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# Spine injury: How to recognize and stabilize patient in golden hours periods

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## ABSTRACT

An acute spinal cord injury (SCI) early management is one of the most challenging tasks in trauma cases. The outcome of its cases depends upon the accuracy, adequacy, and speed of first aid management, diagnosis, and treatment within the "Golden Hours Periods." Rapid and safe transport of the spinal injury patient allows for early medical stabilization designed to preserve and potentially improve the neurological outcome. The complications that arise from spinal instability or neurological must be prevented immediately and involves all members of the multidisciplinary team. Specialized assessment of people with new spinal cord injuries must take place immediately after an injury. The diagnosis of SCI should include the level and severity of the damage of the spinal cord, type of fracture or/and dislocation of the injured vertebrae, and the stability of the spinal column. Imaging begins from plain radiograph and should include a multi-slice CT protocol of the entire spine to delineate the known injury and to exclude non-contiguous damages. The outcome SCI treatment depends on the number of axons that survived – the higher the number of surviving axons, the restoration of neurological functions might be higher. The management of SCI begins before the patient admitted to the hospital. The principles of treatments include early reduction and fixation, combined extramedullary and intramedullary decompression, cell transplantation, early rehabilitation treatment, and complication prevention. The surgical treatment sometimes needed to evaluate, stabilize the spine, decompress the area that injured, and managed any other injuries that may have been associated with the accident. Once a patient was stabilized, care, and treatment focus on supportive care and rehabilitation.

**Keywords:** spine injury, management, treatment, golden periods.
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## INTRODUCTION

Spinal cord injury (SCI) represents one of the most devastating survivable injuries a patient can suffer.[1] The neurological deficit severity depends on the duration of spinal cord injury (SCI) and its degree of compression. The spinal cord compression with paraplegia or quadriplegia as sequelae has enormous socioeconomic and emotional consequences.[2]

The global incidence of SCI was estimated from 40 to 80 new cases per million populations per year. It means that every year, the spinal cord injury case happens between 250,000 and 500,000.[3] Regardless of the cause of spinal cord injury, early decompression is the principal goal for managing this case. The prognosis was noted to be better when decompression is done as early as possible in acute spinal injury. The controversies regarding the importance and timing of decompression are emphasized in recent studies.[4,5]

## PATHOPHYSIOLOGY

Pathophysiological progress in SCI can be described as a biphasic condition. This condition consists of the primary and secondary phases of spinal cord injury (Table 1). The primary phase includes the initial mechanical damage that causes spinal failure as a buffer of the body (fracture and/or dislocation), which directly exerts a destructive force on the spinal cord, damaging axons, blood vessels, and cell membranes. Meanwhile, the secondary injury phase includes vascular dysfunction, inflammation, oedema, ischemia, electrolyte shift, free radical production, excitotoxicity, and delayed apoptotic cell death. Neurological deficits are present immediately after the initial injury; the secondary injury phase will cause a prolonged period of tissue damage (Table 1).[6,7]

The most common form of acute SCI is compressive-contusive type injury, which causes the components of the vertebral column, which consist of intervertebral discs and ligaments to shift. The disruptive force on the spinal cord causes direct traumatic injury and continuous compression. The first pathological change detected after an injury is swelling of the spinal cord, which is usually associated with bleeding in central gray matter, causing cells to experience necrotic death due to direct cell membrane disruption or ischemia due to vascular disorders - Ion dysregulation and excitotoxicity. Ionic homeostasis disorders that occur immediately after SCI and also excitotoxicity have a significant contribution to the spread of cellular injury after SCI. The final effect of the disruption of ionic

**Table 1** **Spinal cord injury phases**[6]

| Phase and key events | ≤2 Hours | ≤48 Hours | ≤14 Days | ≤6 Months | ≥6 Months |
|---|---|---|---|---|---|
| Injury phase | Primary immediate | Early acute | Secondary subacute | Intermediate | Chronic/late |
| Key process and events | Primary mechanical injury, traumatic severing of axon, grey matter haemorrhage, hemorrhagic necrosis, microglial activation, released factor (IL-1β, TNFα, IL-6, and others | Vasogenic and cytotoxic oedema, reactive oxygen species production and lipid peroxidation, glutamate-mediated excitotoxicity, continued haemorrhage, continued haemorrhage and necrosis, neutrophil invasion, peak blood-brain barrier permeability, early demyelination (oligodendrocyte death), neuronal death, axonal swelling, systemic events (systemic shock, spinal shock, hypotension, hypoxia) | Macrophage infiltration, initiation of astroglial scar (reactive astrocytosis), blood-brain barrier repair & resolution of oedema | Continued formation of glial scar cyst formation lesion stabilization | Pronged Wallerian degeneration, persistence of spared, demyelinated axons, potential structural and functional plasticity of spared spinal cord tissue |
| Therapeutic aim | Neuroprotection | Neuroprotection, immune modulation, cell-based remyelination approaches, glial scar degeneration | | Glial scar degradation | Rehabilitation, neuroprostheses |

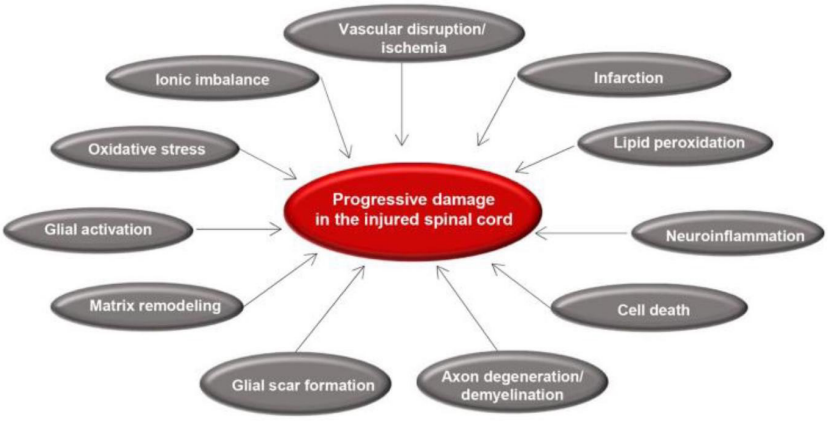


**Figure 1** The primary condition that happens after Traumatic spinal cord injury includes acute changes result in cell death, axonal damage, matrix remodelling, and formation of a glial scar[7]

homeostasis becomes the center caused by necrotic cell death and apoptosis after injury (Figure 1).[3,6,8]

## DIAGNOSIS

Assessment of patients with suspected SCI should include the level and type of fracture or/and dislocation of the injured vertebra, spinal stability, the extent and severity of spinal cord injury, should be included in the diagnosis of SCI (Figure 2). The ASIA scale is one classification to assess the severity of SCI. ASIA grade A is assessed for injuries in the absence of voluntary anal sphincter contractions and perianal sensations. ASIA Grade B shows that some sensations are maintained, but the motor score is zero below the injury level. ASIA Grade C shows some motor function, but motor scores below the injury level are less than 50% of normal. Grade D ASIA means that motor scores below the injury level increase to 50% or higher than normal.[8]

## IMAGING

Imaging and examination must be carried out after initial stabilization. Spinal imaging is an essential part of the initial management of acute SCI; Plain X-rays or computed tomography form the basis of a standard trauma protocol and can identify most fractures and ligament injuries. Computed tomography is the first choice of imaging for patients with SCI and polytrauma. CT-scan provides fast imaging with increased visualization of bone fractures. Three-dimensional axial and CT scans can reveal the shape of the spinal canal and facet joints of the vertebrae. For multiple injuries, a CT scan must be performed.[8,9]

These imaging modalities cannot visualize the spinal cord or surrounding soft tissue to the same level as MRI. Meanwhile, the potential risks and disadvantages of MRI, including patients, must be stretched for up to 30 minutes and maybe risky for trauma patients with respiratory difficulties or unstable hemodynamic.[10]

## MANAGEMENT

### Acute management

The number of neurological dysfunction after a spinal cord injury caused by improper management is around 25%. First aid management for SCI patients must be taken more seriously. Biomechanical studies recommend the use of a rigid cervical collar with supporting blocks on hardboard with a strap. However, tissue necrosis can occur from pressure

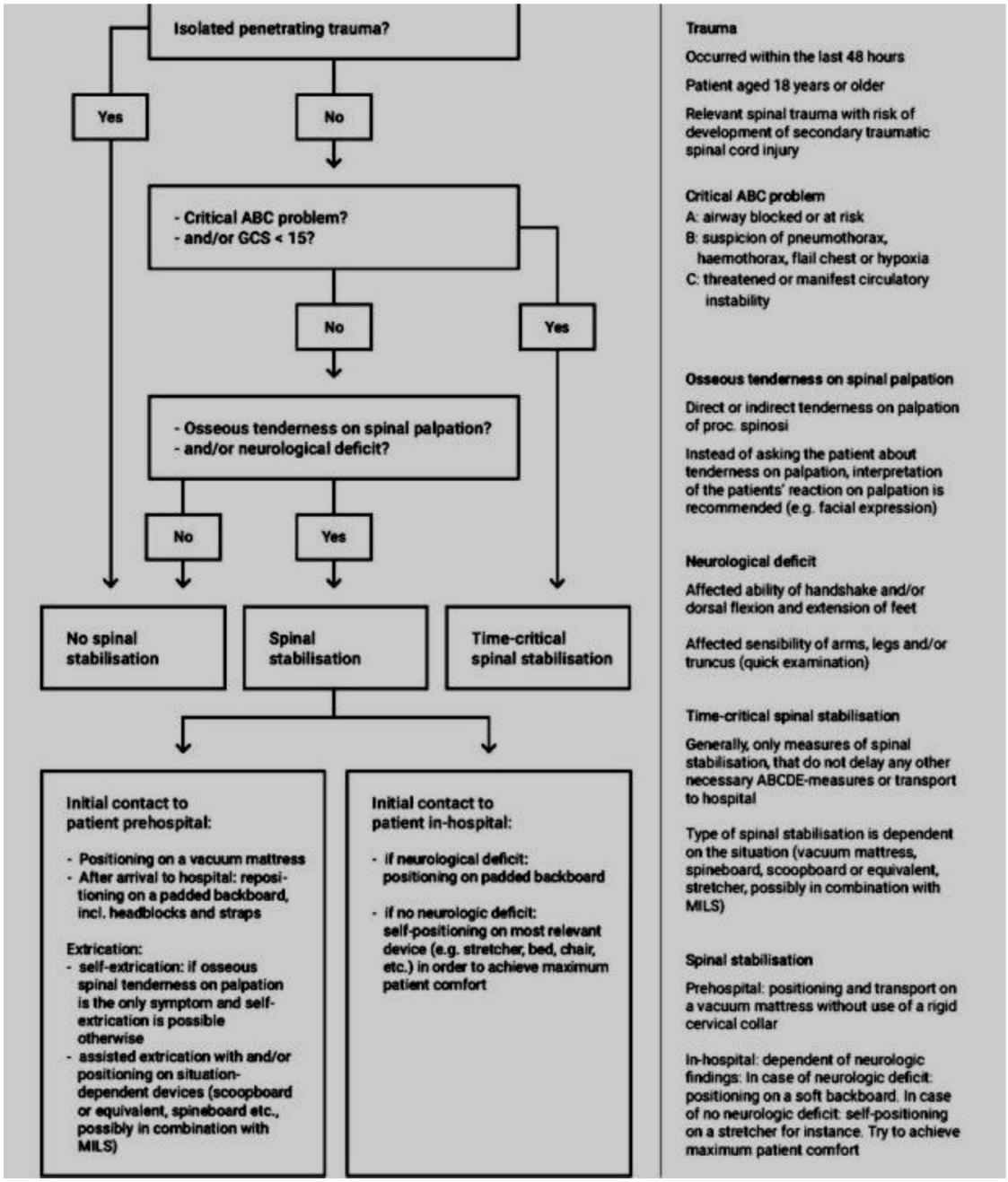


**Figure 2** Algorithm for spinal trauma management[11]

due to rigid boards during prolonged transport, or a short period of rigid immobilization. The use of padded boards may reduce the risk of pressure necrosis.[9]

Any significant trauma victim must be considered to have a spinal cord injury until it can be confirmed otherwise by clinical or radiological assessment. Suspected spinal injuries and SCI can complicate the management of trauma patients because they require special manoeuvres.[12] Preventing secondary damage to the spinal cord from the injured spinal segment, trauma patients must be moved and transported in a safe and stable position, which can be achieved by placing a rigid cervical collar, handling the patient in an en bloc, and transporting in the supine position in spine board or an inflatable transport mattress.[12]

In SCI, early intubation and ventilation are indicated for patients with high cervical injuries (C1–5), which cause disturbances in diaphragmatic breathing, respiratory depression, and CO2 retention - these patients generally have quadriplegia disorders. Emergency management by securing the airway with adequate oxygenation and circulation is the first step to prevent further damage to spinal trauma. Polytraumatic spinal cord injuries are often


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complicated by systemic hypotension due to hemorrhagic and/or neurogenic shock, which aggravates the secondary neurological injury. Aggressive fluid resuscitation must be applied to manage hypotension and/or neurogenic shock that occurs during the initial phase of injury. Recommended treatment targets include SBP 90-100 mm Hg with heart rate 60-100 beats/minute, urine output >30 ml per hour, and normothermia.[9]

## PHARMACOLOGICAL THERAPY

Nerve protection is vital in spinal trauma, which aims to minimize and prevent the expansion of secondary central cord damage by using medical measures that inhibit apoptosis, cell death, and also improve nerve cell survival. High-dose methylprednisolone (MP) therapy in the early stages was once considered positive for neurological restoration in the acute phase of SCI. Intravenous MP injection of 30 mg/kg bolus for 8 hours after SCI (first 3 hours) followed by continuous infusion of 5.4 mg/kg per hour, for the next 23 hours showing an absolute efficiency. While high-dose MP therapy is no longer routinely used in acute SCI, it is still an optional therapeutic method. Methylprednisolone (MP) can even be used in incomplete cervical medulla lesions, and especially in patients with cervical spondylitis myelopathy that requires decompression. Contraindications for the treatment of high doses of MP consist of penetrating injuries and injuries due to shot to the spine, spinal cord injuries without neurological deficits, gastrointestinal bleeding, and diabetes. This treatment should not be given more than 8 hours after injury, and in older patients with a higher risk of pneumonia.

Erythropoietin (EPO) has glioprotective and neuroprotective properties, which reduce cellular infiltration, medullary cavitation, and neuronal cell death. The use of human EPO after a spinal cord injury is carried out by injecting a dose of 5,000 units/kg, either a single intraperitoneal injection or a daily injection for seven days which has shown to have a beneficial effect on neurological recovery; even no definitive conclusions have been drawn because the number of patient subgroups is insufficient. Clinical trials of minocycline, naloxone, and tirilazad also show limited therapeutic effects as nerve protection in patients with SCI. Mannitol can also alleviate secondary spinal cord oedema.

## SURGICAL MANAGEMENT

The time for surgical stabilization in spinal cord injury continues to be controversial, whether early or late, especially in polytrauma cases. The removal of damaged bones, discs, and ligament fragments to decompress swollen cords should limit secondary damage and increase results.[13] The time for surgery usually classified as early, < 72 hours, or late > 72 hours.[14] A prospective study by Cengiz et al. in 27 patients with isolated thoracolumbar spinal cord injury reported the results of the initial surgery in 12 patients (less than 8 hours) and late surgery in 15 patients (15-15 days). This study shows that early surgery has a tendency toward shorter hospitalizations, shorter intensive care units having lower complication rates, and better neurological outcomes.[13]

The decompression operative techniques used were based on the spinal level of injuries or compression, the severity, and the available surgical expertise. The duration and severity of spinal cord compression before surgical intervention significantly affect the postoperative outcome of decompression. The main goal of the surgery in spinal cord injuries is to relieve cord compression as soon as possible and stabilize it to protect the cord. In the trauma setting, there is evidence to suggest that the outcome is better when spinal decompression is done within 48 h.[2,5] The need for surgery in unstable spine injury is not in doubt. Decompression is as essential as stabilization in unstable spinal injuries, where it ensures the cord is no longer at risk of further injury. The neurological function in a patient may not have improved in afterwards, but rehabilitation is more comfortable with a stable spine construct.[2]

## CONCLUSION

The management of spinal trauma continues to evolve from time to time due to a better understanding regarding the mechanism of injury and its biomechanics effect, disease pathophysiology, and improvement for both operative and non-operative treatment. However, there are still some controversial areas regarding management strategies for the treatment of spinal trauma, including the use of corticosteroids such as methylprednisolone sodium succinate, the optimal time for surgical intervention, type and timing of anticoagulation prophylaxis, the role of magnetic resonance imaging, and type and time of rehabilitation. A tailored and multidisciplinary approach must be applied for better results in the management of spinal trauma.

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## DISCLOSURE

The author reports no conflicts of interest in this work.

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