



Spinal Cord Injuries in Emergency Medicine

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Authors' contributions

This work was carried out in collaboration among all authors. All authors read and approved the final manuscript.

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ABSTRACT

Spinal cord injury is a serious medical disorder that frequently leads in significant morbidity and permanent impairment. Direct damage to the spinal cord or compression owing to broken vertebrae or masses such as epidural hematomas or abscesses are the most common causes of spinal cord injury. When examining a blunt trauma victim, medical professionals are taught to presume the

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patient has a spinal column damage unless it is proven otherwise. The early examination of a patient with a suspected cervical spinal injury in the emergency department (ED) is no different than that of any other trauma patient. The ABCs, or airway, breathing, and circulation, procedures are being taken into consideration. In acute spinal cord damage, hypotension can be hemorrhagic or neurogenic. Because of the high prevalence of concomitant injuries and vital sign confusion in acute spinal cord injury, a thorough search for hidden sources of bleeding is required. Surgical removal of bone fragments, foreign objects, herniated discs, or broken vertebrae that appear to be compressing the spine is frequently required. In order to avoid future discomfort or deformity, surgery may be required to stabilize the spine. In this review we'll be looking at spinal cord injury, it's diagnosis and treatment.

Keywords: Spinal cord injury; trauma; traumatic TSIs; tetraplegia; spinal immobilization.

1. INTRODUCTION

Spinal cord injury is a serious medical disorder that frequently leads in significant morbidity and permanent impairment. When the axons of neurons that go through the spinal cord are damaged, motor and sensory function is lost below the degree of lesion. Injury is frequently the outcome of substantial trauma, and initial injury is often irreparable [1] These injuries are very costly and harmful because they disproportionately impact patients under the age of 30, cause considerable functional impairment for the rest of the person's life, and put the person at risk for a variety of consequences that raise morbidity and mortality. SCI is expected to have a 2-to-4-billion-dollar economic impact [2,3,4].

Major trauma is the leading cause of death in people under the age of 50 in the United Kingdom, and survivors often have severe disability. In the United Kingdom, an estimated 1200 traumatic spinal cord injuries occur each year, and over 40000 individuals live with long-term disability as a result of SCI, however other organisations believe the number is greater. Around 14% of spinal fractures cause injury to the spinal cord, with 50% of those fractures being incomplete. Half of patients will walk on discharge if properly handled [5-10] As a result, proper early care is critical, with the goal of preserving cord function while preventing subsequent difficulties [5-10].

The yearly incidence of traumatic spinal injury (TSI) in the United States is believed to be around 40 per million people. Over half of the patients have isolated spine injuries, whereas roughly a quarter have concurrent brain, chest, and/or significant extremities injuries. Though once assumed to be a condition only affecting young men, new epidemiological research on

TSI patients show a bimodal distribution. As predicted, the initial peak comes in teens and young adults. The second peak, on the other hand, occurs in the senior population (those over 65 years old). Injury to the spine is more likely to happen in locations with the most movement. Cervical TSIs account for more than half of all traumatic TSIs, and they're linked to significantly higher short- and long-term morbidity than injuries to the cord at the thoracic or lumbar levels. Incomplete tetraplegia is the most common injury (31 percent), followed by total paraplegia (25 percent), complete tetraplegia (20 percent), and incomplete paraplegia (10 percent) (19 percent) [11].

Traumatic spinal cord injury (TSCI) is a relatively uncommon yet life-threatening illness with significant rates of morbidity and death. Global incidence rates have been reported to range from 10 to 83 incidences per million. In comparison to these global rates, the Dutch incidence rate is modest, with 14.0 cases per million in 2010. TSCI-related mortality rates in the Netherlands are 3.4 per million, which is lower than the European average of 6.7 per million. Global TSCI investigations have revealed a bimodal age distribution, with trends between 15 and 30, and over 65. The major TSCI mechanism in Europe differs per nation. In the Netherlands, the most common causes of TSCI are falls followed by motor accidents [12].

Injury strikes low- and middle-income countries (LMICs) disproportionately; LMICs account for more than 90% of the 5.8 million yearly fatalities attributable to injury. Injury-related mortality account for ten percent of all deaths worldwide, which is 32 percent more than deaths from malaria, TB, and HIV/AIDS combined. Though traumatic spinal cord injury (TSCI) is a substantial contribution to the global burden of injury, it is defined as damage to the spinal cord

nerve tissue and vertebral or ligamentous injuries that compromise spinal cord integrity. Because of insufficient prehospital treatment, limited availability of inpatient speciality TSCI care, and restricted post-TSCI rehabilitation, TSCI morbidity and death are greater in low- and middle-income African nations than in high-income ones. Regional patterns show that as LMICs continue to motorize, the number of road traffic injuries will rise, adding to the global TSCI burden. Therefore, strategies should aim at prevention more due to the major consequences of such injuries [13-17].

2. ETIOLOGY AND PATHOPHYSIOLOGY

Motor vehicle crashes are the main cause of spinal cord injury in the United States, accounting for 38 percent of new SCI each year. Thirty percent are caused by falls, thirteen percent by violence, nine percent by sports injuries, and five percent by medical and surgical causes [1,2].

Direct damage to the spinal cord or compression owing to broken vertebrae or masses such as epidural hematomas or abscesses are the most common causes of spinal cord injury. Compromise of blood flow, inflammatory processes, metabolic derangements, or exposure to toxins are all less prevalent causes of spinal cord injury [1].

abrupt, severe impact on the spine that fractures or dislocates vertebrae is the most prevalent cause of SCI. Primary damage occurs when "displaced bone fragments, disc materials, and/or ligaments bruise or rip into the spinal cord tissue" during the first mechanical pressures imparted to the spinal cord at the time of injury. Notably, most spinal cord injuries do not result in the full amputation of the spinal cord (10). The following are the four main mechanisms of initial damage that have been identified: (1) Consistent compression plus impact; (2) Transient compression plus impact; (3) Distraction; (4) Laceration/transection. Impact with persistent compression is the most frequent type of primary injury, which usually happens as a result of burst fractures with bone fragments compressing the spinal cord or fracture-dislocation injuries. Impact alone with temporary compression is seen less frequently, although hyperextension injuries are the most prevalent [18-23].

Secondary injury is a set of biological events that start within minutes and progress to self-

immolation weeks or months after the main injury. Vascular damage, ionic imbalances, free-radical production, the early inflammatory response, and neurotransmitter buildup are all part of the acute phase of secondary injury following SCI (excitotoxicity). The subacute phase follows, which involves surviving axon demyelination, Wallerian degeneration, matrix remodelling, and glial scar development [1].

In addition, the results of SCI in military and civilian instances varies significantly. Blast damage, which frequently affects numerous segments of the spinal cord, is the most prevalent cause of SCI in the battlefield as compared to civilian SCI. Blast SCI is also connected with greater severity ratings and longer hospital stays. A study of American military veterans who had SCI in a conflict zone between 2001 and 2009 found that their injuries were more severe and had a worse neurological recovery than civilian SCI. Furthermore, in combat injuries, lower lumbar burst fractures and lumbosacral dissociation are more common [18].

Following SCI, neuroinflammation can be useful or harmful depending on the time-point and the condition of immune cells. Inflammatory processes during the first three days after SCI include the recruitment of blood-borne neutrophils, resident microglia, and astrocytes to the injury site. The second phase, which begins three days after the injury, involves the recruitment of macrophages, B-, and T-lymphocytes to the injury site. Antigen-presenting cells activate CD4+ helper T cells, which produce cytokines that drive B cells to make and release antibodies, exacerbating neuroinflammation and tissue death. In the acute phase of SCI, neuroinflammation is more active [1].

Primary damage might result in secondary damage. The most prominent mechanism is a loss of energy caused by ischemia and decreased cellular perfusion. Ischemia can occur within minutes of a traumatic SCI, and if left untreated, further damage can occur within the first three hours and last for at least 24 hours. Following SCI, a series of pathological changes in the nerve tissues, including bleeding, demyelination, edoema, and cavity development with axonal and neuronal death, as well as a number of pathological changes in the nerve tissues, might enhance infarction. Excitotoxicity, oxidative damage, and ischemia can all be caused by high glutamate levels, whereas Ca²⁺-dependent nitric oxide production can produce

secondary spinal cord injury. Increased free radical damage and lipid peroxidation in the cell membrane, as well as additional injury signalling cascades in the wounded tissue locations, can eventually lead to neuronal death following secondary traumas [24-31].

3. EVALUATION

When examining a blunt trauma victim, medical professionals are taught to presume the patient has a spinal column damage unless it is proven otherwise. A number of organisations have recently proposed that the term "spinal immobilisation" be replaced with "spinal motion restriction," and that spinal motion restriction, which includes the use of cervical collars and backboards, should not be used in patients who are at low risk of spinal column injury [11].

It's crucial to remember that the amount of energy required to cause **spinal shock** and fracture after a traumatic incident is quite large, thus patients should be extensively evaluated for additional tissue and organ damage. When a patient with spinal shock arrives at the emergency department, the trauma activation code should be used so that the trauma team may do a comprehensive workup on the patient. Motor and sensory reflexes, such as the bulbocavernosus reflex and the anal wink reflex, should be included in a thorough spinal evaluation. Not only do skeletal muscles lose motor function and strength, but so do internal organs including the colon and bladder. Constipation and urine retention result from this reduction. Before any interaction with family or the patient, it is critical to record an American Spinal Injury Association (ASIA) score so that predictive long-term expectations may be made with reasonable accuracy [32].

These criteria should be considered when evaluating a person with a suspected thoracic or lumbosacral spine injury: [33].

- 65 years of age or older, with thoracic or lumbosacral spine discomfort
- axial load to the head or base of the spine – for example, falls landing on feet or buttocks, high-speed motor vehicle collisions, rollover motor vehicle accidents, lap belt restraint only, ejection from a motor vehicle, accident involving motorised recreational vehicles, bicycle collisions, horse riding accidents)

- pre-existing spinal disease, or osteoporosis, whether recognised or at risk — for example, steroid usage
- There is a possibility of a spinal fracture in another part of the spine.
- On examination, aberrant neurological symptoms (paraesthesia, weakness, or numbness):
 - unusual neurological symptoms (motor or sensory deficit)
 - a new deformity or soreness in the bony midline (on palpation)
 - discomfort in the bony midline (on percussion)
 - discomfort in the midline or in the spine (on coughing)

The early examination of a patient with a suspected cervical spinal injury in the emergency department (ED) is no different than that of any other trauma patient. The ABCs, or airway, breathing, and circulation, take precedence. As long as spinal protection is maintained, the majority of spine injuries may be postponed while other life-threatening injuries, such as bleeding or traumatic brain damage, are addressed. Clinicians should do their primary survey, which includes determining the patient's ABCs and level of impairment. Finally, the physician should expose the patient completely to search for symptoms of damage [11].

Because radiographs are only as excellent as the first and last vertebrae visible, all vertebrae must be accurately shown. Failure to capture appropriate pictures is a common cause of missing injury (eg, cervical spine radiograph that incompletely depicts the C7-T1 junction). however, that radiography is insensitive to tiny vertebral fractures. In symptomatic trauma patients with neck discomfort, published clinical criteria have established guidelines for cervical spine radiography. In extensive clinical studies, the **NEXUS** (National Emergency X-Radiography Utilization Study) criteria and the Canadian C-spine regulations were verified. These algorithms may be used to help doctors decide whether or not cervical spine imaging is necessary [34-37].

The American Spinal Injury Association (ASIA) Impairment Scale is used to grade SCI. From A through E, the grading system is based on the severity of the injury.

- ASIA A: Total paralysis with loss of motor and sensory functions.

- ASIA B: Incomplete damage with sensory function retained but total motor function lost.
- ASIA C: Incomplete damage with retained motor function below the degree of injury; fewer than half of these muscles exhibit MRC grade 3 strength.
- ASIA D: Incomplete damage with retained motor function below the injury level. At least half of these muscles have MRC (Medical Research Council) grade 3 strength
- ASIA E: A normal motor and sensory [1]

To be considered appropriate, the cervical spine radiographs must include the C7-T1 intersection. Subtle symptoms (such as increased prevertebral soft tissue edoema or a widening of the C1-C2 preodontoid space) suggest potentially unstable cervical spine injuries that, if not recognised, might have significant repercussions. In the absence of a fracture, dynamic flexion/extension views are safe and efficient for diagnosing concealed ligamentous damage of the cervical spine. A typical 3-view cervical spine series with flexion/extension images has a negative predictive value of above 99 percent. Because occult injury is uncommon in the presence of normal results on cervical spine radiography and CT scanning, clinical judgement and the nature of injury should be utilised to determine if flexion/extension views should be requested [34].

Historically, the typical first examination for cervical spine injury was a 3-view cervical spine radiograph series. The Eastern Association for Trauma Surgery (EAST) and the American College of Radiology, however, advocate computed tomography (CT) with multi-planar reconstruction as the first imaging modality if imaging is judged acceptable by the physician. If plain radiographs are still utilised in the case of suspected cervical spine injuries, they should only be used in individuals with a low pre-test likelihood [11].

For suspected spinal cord lesions, ligamentous injuries, or other soft-tissue injuries or disease, magnetic resonance imaging (MRI) is the best option. Nonosseous lesions such as extradural spinal hematoma, abscess or tumour, disc rupture, and spinal cord bleeding, contusion, and/or edoema should all be evaluated using this imaging modality. Secondary damage, which results in edoema and/or bleeding, is the most common cause of neurologic impairment. The

best diagnostic imaging for depicting these abnormalities is an MRI [34].

4. MANAGEMENT

Trauma care begins before you arrive at the hospital. Up to 25% of traumatic spinal cord injuries happen after the main injury, such as during extraction, transportation, or handling. Spinal immobilisation is a priority in patients with a mechanism of damage consistent with spinal trauma, and it leads to improved results. The gold standard in spinal protection is three-point immobilisation of the cervical spine with blocks, collars, and tape, as well as a hard spinal board to keep the majority of the spine aligned. Although these procedures are necessary to prevent future spinal damage, they come with their own set of problems, including pressure sores, aspiration, and increased intracranial pressure, as well as the potential to obstruct early examination. Immobilizing devices should be removed as soon as it is safe to do so [5].

All patients with SCI require prompt, therapeutic fluid resuscitation to maintain tissue perfusion, although caution must be exercised to avoid fluid overload. Fluid resuscitation is the initial line of therapy for hypotension. The objective is to keep tissue perfusion at its best while also resolving shock. The ideal mean arterial blood pressure for maintaining spinal cord perfusion and the appropriate resuscitation end point remain unknown. Patients with acute SCI who were given fluids and vasopressors for a minimum of 7 days to attain a mean arterial pressure of 85 mmHg had good results in uncontrolled trials [38].

Although it is acknowledged that the evidence suggesting this target MAP improves the neurologic prognosis after acute SCI is somewhat limited, clinical practise recommendations have suggested that MAP be maintained at 85–90 mmHg for 5–7 days post-injury. The damaged chord, however, appears to be sensitive to variations in blood pressure. Even minor improvements in MAP during the first 3–7 days post-injury can enhance neurologic recovery, according to Hawryluk et al. This indicates that attempts to improve SC perfusion by a few mmHg can result in clinically meaningful neurologic improvement. However, in certain people, raising MAP through the use of high-dose vasopressors might lead to drug-related problems. In fact, managing an acute SCI

patient's hemodynamics within the first week after damage is crucial and difficult [39].

Examine the person for a spinal injury, first considering the factors given below. Check to see if the person has any distracting injuries.

- is inebriated or under the influence of drugs
- has a decreased degree of consciousness is confused or uncooperative
- suffers any kind of back pain
- Is there any weakness in his hands or feet? (motor assessment)
- Sensation in the hands and feet is altered or nonexistent (sensory assessment)
- Priapism is a kind of priapism that occurs when (unconscious or exposed male)
- has a history of previous spinal issues, such as previous spinal surgery or disorders that predispose to spine instability [33].

When establishing a secure airway or treating hypotension compounded by neurogenic shock, treating additional life-threatening injuries in the face of suspected SCI might be difficult. Hypoxia and hypotension can also be caused by other traumas, causing subsequent injury to the spinal cord. To guarantee that life-threatening injuries and other spinal cord injuries are not missed, all trauma patients must be examined and treated according to an Advanced Trauma and Life Support (ATLS) protocol. Priority should be given to life-threatening injuries, but spinal alignment must always be preserved [5].

Hemodynamic instability may be linked to acute SCI. Neurogenic shock is prevalent in individuals with acute tetraplegia or high-level paraplegia (T1–T4), although alternative sources of hypotension should be addressed before presuming the cause of hypotension is the cord injury. All potential causes of hemodynamic instability, including bleeding, pneumothorax, myocardial damage, pericardial tamponade, sepsis associated to abdominal injury, and other traumatic and medical etiologies, should be considered by the physician addressing traumatic SCI. In the insensate spinal cord damaged patient, physical examination and subjective patient reports are difficult, hence a chest/abdomen/pelvis CT or other imaging modalities should be used to rule out other probable causes of hypotension [38].

In contrast to individuals with hypovolemic shock caused by bleeding, people with neurogenic shock are usually hypotensive and have warm, dry skin. This is due to a decrease of sympathetic tone, which prevents blood flow from the periphery from being redirected to the core circulation. Other causes of hypotension, such as hemorrhagic shock or tension pneumothorax, may be present in a patient with numerous traumas. These factors must be recognised and treated as soon as possible. Bradycardia is a common symptom of neurogenic shock and can help distinguish it from other types of shock. Because there is no tachycardia, it is important not to assume that a patient has neurogenic shock. In the case of bleeding, young healthy people, the elderly, and patients on pre-injury beta-blockers are unlikely to experience tachycardia [11].

After excluding occult causes of bleeding, the first line of therapy for neurogenic shock is fluid resuscitation. The initial treatment of choice is judicious fluid replacement with isotonic crystalloid solution up to 2 L. Because these individuals are at risk for acute respiratory distress syndrome, overzealous crystalloid treatment may cause pulmonary edema (ARDS). Patients with full cord lesions should have a systolic blood pressure (BP) of 90-100 mm Hg; systolic BPs in this range are common. Animal and human research show that keeping systolic blood pressure over 90 mm Hg and avoiding hypotensive episodes is beneficial [33].

In the past, methylprednisolone (Solu-Medrol) administered through an arm vein (IV) has been used to treat acute spinal cord injuries. However, a new study has found that the risks of using this medicine, such as blood clots and pneumonia, exceed the benefits. As a result, methylprednisolone is no longer indicated as a first-line treatment following a spinal cord injury.

Patients with spinal shock should be treated in an ICU environment, since various problems are likely to emerge as a result of the injury. treatment with methylprednisone is debatable, with some studies suggesting a little benefit and others showing more bad side effects than advantages. its advisable to begin a brief trial of methylprednisone with a loading dosage of 30 mg/kg followed by a maintenance dose of 5 mg/kg/h for the following 24 hours if the patient is young and has no underlying conditions that might be aggravated by steroid usage. With lesions above the T6 level, neurogenic shock is

common. Initial therapy should include a norepinephrine drip and the use of atropine sparingly for bradycardia. Hypotension recovers after a few days, and intravenous (IV) drips should be progressively reduced [32].

Maintaining sufficient oxygenation and perfusion of the damaged spinal cord is the most critical therapy issue; supplementary oxygenation and/or mechanical breathing may be necessary. Atropine can be used to treat bradycardia that is hemodynamically substantial [33].

Inotropic support with dopamine or norepinephrine is only necessary in rare cases; this should be saved for patients who have reduced urine output despite appropriate fluid resuscitation; in most cases, modest doses of dopamine in the 2- to 5-mcg/kg/min range are acceptable [33].

Surgical removal of bone fragments, foreign objects, herniated discs, or broken vertebrae that appear to be compressing the spine is frequently required. In order to avoid future discomfort or deformity, surgery may be required to stabilise the spine.

Rehabilitation is an important component of the healing process, and these patients benefit from intensive rehabilitation under the supervision of physiatrists, physical therapists, and occupational therapists. Once the patient is ready to be discharged from the inpatient rehabilitation unit, rehabilitation will be maintained on an outpatient basis [1].

5. CONCLUSION

Spinal cord injury is serious condition, management of such cases begins before arriving at emergency department by immobilizing the spine to prevent any serious damage to the spinal cord. ABCs and other trauma/emergency procedures should be done as with any emergency case, assessment of the spinal injury is done first then treatment is done based on the assessment. Hemodynamic instability may be linked to acute SCI, therefore it must be addressed effectively. Surgery is needed in many cases to remove any bodies that may be compressing the spine followed by immobilization.

CONSENT

It is not applicable.

ETHICAL APPROVAL

It is not applicable.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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