Chapter

Spinal Shock: Clinical Pearls

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Abstract

Spinal shock is a clinical manifestation following injury to the spinal cord resulting from multiple mechanisms. It is a complex phenomenon with flaccid paralysis, absent anal wink, and bulbocavernosus reflex. Management strategy for such patients includes rapid evaluation and treatment strategies to minimize the impact of secondary spinal cord injury. The advanced trauma life support (ATLS) guidelines provide the basis for rapid assessment and stabilization of A (Airway), B (Breathing), and C (Circulation) before dealing with the neurological deficits under the primary survey. The emergence of better radiological investigations has been pivotal in categorizing spinal syndromes and reaching a precise diagnosis. Early initiation of treatment measures results in better neurological and functional recovery with minimal residual deficits. The role of steroids in spinal shock has been a highly debated topic, and the timing of surgery is variable, intending to eliminate the secondary injury. Clinical differentiation between neurogenic and hypovolemic shock is vital, enhancing the quality of care with realistic outcome expectations.

Keywords: spinal shock, sympathetic, spinal injury, spinal reflex, spinal cord

1. Introduction

The term "spinal shock" was introduced to differentiate arterial hypotension, which is due to hemorrhage. Spinal shock appears following spinal cord injury leading to loss of sympathetic tone which is described in literature for more than 150 years [1–3]. This shock manifests as transient loss or impairment of all or part of spinal reflex activity below the level of the spinal injury that may be due to physiologic or anatomic transection of the spinal cord [4]. In this chapter, we review the basic concepts in the development of spinal shock, clinical presentations, management strategies, follow-up, and outcomes in patients with spinal shock.

1.1 Overview

In a majority of the cases, spinal shock result secondary to trauma (motor vehicle accidents, falls, sporting accidents, and self-harm) [5] causing either transection, hemorrhage, or ischemic injury to the spinal cord [6], other less-common causes

include mechanical cord compression, hypotension, and hypoxia [7]. In spinal shock, descending facilitation of upper motor neurons in spinal cord injury patients is impaired, leading to difficulties differentiating upper motor neuron lesions from lower motor neuron lesions [8]. The somatic component of spinal shock and autonomic reflexes are variably affected depending on the level of injury and phase of recovery [8]. Clinically, the spinal shock is characterized by reversible and temporary loss of all neurological function (that includes motor and sensory dysfunction, variably depressed reflexes, detrusor and rectal tone) below a particular spinal level [6, 9–12]. During the recovery phase, acute loss of functions is followed by the development of spasticity with increased muscle tone, exaggerated deep tendon reflexes, and muscle spasms [13]. Usually, reflex detrusor contractility returns if the distal portions of the spinal cord are not damaged but rather isolated from higher centers. Initially, such reflex activity is not maintained correctly and the return of reflex bladder activity typically occurs with the recovery of deep tendon reflexes in the lower extremities [8].

2. Clinical evaluation

Resuscitation, hemodynamic stabilization, and clinical assessment of a patient with spinal shock are a simultaneous and ongoing process [4, 8]. Clinical details include a detailed history of the mode and mechanism of injury (hit by another vehicle, fall, rollover crash, ejection outside the car, or seat belt was used or not), any history of alcohol intoxication, history of any comorbid conditions, and a detailed spine and physical examination of all the systems to exclude any associated injuries or dysfunctions [14]. Neurological examination includes assessment of the level of consciousness, motor and sensory functions, and assessment of deep tendon and superficial reflexes [15–17]. This will help determine the lesion's level and the extent of neurological impairment. Additionally, attention should be paid to determine the associated autonomic dysfunction (including bowel and bladder disturbance), autonomic dysreflexia, and the presence and extent of cardiovascular dysfunctions [18]. Involvement of the respiratory system, particularly intercostal muscles and diaphragm, can result in respiratory compromise. Early recognition and appropriate intervention (elective ventilation, early tracheostomy), including chest physiology, will help recover respiratory functions.

2.1 Spinal shock versus neurogenic shock

Although "spinal shock" and "neurogenic shock" are used interchangeably to optimize the outcome, there is a need to identify these two entities separately. Neurogenic shock is characterized by the hemodynamic changes resulting from spinal cord injury (above T6) and a loss of autonomic tone resulting in hypotension and bradycardia [4]. In a broader perspective, neurogenic shock is a distributive shock characterized by hypotension, bradycardia, and peripheral vasodilatation. It can manifest following a significant central nervous system damage (head injury, cervical spinal cord, or high thoracic cord injuries) [4]. **Table 1** shows a comparative description of these two types of shock, frequently encountered in trauma patients [19–22]. In clinical practice, early identification of spinal shock relieves the patient's anxiety and better prognostication of the sequela following spine injury.

	Spinal shock	**Neurogenic shock**
Location of injury	Due to spinal cord injury at any level	Due to head injury and spinal cord injury at cervical and high thoracic spine (above T6) level
Onset	Sudden to days	Sudden
Mechanism	Temporary unresponsiveness of peripheral neurons to brain stimuli leads to loss of reflex activity below the level of lesion	Autonomic pathways disruption leads to loss of sympathetic tone and vasodilation
Affects	Just spinal cord is affected	Entire nervous system is affected
Clinical presentation	Clinically present as flaccidity followed by spasticity at a later stage	Clinically present as instability of blood pressure, heart rate, and temperature regulation
Systemic hypotension	Possible	Always
Treatment	No specific treatment for spinal shock	Phenylephrine/norepinephrine to regain the sympathetic tone and atropine/glycopyrrolate for bradycardia
Resolution	Usually a temporary phenomenon, recovering within 24 to 48 hours, but can persist for 4 weeks to months	Usually short

Table 1.
Comparison between spinal shock and neurogenic shock.

2.2 Management

In the majority, spinal shock is associated with traumatic spinal cord injuries and requires a comprehensive interprofessional team approach (consisting of emergency teams, neurosurgeons, neuro-rehabilitation experts, and social workers). Imaging evaluation includes magnetic resonance imaging (MRI) and a detailed spinal computed tomogram (CT) with bony details. Before performing the detailed imaging, initial evaluation, and management follow the protocol to manage any patient who presents to the emergency room and manage "Airway, Breathing and Circulation" [23]. These patients may need intubation, mechanical ventilation, central venous access, invasive monitoring, and vasopressors to manage hemodynamic instability and neurogenic shock. They may require management of the source of hemorrhage, pneumothorax, myocardial injury, pericardial tamponade, or any other source of hypotension [23, 24]. Patients with a high cervical injury who present with spinal shock shall need special attention as these may frequently require cardiovascular interventions, including pacemakers for symptomatic bradycardia [25]. Elective ventilation or early tracheostomy to prevent or manage respiratory complications [26]. These patients shall need nutritional support, prophylaxis to prevent gastric ulcers, deep vein thrombosis, a long-term indwelling urinary catheter for bladder dysfunction, toilet training for bowel dysfunction, and care from preventing pressure ulcers [6, 7].

2.3 Outcome

Although there is improved survival in the patients, the severity of neurological deficits determines the overall outcome of these patients [6–8]. Overall, spinal cord

injury and shock are associated with poorer functional and overall outcomes requiring long-term rehabilitation care [15, 27].

2.4 Respiratory

The level of spinal cord injury usually determines the degree of respiratory support required in these patients. Complete injury above the level of C3 results in apneic respiratory arrest and death in the absence of prompt ventilatory support. Less-severe ventilatory impairment is associated with injuries below the C5 vertebra with various levels of respiratory failure for injuries between C3 and C5.

2.5 Cardiovascular

Involvement of cardiac accelerator fibers (T1–T4) is the cause of bradycardia and decreased myocardial contractility in these patients, often resulting in systemic hypotension and subsequent reduced spinal cord perfusion.

2.6 Deep venous thrombosis

Venous thromboembolism is one of the major causes of death, in addition to infectious complications after spinal cord injury. Antithrombotic prophylaxis by means of low molecular weight heparin or low-dose unfractionated heparin along with non-pharmacologic devices is helpful.

2.7 Gastrointestinal

There is increased risk of stress ulcers and upper gastrointestinal bleeds in these patients especially in those who are on mechanical ventilation and receiving high-dose steroids.

2.8 Neuropsychiatric

Depression, anxiety disorders, substance-related disorders, and suicidal tendencies are neuropsychiatric complications in these patients. Psychological support and counseling are essential.

Ditunno et al. [6]. described the loss of reflexes and recovery patterns in spinal shock patients in much detail depending upon duration following injury. He recognized four phases (Phase I–IV), phase I (Areflexia/hyporeflexia) postinjury day 0–1, phase II (Initial reflexes return) postinjury days 1–3, and phase III (Initial hyperreflexia) postinjury between days 4 and 1 month and phase IV (Final hyperreflexia) occurs between 1 and 12-months after injury.

3. Conclusions

In patients with spinal injuries, spinal shock is associated with poor outcomes. In a case of a history of trauma, careful attention should be paid to recognizing the spinal injuries and reasonable efforts need to be made to avoid the aggravation of injuries. Management needs to focus on airway and respiratory dysfunction, hypotension, and cardiovascular abnormalities. Imaging modalities, including CT and MRI, can help

identify the extent and type of injuries and expedite the decision to facilitate spinal cord decompression and stabilization as required.

Conflict of interest

The authors have no conflict of interest to declare.

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