortality rate associated with spinal cord injuries highlights the critical nature of early diagnosis and intervention. Such injuries can result in symptoms like bradycardia and respiratory complications. For those presenting with trauma-related symptoms, thorough neurological evaluations, complemented by imaging such as computed tomography (CT) and MRI, are essential. Neurogenic shock should be suspected in cases of severe hypotension and bradycardia post-spinal cord injury. Early interventions, including fluid administration and vasopressor support, are pivotal, while the role of steroids remains controversial. Emergency physicians must maintain a high degree of suspicion for neurogenic shock, even in the absence of evident spinal displacement, and prioritize early diagnostic measures to optimize outcomes.

Key words: spinal cord injury, neurological shock, autonomic dysfunction, hypotension, bradycardia

Introduction

Traffic accidents account for half of spinal cord injuries, which may be accompanied by neurogenic shock caused by direct trauma.¹ Neurogenic shock, an autonomic dysfunction

causing an abnormal blood distribution (i.e., “distributive shock”) instead of blood loss, is characterized by decreased systemic vascular resistance, profound hypotension, bradycardia, and unopposed vagal tone.² Compromised respiration is another major contributor to mortality and morbidity, and while high-level

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spinal injury (e.g., C2 – C4) may be life-threatening through a complete disruption of spontaneous respiratory effort that requires immediate mechanical ventilator support, injury at a lower cervical level may cause weakness or paralysis of intercostal and abdominal muscles.³ Despite the seriousness of this condition, early diagnosis remains challenging to emergency physicians, particularly in patients without apparent spinal displacement on initial presentation. Immediate interruption of spinal cord vascular blood supply after spinal trauma may contribute to neurogenic shock.⁴ Here, we presented a case of neurogenic shock without apparent spinal displacement and reviewed the characteristics, treatment strategies, and prognosis of spinal cord injury (SCI).

Case Report

A 59-year-old man, a motorcycle passenger at the time of the accident, was sent to our emergency department by ambulance and diagnosed with out-of-hospital cardiac arrest. Before he lost consciousness at the accident site, he complained of neck and chest tenderness along with dyspnea and dizziness to the emergency medical service personnel. On arriving at the emergency department, the patient was unresponsive and pulseless. Return of spontaneous circulation (ROSC) was noted after 5 minutes of cardiopulmonary cerebral resuscitation. Electrocardiogram demonstrated sinus tachycardia. Focused assessment with sonography in trauma showed neither ascites nor pericardial effusion. Ecchymosis was observed on the right thigh, with abrasion wounds on both feet. Physical examination of the chest revealed normal breath sounds. Chest radiography showed no pneumothorax, with a properly placed endotracheal tube. Initial laboratory data demonstrated unremarkable hematological and biochemical findings except mild metabolic acidosis. About 40 minutes after the initial ROSC, bradycardia and hypotension



Fig. 1 A computed tomography (CT) scan of the cervical spine in a sagittal view reveals a C4 spinous process fracture without displacement of the cervical spine (white arrow).

became evident, followed by pulseless electrical activities for which chest compression was resumed. In addition to the administration of vasopressin and fluid resuscitation, emergent blood transfusion was started with leukocyte-poor red blood cells, accounting for the possibility of hemorrhagic shock. Whole body computed tomography revealed a fracture at the spinous process of the C4 vertebra without cervical spine displacement (Fig. 1), fracture of the right third to sixth ribs, left third to seventh ribs, and comminuted intertrochanteric fracture of the right femur without abnormal peritoneal fluid accumulation. Furthermore, no lung contusion, hemothorax, or pneumothorax was observed. Additionally, no pericardial effusion or aortic injury was found. Moreover, electrocardiography showed no T wave abnormalities.

Due to persistent bradycardia with hypotension refractory to vasopressin infusion, neurogenic shock was highly suspected. Magnetic resonance imaging (MRI) of the cervical spine revealed spinal cord edema

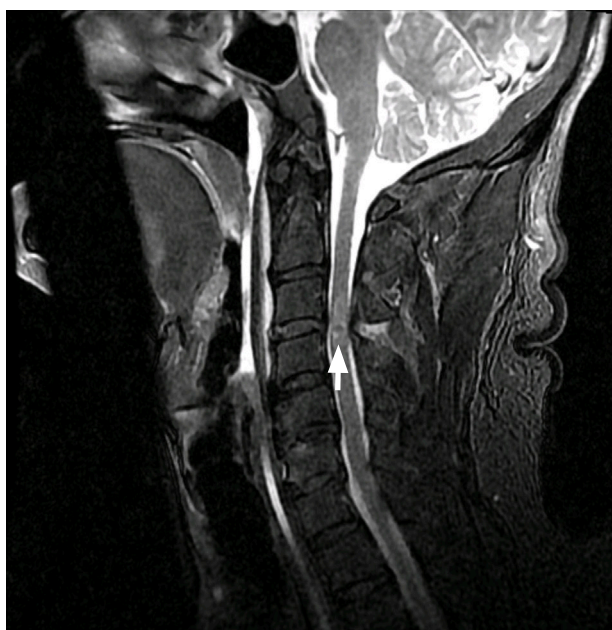


Fig. 2 A T2 sagittal view magnetic resonance imaging (MRI) scan of the cervical spine reveals spinal cord edema at the C3 – C4 level (white arrow).

at the C3 – C4 level with mild degenerative neural foramen stenosis over C6 – C7 (Fig. 2). Hemodynamic stability was maintained with dopamine infusion to treat neurogenic shock after consultation with our neurosurgeon who suggested no surgery for C3 – C4 spinal injury. After being admitted to the neurosurgery intensive care unit (ICU), persistent hypotension refractory to inotropic agents was noted for 48 hours, followed by acute kidney failure with metabolic acidosis for which continuous veno-venous hemofiltration was suggested but refused by his family. The patient finally succumbed to multiple organ failure with metabolic acidosis 3 days after ICU admission.

Discussion

The prehospital mortality rate for SCI ranges from 48.3% to 79%, with a higher mortality rate observed in those with cervical spine injuries.¹ Additionally, the probability of developing an SCI among patients with traumatic cervical spine fractures ranges from 3.62% to 11.6% in the general population.^{5,6} Bradycardia is the most common symptom after SCI.⁷ The

patient may later develop organ dysfunction or failure associated with trauma to other organs or related to the treatment process (e.g., use of steroids).⁸ Approximately 20% – 57% of patients diagnosed with SCI may have significant injuries to the brain or chest.¹ Moreover, up to 36% – 83% of patients with SCI may suffer from respiratory complications, with the incidence and severity being increased in those with a high-level injury. At the acute stage, those with C3 – C5 injury may experience phrenic paralysis requiring mechanical ventilation. Endotracheal intubation is always needed for those with a complete cord transection above C5. At a later stage, pneumonia and atelectasis are also common complications because of poor cough function and ventilatory failure.³

Immediate manifestations of SCI may include the loss of motor, sensory, and autonomic functions. Primary SCI is most likely caused by direct spinal cord compression.⁸ Contrarily, secondary injury, developing over hours to a lifetime after the initial trauma through several pathophysiological mechanisms, can be categorized into three phases: acute, sub-acute, and chronic. The acute phase begins with hemorrhage, ischemia, inflammation, cytotoxic edema, and pro-inflammatory cytokine production, while the sub-acute phase is characterized by further cell necrosis caused by edema or hemorrhage. In contrast, the chronic phase may start 6 months after the initial trauma and develop throughout the lifetime of the patient as a long-term neurological sequelae.¹

A thorough neurological examination should be performed and documented on the American Spinal Injury Association (ASIA) chart. Patients presenting with symptoms pertinent to spinal injuries, including limb weakness, paralysis, or consciousness change, should require a computed tomography (CT) scan for the identification of the injury location. Conversely, although plain radiog-

raphy may offer information about possible thoracolumbar spine injuries, its use may be limited in those with suspected cervical spine injuries as it requires full patient cooperation. Patients with severe symptoms should undergo MRIs, especially those with strong evidence suggestive of spinal injury.²

Neurogenic shock should be suspected in patients presenting with severe hypotension and bradycardia within 24 – 48 hours after SCI. Besides the standard administration of fluid, vasopressin, and oxygen, atropine should be given to those with bradycardia (i.e., < 50/min). The symptoms may persist for 3 – 5 days after injury and require 2 – 6 weeks to resolve.⁷ Although steroids have previously been suggested to be given as a 24-hour infusion of high-dose methylprednisolone in patients with SCI within 8 hours after trauma according to the AOSpine practice treatment guidelines in 2017,⁹ a recent meta-analysis showed no steroid-related neurological improvements in patients with SCI. That study also highlighted the downsides of steroid therapy, including increased risks of infection, gastrointestinal bleeding, and hyperglycemia.¹⁰ For patients showing evident spinal compression, early decompression may be beneficial to the final outcomes. Similarly, the Surgical Timing in Acute Spinal Cord Injury Study demonstrated that decompression within 24 hours following injuries could confer a significantly improved neurologic outcome of at least a Grade 2 ASIA Improvement Score in adults with a cervical SCI.²

Concerning our patient, he presented a diagnostic dilemma owing to impaired consciousness. The neurological examination was difficult to conduct during the initial presentation. Consequently, protection of the cervical spine is advocated during prehospital care. Given the severe traumatic condition, a whole-body CT scan is indicated. Although imaging revealed only a nondisplaced spinous process fracture, the patient's profound hypotension and bradycardia necessitated the consideration

of neurological shock, especially when ruling out internal bleeding.

Author Contributions

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Conflicts of Interest

The authors declare no conflict of interest.

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