Chapter

Introductory Chapter: Spinal Cord Injury

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1. Introduction

The annual global incidence of traumatic spinal cord injury (SCI) was estimated by the Global Burden of Disease Study in 2016, and it resulted in as high as 0.93 million (0.78–1.16 million) per year, with an age-standardized incidence rate of 13 (11–16) per 100,000 population [1]. In the USA, the principal causes of SCI are represented by motor vehicle accidents (36–48%), violence (5–29%), falls (17–21%), and recreational activities (7–16%) [2]. The socioeconomic burden is extremely high due to the young age, the severity of acquired disabilities, and both direct and indirect health-related costs. In fact, the annual national cost in 2009 was as high as $1,7 billion [3], and for each patient ranged from $30,770 to $62,563 in 2016 [4]. The most significant cost derived from the severity of disability and complications developed during the hospitalization such as pressure ulcers and infections [5]. The SCI burden is extended also to the psychology of the younger patients, suddenly experiencing paraplegia or quadriplegia [6, 7]. It has been reported that people suffering from SCI are 2–5 times more likely to die prematurely compared to the healthy population [8, 9].

In SCI, the timing for intervention is crucial. Several studies have shown that early medical-surgical intervention could effectively improve functional outcomes. According to the Advanced Traumatic Life Support (ATLS) guidelines, any obstruction of upper airways should be restored while paying attention to neck and spine mobilization. The immobilization procedures should be fastidiously observed even in penetrating trauma without interfering with resuscitation efforts [10]. After immobilization, the patient should be quickly transferred to the closest trauma center hospital.

2. Clinical presentation

Clinical symptoms are SCI depend on the level of injury and include autonomy-related neurological dysfunctions such as cardiovascular disorders, sexual, bowel, and bladder dysfunction, sensory and motor deficit such as paresis, and spasticity [11].

The American spinal injury association developed a clinical classification (ASIA scale, see **Table 1**) for grading the severity of injury that now represents the international standard tool for evaluation [12, 13]. Unfortunately, epidemiology has

	American spinal injury association impairment scale
A	No motor or sensory function is preserved in the sacral segments S4–S5.
B	Sensory function preserved but not motor function is preserved below the neurological level and includes the sacral segments S4–S5.
C	Motor function is preserved below the neurological level, and more than half of key muscles below the neurological level have a muscle grade less than 3.
D	Motor function is preserved below the neurological level, and at least half of key muscles below the neurological level have a muscle grade of 3 or more.
E	Motor and sensory functions are normal.

Table 1.
ASIA scale [12].

confirmed that SCI often affects the cervical spine, likely due to the high mobility of the segment that may represent a poor factor in terms of clinical outcome.

3. Mechanism of SCI

The pathophysiology of SCI may be divided into phases: primary and secondary injury.

- **Primary injury** is defined as direct physical trauma to the spinal cord due to different mechanisms such as laceration, distraction, and transient or persistent compression [14]. The local damage of the spinal cord occurs during primary injuries that are irreversible.

Primary injury	• Laceration • Distraction • Transient compression • Persistent compression
Secondary injury	
Acute:	• Spinal shock • Vascular dysfunction • Membrane e ionic dysregulation • Neurotoxic transmission
Subacute:	• Free radical injury • Lipid peroxidation • Immune-associated neurotoxicity • Astrocytic glial scar formation
Chronic:	• Glial scar formation • Nogo receptors

Table 2.
Phases of SCI injury.

- **Secondary injury** consists of multiple cascades of biochemical events that determine craniocaudal damage extension and loss of functionality. Secondary injury is subdivided into acute, subacute, and chronic phases [15–17] (see **Table 2**). Principal actors of the acute phase are spinal shock, vascular disfunction neurotoxicity transmission, membrane, and ionic dysregulation. Those phenomes start immediately after the injury, disrupting the structural integrity of the CNS and activating the cascade events [18, 19]. Most of these phenomes overlap during the subacute phase.

During the subacute phase, the damage progressively extends to the surrounding districts, and new processes are determined by the production of free radicals such as free radical injury, lipid peroxidation, immune-associated neurotoxicity, astrocytic glia scar formation [15, 17]. The chronic phase is characterized by glial scar formation [20, 21] and activation of Nogo Receptor [22, 23].

4. Treatments in acute phase

4.1 Surgical

The Surgical Timing in Acute Spinal Cord Injury study [24] and the Observational European Multicenter study on the efficacy of acute surgical decompression after traumatic Spinal Cord Injury: the SCI-(POEM) study [25] had shown that early surgical decompression (<24 h) significatively improves the clinical outcome.

4.2 Medical

4.2.1 Corticosteroid-based therapy

The use of high-dose methylprednisolone is currently under discussion due to the risk related to high corticosteroids doses, while its clinical-functional advantages have been not confirmed yet. Historically, methylprednisolone has been administered at high doses for 48 hours after the National Acute Spinal Cord Injury Study (NASCIS) [26, 27]. It was also demonstrated that the clinical improvement could occur only if the treatment was started within 8 hours from trauma [26]. The AO spine, in 2017 [28, 29], suggests to use the NASCIS protocol for only 24 h (methylprednisolone: 30 mg/kg + 5,4 mg × 23 h), as reported by Bracken et al. [30].

4.2.2 High blood pressure

In order to supply the spinal cord, the AANS/CNS guideline suggests maintaining the mean arterial pressure ≥ 85–90 mm/hg in the 7 days after the injury.

5. Adult spinal cord injury without radiographic abnormalities (SCIWORA)

SCIWORA is a rare syndrome that results in objective signs of myelopathy after traumatic injuries without any radiological findings in TC or MRI imaging. This

syndrome usually affects children, while it is reported rarely in the adult population [31, 32]. The genesis of SCIWORA seems related to hyperextension forces, as cervical acceleration causing whiplash injuries in car accidents, or from a direct impact to the face, very similar to the diffuse axonal injury in the brain trauma [32, 33]. The treatment is usually conservative with early immobilization of the neck [34]. However, up to 16% of these patients suffer from relevant post-traumatic disorders.

6. Conclusion

Spinal cord injury represents a scenario of multidisciplinary interest in which the injury-to-treatment time represents the most relevant factor in determining the functional outcome. Functional disorders after SCI represent socioeconomic burdens, in terms of direct and indirect health-related costs. Therefore, there is a growing interest in both ameliorating the treatment strategies in the acute management of SCI and standardizing rehabilitation and long-term care protocols for these patients.

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