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Imaging of Spinal Cord Injury: Acute Cervical Spinal Cord Injury, Cervical Spondylotic Myelopathy, and Cord Herniation

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We review the pathophysiology and imaging findings of acute traumatic spinal cord injury (SCI), cervical spondylotic myelopathy, and briefly review the much less common cord herniation as a unique cause of myelopathy. Acute traumatic SCI is devastating to the patient and the costs to society are staggering. There are currently no "cures" for SCI and the only accepted pharmacologic treatment regimen for traumatic SCI is currently being questioned. Evaluation and prognostication of SCI is a demanding area with significant deficiencies, including lack of biomarkers. Accurate classification of SCI is heavily dependent on a good clinical examination, the results of which can vary substantially based upon the patient's condition or comorbidities and the skills of the examiner. Moreover, the full extent of a patients' neurologic injury may not become apparent for days after injury; by then, therapeutic response may be limited. Although magnetic resonance imaging (MRI) is the best imaging modality for the evaluation of spinal cord parenchyma, conventional MR techniques do not appear to differentiate edema from axonal injury. Recently, it is proposed that in addition to characterizing the anatomic extent of injury, metrics derived from conventional MRI and diffusion tensor imaging, in conjunction with the neurological examination, can serve as a reliable objective biomarker for determination of the extent of neurologic injury and early identification of patients who would benefit from treatment. Cervical spondylosis is a common disorder affecting predominantly the elderly with a potential to narrow the spinal canal and thereby impinge or compress upon the neural elements leading to cervical spondylotic myelopathy and radiculopathy. It is the commonest nontraumatic cause of spinal cord disorder in adults. Imaging plays an important role in grading the severity of spondylosis and detecting cord abnormalities suggesting myelopathy.
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Acute Cervical Spinal Cord Injury

Spinal cord injury (SCI) is a devastating, life-altering event. Approximately 12,000 new injuries occur annually in the United States,[1] and currently there are approximately 227,080-300,938 individuals living in the United States with the sequelae of SCI including permanent paralysis. Not surprisingly, the costs to society of SCI are staggering and in 1998 were estimated at $9.7 billion per year.[2] The lifetime direct costs of a high tetraplegic injured at age of 25 years can exceed $3 million.[1] Males are disproportionately affected with a 4:1 male-to-female ratio, and most of injuries occur between the age of 16 and 30 years. Mirroring the increasing age of the U.S. general population, the average age at injury has increased from 28.7 years of age in the mid-1970s to 39.5 years since 2005.

There are currently no "cures" for SCI and the only accepted pharmacologic treatment regimen for traumatic SCI is high dose methylprednisolone (MP), which has been reported to show efficacy in Phase II randomized trials.[3] Subsequently, MP administration for acute SCI has become widespread in the United States. Recently the efficacy of this treatment has been

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questioned and currently is the subject of ongoing debate.[4,5] Much of the debate has centered on whether the magnitude of reported improvement with MP is clinically important. The controversy regarding the utilization of MP highlights the critical need for new treatment strategies. To date, the treatment of acute SCI has been characterized, unfortunately, by the paucity of clinical trials. Although the efficacy and safety of MP remains controversial, there is general agreement that any pharmacologic measure should be employed in the first few hours after injury. Patient selection for a specific therapy can be problematic in the acute period because the classification system used to grade neurologic impairment is completely dependent upon the accuracy of the neurologic examination. The neurologic examination is accurate and reproducible in ideal conditions. However, the results can vary substantially based upon the level of cooperation, communication, and consciousness of the patient, associated patient comorbidities and the skills of the examiner. Moreover, the full extent of a patient's neurologic injury may not become apparent for days after injury. By then, late implementation of a drug based upon a delayed neurologic assessment is less likely to demonstrate a therapeutic response.

In that respect, it is proposed that in addition to characterizing the anatomic extent of injury, metrics derived from conventional magnetic resonance imaging (MRI), and diffusion tensor imaging (DTI), in conjunction with the neurological exam, can serve as a reliable objective biomarker for determination of the extent of neurologic injury and early identification of patients who would benefit from treatment.

Pathophysiology of SCI

Similar to acute traumatic brain injury, acute SCI can be divided into primary and secondary injury models. Compared with the brain, the mechanism of acute SCI is less well understood, and most of the research data currently available is derived from animal trials.[6] Although transection injuries of the spinal cord do occur in acute trauma, most of acute SCI in humans is caused by blunt trauma, usually in the setting of motor vehicle accidents.[7,8]

The primary insult to the cord is initiated by transient or fixed loss of integrity of the surrounding bony and ligamentous structures with resultant blunt impact on the spinal cord. In general, the amount of force transmitted to the cord determines the severity of the underlying cord injury.[9] The injury may range from transient neurologic deficits because of abnormal axonal firing to dense neurologic deficits due to axonal disruption.[7] Aside from preventive measures like lowering the speed limit and enforcing drunk-driving laws, primary SCI is immutable and current interventions are aimed at mitigating secondary spinal injury.

Secondary SCI is characterized by subsequent cellular dysfunction, necrosis, and death of initially intact neurons adjacent to the site of primary affect. *Several processes are triggered by the injured or dying neurons at the site of primary impact that spread to nearby normal axons and result in propagation of the initial injury*. Immediately following acute injury, spinal cord edema occurs, resulting in decreased perfusion pressure and ischemia related to microvascular perfusion abnormalities.[10] The injured axons also release glutamate in large amounts, a potent excitatory neurotransmitter.[11,12] Exposure of uninjured neurons to excessive amounts of glutamate is toxic, resulting in influx of calcium and sodium into the cells and initiating a number of deleterious processes including cell death in some cases.[11,12] Vulnerable intact neurons close to the site of initial SCI may undergo cell death in the ensuing hours or days via necrosis or apoptosis.[13] Apoptosis is controlled cell death that results in minimal inflammation, whereas necrosis is a disorderly process of cell death that causes significant inflammation.[10] Severe cord injury tends to result in more extensive necrosis.[7] Another important mechanism of secondary SCI is generation and propagation of free radicals. Free radicals are highly reactive molecules that interact with lipids and proteins in cell membranes to cause cellular dysfunction. Free radicals are especially abundant during the reperfusion phase of SCI, and several therapies are specifically targeted at halting the production of free radicals.[14] The processes in secondary spinal injury are complex and interdependent. *Optimal management of acute SCI in the future will likely be directed at inhibiting multiple sites in the secondary spinal injury cascade in hopes of achieving a synergistic therapeutic effect*.

Clinical Assessment of Acute SCI

Initial clinical presentation of patients with acute cervical SCI is the main factor determining triage, defining therapeutic options, and predicting prognosis. As such, the initial neurologic assessment should be accurate, consistent, and reproducible in defining the neurological deficits. In addition, an ideal neurologic assessment scale should have prognostic value in determining patient's potential for recovery. Numerous assessment scales have been employed to evaluate SCI patients, and can be divided into 2 general types. The first type focuses on the neurological deficits resulting from SCI and is examination specific. The International Standards for the Neurological Classification of SCI (ISNCSCI) is the most widely used and validated system, having undergone multiple revisions, most recently in 2011.[15] The second type of scale focuses on SCI patient's functional skills, including ability to care for oneself, perform personal hygiene, ambulate or transfer. These scales aim to determine patient's ability or inability to functionor live independently. In general, the first type of scale is used to acutely assess patients with SCI, while both scales are used to define the chronically injured patient. Scales for functional outcomes include the Barthel Index, the modified Barthel Index, the Functional Independence Measure (FIM), the Quadriplegic Index of Function, the Spinal Cord Independence Measure (SCIM), the Walking Index for SCI, the SCI Functional Ambulation Inventory, and more recently proposed SCI Computer Adaptive Test.[16]

The central aspect of the ISNCSCI is classification of SCI patients into American Spinal Injury Association (ASIA) Impairment Scale—the AIS, which is a 5-point ordinal scale that classifies individual's injury from A through E (Table). The neurologic level of injury (NLI) refers to the most caudal