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Review Article

What to know in Managing Spinal Cord Injury for Anesthesiologists?

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ABSTRACT

Background: Spinal cord injury (SCI) is trauma to the area of the vertebrae resulting in spinal cord lesions resulting in neurological disorders, depending on the location of the spinal nerve damage and the injured nerve tissue. The symptoms of SCI can range from pain and paralysis to incontinence. SCI due to trauma is estimated to occur in 30–40 per million population per year, and about 8000–10,000 sufferers each year, generally, occurs in adolescents and young adults. Although the annual incidence of events is relatively low, the cost of care and rehabilitation for spinal cord injuries is very high, at around US $ 53,000/patient.

Methods: This study aims to provide an overview of how to manage SCI. This study reviewed various sources then reviewed as a literature review.

Conclusion: Treatment in the hospital includes all systems that may experience complications from SCI, starting from the respiratory, cardiovascular, urological, gastrointestinal, skin, to non-operative. and operative reduction measures.

Keywords: Spinal cord injury, Emergency, Management

INTRODUCTION

Spinal cord injury (SCI) is trauma to the area of the vertebrae resulting in spinal cord lesions resulting in neurological disorders, depending on the location of the spinal nerve damage and the injured nerve tissue. The symptoms of SCI can range from pain and paralysis to incontinence.[1]

SCI was first recorded in the history of medical science around 1700 BC on the Edwin Smith papyrus. The most common causes of SCI were traffic accidents (50%), falls (25%), and sports-related injuries (10%); and the rest is due to work accidents. SCI due to trauma is estimated to occur in 30–40 per million population per year, and about 8000–10,000 sufferers each year, generally, occurs in adolescents and young adults. Although the annual incidence of events is relatively low, the cost of care and rehabilitation for spinal cord injuries is very high, at around US $ 53,000/patient.[1]

The mortality rate is estimated to be 48% in the first 24 h. About 80% died on the spot from cervical spine injury with the greatest risk of trauma, with the most frequent levels at C5, followed by C4, C6, then T12, L1, and T10. Based on the disability that occurred, 52% of cases had paraplegia and 47% had tetraplegia.[1]

ETIOLOGY

As many as 10% of patients with decreased consciousness who are sent to the emergency room (ER) due to traffic accidents always get cervical injuries, both injuries to the cervical spine, supporting tissues, and injuries to the cervical spine. Traffic accidents and falls are the cause of the majority of cervical fractures. Sub axis cervical trauma (C3–7) is more common than C1 and C2 and is a potential trauma that deserves a lot of attention. It is almost always thought that cervical trauma will occur in patients with a history of high-speed motor vehicle accidents, significant face and head trauma, neurological deficits, neck pain, and multiple traumas. According to the mechanism of injury, cervical injuries are divided into flexion, rotational flexion, extension, rotational extension, vertical compression, lateral flexion, and mechanisms that are not yet clear.[1]

ANATOMY OF THE SPINAL MEDULLA

The spinal cord is a cylindrical organ that starts from the foramen magnum in the skull up to two thirds of the entire length of the vertebral canal, is continuous with the medulla oblongata in the brain, and the tip of the spinal cord is located at the lower boundary of the first lumbar vertebra in adults and the lower border of the vertebrae third lumbar in children. The spinal medulla is surrounded by three layers of the meninges, including the dura mater, arachnoid mater, and pia mater. In addition, the cerebrospinal liqueur (CSF) in the subarachnoid cavity also provides additional protection for the spinal cord.[2]

The spinal cord consists of 31 segments, including eight cervical segments, 12 thoracic segments, five lumbar segments, five sacral segments, and one coccygeal segment. The spinal nerves come out of each segment of the spinal cord (totaling 31 pairs of spinal nerves) and consist of motors or anterior roots (roots) and sensory or posterior root. The name of the spinal nerve is carried out based on the area where the nerve arises through the vertebral canal. Spinal nerves C1 to C7 arise from above the vertebral column C1-C7, while C8 between vertebral columns C7-T1. The other spinal nerves emerge from below the vertebral column in question.[2]

The motor functions of the spinal nerves include, C1-C2 innervates the neck muscles, C3-C5 forms the phrenic nerve which supplies the diaphragm, C5-T1 supplies the muscles of the upper limb, thoracic segment supplies the thoracoabdominal muscles, and L2-S2 supplies the muscles of the lower extremities. Some important dermatomes that provide an overview for the sensory function of the spinal nerve include C2-C3 for the posterior part of the head-neck, T4-5 for the areola mammae area, and T10 for the umbilicus, parts of the upper limb: C5 (anterior shoulder), C6 (mother fingers), C7 (index and middle fingers), C8 (little finger), T1 (medial antebrachia), T2 (medial part of the brachial), and T2/T3 (axillary), and lower limb parts: L1 (anterior and medial part of femoral), L2 (anterior part of the femoral), L3 (knee), L4 (medial malleolus), L5 (dorsum pedis and fingers 1–3), S1 (finger 4–5 and lateral malleolus), and S3/Cox (anus).[2]

The spinal cord consists of two substances, namely, the gray matter which is located internally and the white matter which is located externally. In general, the substantia alba consists of ascending (sensory) and descending (motor) tracts, while the gray matter can be divided into ten laminae or three parts (anterior, posterior, and lateral horns) which are composed of nuclei that play a role in the action potential of neurons.[2]

The sensory tracts (ascending tracts) of the spinal cord include, among other things, the lateral spinothalamic tract which carries the senses for pain and temperature, the anterior spinothalamic for touch (coarse/crude touch) and pressure, the dorsal column tract (posterior white column) for smooth touch (two-point discrimination), proprioceptive function and vibration, and other tracts such as, spinocerebellar (posterior and anterior), cuneocerebellar, spinotectal, spinoreticular, and spino-olivary.[2]

Studies of the course of the tracts (especially regarding the level at which decussation occurs) in the white matter of the spinal cord will provide a comprehensive understanding of the clinical manifestations of patients with spinal cord trauma. Perceptions of soft touch, proprioceptive, and vibration (from the dorsal column tract) do not cross (decussation) before they reach the medulla oblongata, whereas the lateral and anterior spinothalamic tracts intersect in three levels of the segment where it enters. On the other hand, the main motor tract (corticospinal) undergoes a decussation at the level of the medulla oblongata. This results in a lesion of the corticospinal tract or dorsal column causing ipsilateral (for corticospinal) motor paralysis and loss of ipsilateral (for corticospinal) sense of touch, proprioceptive, and vibration of the lesion. Conversely, lesions on the tract that carry the perception of pain, temperature, pressure, and rough touch cause a loss of that perception in the contralateral region of the lesion.[2]

Apart from the tract for sensory and motor functions, the spinal cord also plays a role in autonomic function. Sympathetic nerve function is influenced by the cranial nerve T1-L3 (thoracolumbar), while the parasympathetic nerve functions in S2-S4. Spinal cord lesions in the area concerned can cause autonomic nerve disorders according to the level of the lesion. One of the clinical correlations of impaired sympathetic nerve function due to lesions higher than T6 is neurogenic shock due to loss of sympathetic tone in arterial vessels, whereas micturition and erectile dysfunction are due to parasympathetic tone disorders.[2]

The perfusion of the spinal cord consists of one anterior spinal artery and two posterior spinal arteries. The anterior spinal arteries provide blood supply to two-third of the anterior part of the spinal cord. The presence of lesions in these vessels causes dysfunction of the corticospinal tract, lateral spinothalamic, and autonomic pathways (paraplegia, loss of pain and temperature perception, and autonomic dysfunction). The posterior spinal arteries primarily provide blood supply to the dorsal column and posterior gray matter. Both arteries arise from the vertebral artery. Several radicular branches of the thoracic and abdominal aorta provide collateral hemorrhage to the spinal cord.[2]

SECONDARY DAMAGE MECHANISM

Primary damage is a nidus or starting point for secondary damage. Secondary damage is caused, among other things, by neurogenic shock, vascular processes, such as bleeding and ischemia, excitotoxicity, calcium-mediated secondary lesions, electrolyte disturbances, damage due to immunological processes, apoptosis, disorders of the mitochondria, and other processes.[3,4]

CLASSIFICATION OF SCI

Spinal cord injuries can be divided into complete and incomplete based on the presence/absence of sustained function under the spinal medullary injury (SCI) lesion classified as complete and incomplete. Complete SCI is a total loss of sensation and voluntary motor function whereas incomplete is a mixture of loss of sensation and voluntary motor function. Another definition is that complete SCI is characterized by the absence of sensory and motor functions that come from below the level of the injury while incomplete SCI still has sensory and motor functions below the level of the injury.[5]

CLINICAL MANIFESTATIONS

There are five main syndromes of incomplete SCI, according to the American SCI Association, namely: (1) Central cord syndrome (CCS); (2) anterior cord syndrome; (3) brown-sequard syndrome; (4) cauda equina syndrome; and (5) conus medullary syndrome. A very rare incomplete syndrome is posterior cord syndrome. CCS results from incomplete injury to the central cervical segment of the spinal cord, most commonly the middle to the lower cervical segment. Trauma that causes the ligamentum flavum (a strong ligament that connects the laminae of the vertebrae to protect the nerves and spinal cord and stabilizes the spina so that there is no excessive movement of the vertebrae) which eventually pinches the spinal cord from the posterior and/or as a result of compression by osteophytes or disc material from the anterior. Compression also causes impaired perfusion of the anterior spinal artery. On physical examination, CCS is usually limited to disorders of the neurologic system, consisting of combined upper motor neuron (UMN) and lower motor neuron lesions supplying the upper limb and resulting in partial flaccid paralysis; and lesions predominantly on the UMN supplying the lower extremities resulting in spastic paralysis. Upper limb disorders are usually more severe than lower limb disorders, and mainly occur in the distal hand muscles. Sensory loss occurs to some degree, although sacred sensations are usually intact. The ability of anal contraction and sphincter tone and Babinski reflex should be checked.[1]

CCS may result from bleeding into the central portion of the spinal cord, or as a result of disruption of axons in the lateral cornu at the level of injury but does not result in significant damage to the gray matter. CCS can also occur as a result of dislocation and compression fractures, particularly in individuals with congenital narrowing of the spinal canal. Compression pressure which is anteroposterior in direction results in more severe damage to the central area. The above injury mechanisms result in the most severe damage to the central spinal cord and less damage to the periphery of the spinal cord. Injury to this area results in damage to the lateral spinothalamic tract and corticospinal tract with characteristic symptoms. Both motor and sensory disturbances in CCS result from the distinctive lamination pattern of the corticospinal and spinothalamic tracts in the spinal cord. The lateral spinothalamic tract has a laminated arrangement in a somatotopic pattern, where the fibers originating from the sacral segment are most dorsolateral, followed by the fibers of the lumbar and thoracic segments, while the fibers of the cervical segments are the most ventromedial. Because CCS is caused by an injury to the central part, the cervical fibers are seriously injured while the sacral fibers are not injured.[6]

SCI is characterized by the presence of tetraplegia or paraplegia, partial, or complete and the grade or level depends on the area of the lesion or SCI. Tetraplegia or quadriplegia is a loss of sensory and motor function in the cervical segment of the spinal cord. Meanwhile, paraplegia is a disturbance of sensory and motor function in the lumbar thoracic segment and the sacrum. Respiratory failure and pulmonary disorders mainly occur in SCI to the cervical and thoracic segments. Lesions above C3 will result in complete paralysis of the respiratory muscles and diaphragm. In any lesion above C4, function of the diaphragm muscles, intercostal muscles, and additional respiratory muscles may be lost. Lesions above C5 can affect diaphragm function and clearly this necessitates mechanical/artificial ventilation. C4-C5 lesions will present a variety of diaphragm disorders. Lesions of C6-T12 are usually characterized by an intact diaphragm that can provide 90% of the volume of the diaphragm expansion but the intercostal muscles cannot function properly to stabilize the ribs.[6]

MANAGEMENT

Treatment in the hospital includes all systems that may experience complications from SCI, starting from the respiratory, cardiovascular, urological, gastrointestinal, skin, to non-operative, and operative reduction measures.[7]

EARLY HANDLING

The initial principle when accepting patients in the hospital ER is generally the same, namely, being followed-up according to trauma management; advanced trauma life support, namely, primary and secondary surveys. If at the time of admission to the hospital, immobilization of the spine has not been carried out, then the initial action that must be taken is immobilization. The difference with pre-hospital treatment is that a complete neurological examination must be performed (if the ABC vital signs have stabilized). A complete neurological examination was performed according to the 2011 revised International Standards for Neurological Classification of SCI published by ASIA. At the time of initial neurologic examination, the level of the lesion can be determined, complete or incomplete lesions, and the presence or absence of a spinal shock phase. Radiological examination is then performed to see or rule out the possibility of SCI.[3,7]

SPECIFIC TREATMENT FOR COMPLICATIONS OF SCI

Respiratory system

Respiratory tract complications are a major cause of morbidity and mortality in patients with SCI. Lesions directly related to respiratory function were lesions at the level of C5 and above, whereas lesions at the thoracic level only interfered with coughing and lesions in the lumbar did not affect at all. Patients with lesions above C5 should be intubated and mechanically ventilated because a gradual decrease in respiratory function may occur. Respiratory function must be closely monitored by checking oxygen saturation, vital capacity of the lungs, and periodic blood gas analysis. Sputum retention generally occurs within few days of injury due to impaired effective cough function, leading to atelectasis, and pneumonia. Chest physiotherapy, assisted cough, and regular breathing exercises can prevent atelectasis and lung infections.[3,5,7]

Cardiovascular system

The major and crucial complication of the cardiovascular system due to SCI is neurogenic shock resulting from spinal shock. In general, neurogenic shock occurs in lesions above T6 due to loss of sympathetic tone. The loss of tone causes vasodilation and bradycardia leading to hypotension and shock. Shock in SCI must be distinguished between hypovolemic and neurogenic because if too much fluid is given in neurogenic shock, pulmonary edema will occur. Management of neurogenic shock includes IV fluids, vasopressors with alpha- and beta-adrenergic characteristics (such as norepinephrine, epinephrine, and dopamine), atropine to increase pulse, and avoidance of hypothermia due to vasodilation. Mean arterial pressure (MAP) should be targeted at above 70 mmHg, although several studies have shown that MAP >85 mmHg provides a better prognosis.[3,5,7]

Thromboembolism is a complication that may also occur in paraplegia/tetraplegia patients due to SCI. The highest incidence of pulmonary embolism occurs at week 3 after injury and is the most common cause of death in SCI patients who survive trauma. If there are no contraindications such as capitis or thoracic trauma, anti-embolism stockings are used during the first 2 weeks after trauma and anticoagulant use is started within 72 h after trauma for 8–12 weeks (low molecular weight heparin is better than warfarin).[3,5,7]

Urological system

After the onset of severe SCI, the bladder is unable to pass urine spontaneously, and untreated patients may develop urinary retention which leads to urinary reflux and renal failure. As soon as the patient arrives at the hospital, a Foley catheter must be placed. The recovery time for the micturition reflex varies, generally 6–8 weeks, but can be up to 1 year (there is literature that says no return).[3,5,7]

The intermittent catheterization program begins during the subacute phase, when fluid intake and output stabilize. This is done to prevent urinary tract infections (UTIs). However, if the Foley catheter is removed too early, detrusor muscle damage and reflux may occur due to the high-pressure filling of the bladder.[3,5,7]

Complications of SCI in the urinary tract are UTIs. Symptomatic UTIs accompanied by fever, leukocytosis, and pyuria should be treated with adequate antibiotics for 7–14 days, whereas asymptomatic infections do not need to be treated routinely. The application of sterile methods is important for prevention of UTIs.[3,5,7]

Gastrointestinal system

Patients with SCI should at least receive fluids intravenously for 48 h because paralytic ileus is common with severe SCI. In this condition, a nasogastric tube is inserted (NGT) and nil per oral is done until the bowel sounds return to normal. Total parenteral nutrition should be given. If the paralytic ileus lasts a long time, abdominal distension occurs and can cause disruption of diaphragmatic movement. Acute peptic ulcers can occur with bleeding or perforation, although they are not common, they are dangerous complications. Therefore, the administration of H2 receptor antagonists or

proton pump inhibitors should be started immediately and given at least 3 weeks after trauma.[3,5,7]

Evaluation of defecation function should be performed early and management started aggressively as soon as bowel sounds develop and bowel motility is normal. The height of the lesion determines the defecation function, among others, the lesion above T12 causes hyperreflexia and spasticity of the anal sphincter, while the lesion below it causes areflexia and flaccid of the sphincter. Bowel emptying method, with a combination of suppositories and anorectal stimulation, stimulates an evacuation pattern in the distal colon.[3,5,7]

SKIN

Decubitus ulcers will always be a complication of SCI; therefore, prevention needs to be done early. In the acute phase, the patient is positioned on a left-right oblique every 2 h to prevent ulcers. The use of a foam or water mattress can help reduce the pressure on the bony ridges; however, the patient's position must be changed every 2 h.[3,5,7]

USE OF CORTICOSTEROIDS

The use of corticosteroids (especially high doses of methylprednisolone) is currently subject to controversy. A study conducted by National Acute SCI Study (NASCIS) 2 shows that high doses of methylprednisolone (bolus 30 mg/kg in 15 min then followed by 5.4 mg/kg in 23 h) starting within 8 h after closed SCI improve prognosis neurological patient. The NASCIS 3 study later added that methylprednisolone therapy initiated within 3 h after trauma should be continued for 24 h, whereas those initiated between 3 and 8 h post-trauma should be continued for 48 h. The consortium for spinal cord medicine does not recommend the use of any neuroprotectants (steroids, ganglioside GM-1, gacyclidine, tirilazad, and naloxone) because clinical evidence of improvement in the final prognosis has not been definitively obtained.[3,5,7]

NON-OPERATIVE AND OPERATIVE REDUCTION THERAPY

After the systemic parameters have stabilized, attention will be paid to stabilization and alignment of the spine and spinal cord. Any unstable SCI must be stabilized to prevent further damage from movement and also to release compression of the spinal cord. Patients with cervical SCI can be treated using skeletal traction to reduce dislocations, release compression of the spinal cord in burst fractures, and spinal splints. Skeletal traction to restore or maintain normal alignment is a fast and effective method. Some of the tools that can be used include spring-loaded tongs (Gardner-Wells), cones, and the University of Virginia. The load used depends on the dislocation or not. In a non-dislocated fracture, the weight is, generally, 3–5 kg, whereas in a dislocation a 4 kg increase every 30 min (up to a total of 25 kg) is used in a flexed neck position. The patient should be checked for neurologic status at any increase in load, and the traction load should be reduced as soon as possible in the event of deterioration in neurological status. In addition, halo traction can be used as an alternative means of skeletal traction.[3,5,7]

CONCLUSION

Treatment in the hospital includes all systems that may experience complications from SCI, starting from the respiratory, cardiovascular, urological, gastrointestinal, skin, to non-operative, and operative reduction measures.

Declaration of patient consent

Patient's consent not required as there are no patients in this study.

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Conflicts of interest

There are no conflicts of interest.

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