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Management of acute spinal cord injuries: Emergency view

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***Abstract*---Background**: Acute spinal cord injuries (SCIs) are devastating events often leading to irreversible neurological damage and significant long-term disabilities. SCIs affect young, otherwise healthy individuals, contributing to a considerable economic and quality-adjusted life years (QALY) burden. **Aim**: This review aims to provide an overview of current acute management strategies for SCI and to explore emerging therapeutic approaches that may mitigate injury progression and improve outcomes. **Methods**: The study reviewed existing literature on SCI management, including established protocols and experimental therapies. A comprehensive analysis of surgical and medical treatments, such as early decompression, hemodynamic management, and novel interventions targeting ischemia, inflammation, and cytotoxicity, was conducted. **Results**: Early surgical decompression, within 24 hours of injury, has been shown to enhance neurological recovery, reduce hospital stays, and lower complication rates. Hemodynamic management, aiming to maintain a mean arterial pressure of 85–90 mmHg, was crucial in improving functional outcomes. Experimental treatments such as cerebrospinal fluid diversion, spinal cooling, and minocycline demonstrated promising results, though further clinical trials are necessary to establish efficacy. **Conclusion**: While significant advances have been made in the surgical management of SCI, challenges remain in optimizing medical treatments. Novel therapies targeting ischemia, inflammation, and cytotoxicity show potential, but require more robust clinical evidence. Early intervention remains critical to improving outcomes for patients with acute SCI.

***Keywords*---**Spinal cord injury, acute management, early decompression, neuroprotection, ischemia, inflammation, experimental therapies.

Introduction

Spinal cord injuries (SCIs) represent profoundly traumatic events in patients' lives, frequently leading to significant and/or irreversible neurological impairments. In the United States, approximately half a million individuals are living with permanent disabilities resulting from traumatic SCI, with 12,000–15,000 new cases reported annually [1,2]. The prevalence of SCI differs across nations, with most developed countries documenting rates between 20 and 50 per million [3,4]. These injuries often result in a range of immediate consequences and long-term complications, such as loss of motor and sensory functions,

impaired autonomic regulation, and elevated risks of medical issues and mortality [5].

Since traumatic SCI disproportionately affects young and otherwise healthy individuals, the impact on quality-adjusted life years and economic burden can be substantial [6]. Although advancements have been made in understanding the pathogenesis and improving acute management of SCI, there is still a pressing need for further exploration of novel molecular targets and therapeutic interventions during the acute injury phase, aiming to optimize recovery and mitigate long-term effects. While extensive research has been conducted on the timing of surgical interventions for decompression and stabilization of acute SCI [7], the evidence supporting most medical treatments for acute neurological injury remains limited. This review addresses the current established management protocols and explores emerging therapeutic approaches for acute SCI. Additionally, we provide an updated review of experimental therapies, bridging the gap from laboratory research to clinical application.

Mechanism of Injury in Spinal Cord Injury:

Spinal cord injury (SCI) may result from high-energy trauma, such as motor vehicle accidents, diving, or sports-related incidents, or from lower-impact trauma that often occurs in the context of pre-existing degenerative or congenital spinal spondylosis. The cervical spinal cord is the most frequently affected region due to its reduced bony and muscular protection compared to the thoracic and lumbar segments [8]. SCI damage is typically categorized into primary and secondary processes. Primary injury involves the initial trauma, including compression, penetration, or strain on neural or vascular structures. This leads to perfusion disruption, which can cause local hypoxia, exacerbated by systemic vasogenic shock. Hypoxic cell death triggers a complex, progressive secondary injury process that occurs over hours to weeks. Ischemia induced by the primary injury promotes the release of vasoactive proteins and cytokines, which cause edema, inflammation, and further cellular damage. Accumulation of reactive oxygen species (ROS), oxidative stress, lactic acidosis, fibrin and platelet deposition, and toxic neurotransmitter release exacerbate axonal demyelination and contribute to progressive tissue degeneration and scarring.

Clinical Presentation:

The initial management of patients suspected of having acute traumatic SCI follows established trauma protocols that prioritize stabilizing the airway, breathing, and circulation. Immobilization is crucial to prevent further damage in patients with suspected SCI, which may be indicated by polytrauma, spinal pain in the neck or back, dysesthesia, loss of consciousness, or a mechanism of injury consistent with SCI [9-11]. Patients may present with either a complete injury, characterized by the absence of motor or sensory function in the anal and perineal regions (the lowest sacral segments S4–S5), or an incomplete injury, with varying degrees of motor and sensory function below the level of injury [12]. The American Spinal Injury Association (ASIA) scoring system stratifies injury severity from complete injury (ASIA-A) to normal neurological function (ASIA-E), facilitating standardized assessment and treatment [2]. Additionally, patients may

exhibit spinal shock, a temporary physiological depression of spinal cord function following cell damage, which is monitored by the progressive return of reflexes, including the bulbocavernosus, anal cutaneous, and plantar reflexes [12]. Spinal shock, along with medication effects, can obscure the accuracy of ASIA scoring within the first 48–72 hours post-injury [13]. This phenomenon differs from neurogenic shock, which involves autonomic dysfunction due to injury at spinal levels C1–T6 or associated sympathetic ganglia, and is marked by persistent hypotension, bradycardia, and hypothermia [12].

Established Acute Management Strategies:

Initial evaluation of traumatic SCI patients requires computed tomography (CT) imaging of the entire spine [14]. ASIA motor scores and the neurological level of injury are key prognostic indicators of patient outcomes [15]. Magnetic resonance imaging (MRI) is valuable for surgical decision-making and prognostication, though it should not delay surgery in cases of neurological decline [12,15]. Efforts are underway to standardize MRI findings of varying severity in acute SCI, such as the BASIC score [16]. Surgical candidacy is determined by assessing neurological deterioration, instability, and progressive spinal cord compression. Direct spinal cord compression is a common cause of SCI and can worsen after the initial injury, necessitating emergent surgical decompression. A meta-analysis of 16 studies involving nearly 4,000 patients revealed that early spinal surgery (within 24 hours of injury) is associated with greater neurological recovery, shorter hospital stays, reduced costs, and fewer complications compared to surgery performed after 24 hours [17]. A recent multicenter clinical trial comparing early surgery (≤24 hours post-injury) with delayed surgery (≥2 weeks post-injury) showed improved motor recovery at 2-week, 3-month, and 6-month follow-ups with early surgical intervention, indicating accelerated recovery [18]. These findings, supported by several large studies, emphasize the importance of early decompression to maximize motor recovery after SCI [19-21].

Medical management plays a crucial role in optimizing outcomes for SCI patients [12,22]. Maintaining hemodynamic stability, with a target mean arterial pressure (MAP) of 85–90 mmHg for 5–7 days, is recommended to minimize ischemic damage and improve functional recovery [23]. Preventing complications such as pneumonia, bowel obstruction, and dysautonomic uropathy—resulting from loss of voluntary and autonomic control—is also a key focus of care [12,14]. Ongoing clinical trials are investigating interventions such as low versus high tidal volume mechanical ventilation in patients requiring intubation after acute SCI (NCT04912583) and the use of early epidural and sacral nerve stimulation to improve bladder function (NCT03083366) [24,25]. The use of methylprednisolone (MPSS) in SCI treatment has been a subject of debate. While early results from the North American Spinal Cord Injury Study (NASCIS) suggested potential benefits, more recent studies have failed to confirm these findings, and there is now substantial evidence linking high-dose steroid use with increased risk of complications, including infection [26-28]. Animal studies suggest that high-dose steroids may exacerbate tissue swelling and edema by increasing plasma component extravasation after SCI [29]. Consequently, routine high-dose steroid administration is no longer recommended; however, a short course may be considered in young, otherwise healthy individuals presenting within 8 hours of

injury, based on expert consensus [27,28,30]. Early rehabilitation, nutritional support, and mental health interventions are also essential to improving functionality and quality of life following SCI [31-33]. These therapies are currently under investigation in trials assessing the impact of interval versus continuous aerobic training on autonomic dysreflexia in the acute phase of SCI (NCT05061160).

Experimental Acute Management Strategies:

Innovative treatments for acute spinal cord injury (SCI) are often classified according to the biological mechanisms they aim to target, mitigate, or reverse. These mechanisms, commonly discussed in existing literature, are categorized based on the prevailing understanding of SCI pathophysiology.

1. **Mitigating Ischemia**: In the initial phases of SCI, damage to local blood vessels can cause ischemic hypoxia in the spinal grey matter. This hypoxia affects metabolically active regions, resulting in neuronal dysfunction, demyelination, and triggering of cell death through apoptosis and necrosis [34]. If ischemia remains unaddressed, it may lead to further damage due to cellular swelling and the early inflammatory response mediated by macrophages, typically lasting between 3–24 hours post-injury [34]. Additionally, insufficient blood flow impairs the vascular and regenerative astrocytic environment, limiting its capacity to repair axonal and neuronal damage. Maintaining appropriate systemic perfusion and blood flow within the spinal vasculature is vital for minimizing the pathological impact of SCI.
   Neurogenic shock can exacerbate ischemia, and treating hypotension with fluid resuscitation, transfusion, or traditional vasopressors is often challenging due to local disruptions in capillary auto-regulation. Clinical trials have tested dopamine and midodrine with mixed outcomes, but no high-level evidence favors one vasopressor over another [35]. Dopamine was found to maintain mean arterial pressure (MAP) comparably to norepinephrine, but it increased intrathecal pressure (ITP) and reduced spinal cord perfusion pressure (SCPP), while also being linked to more vasopressor-related complications [22]. Some case reports and small trials of midodrine have shown success in stabilizing blood pressure and alleviating orthostatic symptoms without serious adverse effects, though long-term outcomes were not explored [36]. However, in acute SCI, it is essential to avoid hypertension to reduce the risk of hyperemia and hemorrhage.
   Cerebrospinal fluid (CSF) diversion may help alleviate ischemia caused by venous leakage and edema post-SCI by reducing ITP. This approach is derived from cranial trauma practices, such as ventriculostomy, and vascular surgery's use of lumbar drains to improve spinal cord perfusion during aortic dissection [37]. Preclinical studies using a pig SCI model demonstrated substantial and sustained improvement in blood flow and SCPP with CSF drainage [38]. Early human studies involving CSF drainage within 48 hours of injury did not report significant side effects but also did not show clear neurological benefits, possibly due to low statistical power [39]. Later studies indicated more positive outcomes, although a consistent effect has yet to be confirmed [40]. Patch duraplasty, performed during initial surgical decompression, is another method for reducing ITP. A study comparing laminectomy with patch duraplasty to laminectomy alone in acute SCI

patients showed that the duraplasty group had lower ITP, improved spinal perfusion pressure, and better ASIA impairment scores, though the small sample size hindered statistical significance in clinical outcomes [42]. Conducting large-scale, well-powered clinical trials in acute SCI remains a significant challenge. Hyperbaric oxygen therapy (HBO), if administered early, may counteract spinal cord ischemia and hypoxia, potentially reducing apoptosis, oxidative damage, inflammation, and edema, while promoting angiogenesis and autophagy. Clinical trials that began therapy within 9–20 hours post-injury reported varying levels of neurological improvement [43]. However, technical parameters, such as the onset, duration, frequency, and pressure, as well as the benefits for diverse SCI presentations, still require further investigation. Ongoing clinical studies in China (NCT03112941) and Austria (NCT03101982) aim to address these gaps.

2. **Reducing Inflammation-Induced Secondary Damage**: The inflammatory response, which occurs within hours to days after the initial SCI, is a key contributor to worsened neurological outcomes [34]. This secondary damage exacerbates the injury by influencing compression, ischemia, and scar formation through both cellular and systemic factors [44]. The synthesis of proinflammatory prostaglandins, cytokines, calcium-dependent nitric oxide, opioid peptides, and necroptotic factors has been shown to amplify inflammatory damage during the acute phase. To address this, several common pharmacological agents have been effective in preclinical studies for maintaining blood flow and reducing cellular damage [34]. Cyclooxygenase inhibitors like ibuprofen and meclofenamate, as well as nonselective opioid antagonists like naloxone, have improved blood flow and functional outcomes in animal SCI models [45-48]. However, few studies have evaluated these agents in human SCI. For example, NASCIS II in 1990 reported no neurological benefit with naloxone compared to placebo within the first 24 hours of SCI, though recommendations on naloxone use are under reconsideration [26]. A phase-I trial investigating ibuprofen in acute SCI is currently underway (NCT02096913).

   Minocycline, a tetracycline antibiotic, has demonstrated neuroprotective effects against secondary SCI progression by inhibiting the production of proinflammatory cytokines, such as IL-1beta and TNF-alpha, and proapoptotic enzymes like caspase-1/-3, while reducing inducible nitric oxide synthase [49,50]. In preclinical models, minocycline improved tissue preservation and motor recovery within 3–4 weeks of SCI [51,52]. A phase-II trial in patients with incomplete cervical SCI reported a 14-point improvement in ASIA motor scores following minocycline treatment compared to placebo [53]. Spinal cooling, a non-pharmacological intervention, has also shown promise in reducing biochemical secondary damage. In one study, combining surgical decompression, steroids, and regional hypothermia resulted in functional improvements for 13 of 20 SCI patients initially classified as ASIA-A [54]. However, the absence of randomized control arms and the limited availability of data from similar reports restricts the generalizability of spinal cooling as an acute SCI treatment. Nonetheless, the promising initial outcomes and lack of adverse effects suggest that further investigation is warranted [12].

3. **Modulating the Cytotoxic Response**: The acute cytotoxic response following SCI, marked by ion dysregulation and the release of excessive glutamate,

contributes to excitotoxicity and oxidative stress. These processes promote reactive oxygen species (ROS) formation and lipid peroxidation, increasing the risk of neuronal death. Several antioxidants, including cyclosporin A, vitamins C, D, and E, selenium, and lithium, have shown potential in preclinical models to preserve cell viability [34]. A clinical trial in China involving lithium is the first human study to assess its use as an adjunct therapy for SCI (NCT01471613).

Unregulated activation of voltage-gated sodium channels has been implicated in the cytotoxic cascade following SCI. Riluzole, a benzothiazole that inhibits sodium channel-mediated glutamate release, has been found to reduce neuronal loss and improve sensorimotor function in animal SCI studies [55]. A phase-I trial involving 36 patients reported enhanced motor recovery in cervical SCI patients administered Riluzole, although the improvement did not persist beyond six months [55]. Additionally, concerns over liver enzyme elevations warrant further investigation into the drug's safety profile. A larger multicenter trial, the Riluzole in Acute Spinal Cord Injury Study, is currently being conducted by the AO Spine North America Research Network to assess its therapeutic potential in SCI.

Blocking α-amino-3-hydroxy-5-methyl-4-isoxazolepropionic acid (AMPA) and N-methyl-D-aspartate (NMDA) receptors has been explored as a strategy to mitigate glutamate-induced excitotoxicity in the hyperacute phase of SCI. Gacyclidine, an NMDA antagonist, was tested in a phase-II trial involving 280 SCI patients but failed to show significant improvements in ASIA scores compared to placebo [56]. Despite this, other NMDA antagonists, such as magnesium, have demonstrated promise in preclinical studies by reducing excitotoxicity and inflammation. However, clinical trials examining these agents for SCI are still pending. Calcium-channel blockers (CCBs) may help regulate calcium influx and improve vascular tone following SCI. Nimodipine, in particular, was evaluated in a French clinical trial of 106 SCI patients, but no significant neurological benefit was observed [27]. Although potassium channel antagonists like fampridine may offer therapeutic benefits through enhanced axonal conduction, their efficacy is currently being tested in phase-III trials [34].

4. **Modulating the Immune Response:** Immunomodulative therapies are being explored for treating acute spinal cord injury (SCI), with a focus on controlling the inflammatory response triggered by peripheral immune cells such as macrophages, neutrophils, and T cells. This inflammation can worsen spinal cord damage by promoting cavitary lesion growth. Inflammatory cytokines like IL-1beta, TNF-alpha, and IL-6 are released post-injury, followed by neuroprotective IL-10. Microglia, the central nervous system's resident immune cells, play a key role in regulating this response. A phase-I/II trial in China is testing TNF-alpha monoclonal antibodies for acute SCI treatment, expected to involve 90 participants with results anticipated in late 2023 (NCT04988425). Additionally, granulocyte-colony stimulating factor (G-CSF), which reduces TNF-alpha and IL-1beta at SCI sites, has shown potential in enhancing neurogenesis and improving motor scores in clinical trials. A phase-III trial in Japan has been completed, with results pending.
5. **Nerve Regeneration Techniques:** Nerve regeneration after SCI is a critical focus in research, with several experimental approaches under investigation:
   1) **Stem Cell Implantation**

Stem cell therapies aim to promote neural regeneration and functional recovery, using cells such as autologous bone marrow mononuclear cells (BM-MSCs), neural progenitor cells (NPCs), and olfactory ensheathing cells (OECs). BM-MSCs show some promise in matrix repair and angiogenesis, though their efficacy varies. Clinical trials have reported mixed results, with some patients experiencing sensory or motor improvements. NPCs, which promote axonal regeneration and remyelination, have also shown potential, but challenges remain, including the high cost of research. ESCs and iPSCs, while promising, are associated with risks like genomic instability and ethical concerns.

2) **Growth Factor Conditioning**
   Growth factors such as acidic fibroblast growth factor (aFGF) and hepatocyte growth factor (HGF) are being studied for their potential to promote neuronal growth and reduce scarring in SCI. Human trials of aFGF have shown improvements in motor and sensory function, and recombinant aFGF is currently in a phase-III trial (NCT03229031). HGF has demonstrated success in preclinical studies, with ongoing trials assessing its therapeutic benefit in humans.
3) **Inhibiting Neural Growth Inhibitors**:
   Cethrin (VX-210) inhibits the Rho pathway, involved in cytoskeletal regulation, promoting axonal outgrowth in SCI models. Elezanumab, a monoclonal antibody targeting inhibitors of axonal growth, is under investigation in a phase-II trial (NCT04295538).
4) **Tissue Scaffolding**:
   Biomaterials like synthetic or natural polymers are used to create tissue scaffolds that support neural regeneration. The INSPIRE trial, focused on poly(lactic-co-glycolic acid)-b-poly(L-lysine) scaffolds for thoracic ASIA-A patients, will conclude in 2024 (NCT02138110). Other trials are investigating the use of collagen scaffolds combined with stem cell therapy. Ganglioside-1 (GM-1) has also been explored for SCI treatment, but meta-analyses suggest its therapeutic benefit remains inconclusive.

Future Perspectives:

Numerous approaches are currently being explored in animal models, which may soon progress to clinical trials aimed at demonstrating functional improvements in spinal cord injury (SCI). Necroptosis, a caspase-independent mechanism of programmed cell death, emerges following cellular damage. Unlike apoptosis, necroptosis leads to the leakage of cellular contents into the extracellular space, which induces a proinflammatory state by activating both the innate and adaptive immune systems [91]. This process is mediated by receptor-interacting serine/threonine kinase 1 and 3, and studies suggest it could be a predominant form of cell death following traumatic SCI [91]. Inhibiting this pathway may reduce secondary damage and mitigate cell death [92]. Autologous omental transplantation has also been suggested as a method to introduce blood- and lymphatic-rich tissue into the injured spinal cord [93]. Although small human trials in the 1990s indicated the continued viability of omental grafts in patients with chronic SCI, these studies did not demonstrate any significant functional improvements, leading to a decline in interest for its use in SCI treatment [94]. However, omental transplant therapy has seen advancements in other medical

contexts, such as neovascularization and neurotrophic effects following its placement on the brain surface in cases of epilepsy or ischemic brain injury [95]. Renewed interest in the potential for omental angiogenesis to treat SCI has emerged, with recent rat trials showing enhanced neural preservation, a reduction in injury cavities, and improved neovascularization at the injury site [93].

A recent addition to regenerative cellular therapies is the use of peripheral nerve-derived stem cells (PNSCs). Spheroid formations of PNSCs have demonstrated significant therapeutic potential, primarily through the release of neurotrophic factors and extracellular matrix expression, which have led to considerable functional recovery, neuronal regeneration, and a reduction in neuropathic pain in animal models [96]. Estrogen-based therapies are also gaining attention. Estradiol has been shown to provide neuroprotection in the central nervous system (CNS) in animal models of SCI, traumatic brain injury, and ischemic brain injury [97]. Bazedoxifine, a third-generation selective estrogen receptor modulator, was studied in rats, where it was found to reduce inflammatory responses and promote remyelination by inhibiting the mitogen-activated protein kinase/nuclear factor-kappa B (NF-κB) pathway, as well as promoting oligodendrocyte precursor cell differentiation and proliferation [98]. Although estrogen therapy has complex implications due to its role in maintaining and altering normal physiological functions, it holds promise as a potential treatment avenue for SCI.

Entinostat, a class I histone deacetylase (HDAC) inhibitor targeting HDAC1 and HDAC3, has shown potential in reducing SCI-induced inflammation by inhibiting NF-κB-mediated microglial activation [99]. Treatment of a mouse model of SCI with Entinostat resulted in improved grip strength, enhanced locomotion scores, and reduced spinal edema, cell death, and NLRP3 inflammasome activation [99]. Another potential strategy to counteract SCI-induced inflammation is through IgM's regulatory role against IgG-mediated autoimmune responses. A study revealed that IgM-knockout mice with induced C6–7 SCI exhibited significantly greater impairment in neurobehavioral recovery compared to their wild-type counterparts. These mice displayed worsened coordination, decreased fore- and hind-limb movement, and a larger lesion size, with reduced white matter preservation and increased deposition of complement-fixing IgG antibodies in the spinal cord [100]. These innovative methods are expected to undergo further investigation as the scientific community continues to expand the treatment options for this multifaceted condition.

Conclusion

The management of acute spinal cord injuries (SCIs) has evolved significantly over recent decades, with advancements in both surgical and medical approaches contributing to improved patient outcomes. Early surgical intervention, particularly within the first 24 hours post-injury, is now widely accepted as a key factor in promoting neurological recovery, shortening hospital stays, and minimizing complications. However, medical management, though crucial, remains more challenging to optimize. Hemodynamic stabilization, ensuring adequate mean arterial pressure, has been shown to prevent further ischemic damage and improve long-term functional recovery, but the search for effective pharmacological interventions continues. The role of experimental therapies in

acute SCI management offers promising avenues for future treatments. Approaches such as cerebrospinal fluid (CSF) diversion, spinal cooling, and the use of neuroprotective agents like minocycline have shown potential in early clinical studies. These interventions target key mechanisms of secondary injury, such as inflammation, ischemia, and oxidative stress, which exacerbate initial trauma. However, while preclinical findings are encouraging, larger-scale clinical trials are needed to confirm their efficacy and safety in human populations. One of the critical challenges in SCI management lies in addressing the cytotoxic cascade that follows the initial injury, leading to excitotoxicity and further neuronal damage. Experimental treatments targeting these pathways, including NMDA receptor antagonists and calcium-channel blockers, have shown limited success, underscoring the need for continued research into novel neuroprotective strategies. In conclusion, while significant strides have been made in understanding and managing acute SCIs, there remains a considerable gap between experimental therapies and their clinical application. Early intervention, particularly in terms of surgical decompression and hemodynamic management, continues to be the cornerstone of SCI treatment. However, ongoing research into neuroprotective and anti-inflammatory treatments holds the potential to further improve outcomes for patients suffering from these devastating injuries. Collaboration between clinical researchers and neuroscientists will be essential in translating promising experimental therapies into standard practice, ultimately leading to enhanced recovery and quality of life for SCI patients.

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إدارة إصابات الحبل الشوكي الحادة - نظرة طبية طارئة

الملخص:

الخلفية: تُعد إصابات الحبل الشوكي الحادة (SCI) أحداثاً مدمرة تؤدي غالباً إلى تلف عصبي لا يمكن عكسه وإعاقات طويلة الأمد. تؤثر إصابات الحبل الشوكي على الأفراد الشباب الأصحاء، مما يساهم في عبء اقتصادي كبير وتراجع في سنوات الحياة المعدلة حسب الجودة .(QALY)

الهدف: تهدف هذه المراجعة إلى تقديم نظرة عامة على استراتيجيات الإدارة الحادة الحالية لإصابات الحبل الشوكي واستكشاف النهج العلاجية الناشئة التي قد تحد من تطور الإصابة وتحسن النتائج.

الطرق: استعرضت الدراسة الأدبيات الحالية حول إدارة إصابات الحبل الشوكي، بما في ذلك البروتوكولات المعتمدة والعلاجات التجريبية. تم إجراء تحليل شامل للعلاجات الجراحية والطبية، مثل إزالة الضغط المبكر، وإدارة الديناميكا الدموية، والتدخلات الجديدة التي تستهدف نقص التروية والالتهابات والسمية الخلوية.

النتائج: أثبتت إزالة الضغط الجراحي المبكر، خلال 24 ساعة من الإصابة، أنها تعزز التعافي العصبي وتقلل من مدة الإقامة في المستشفى وتخفض معدلات المضاعفات. كانت إدارة الديناميكا الدموية، التي تهدف إلى الحفاظ على ضغط شرياني متوسط يتراوح بين 85-90 ملم زئبقي، حاسمة في تحسين النتائج الوظيفية. أظهرت العلاجات التجريبية مثل تحويل السائل النخاعي وتبريد الحبل الشوكي والمينوسيكلين نتائج واعدة، لكن لا تزال هناك حاجة إلى تجارب سريرية إضافية لتحديد الفعالية .

الخلاصة: رغم التقدم الكبير في الإدارة الجراحية لإصابات الحبل الشوكي، لا تزال التحديات قائمة في تحسين العلاجات الطبية. تظهر العلاجات الجديدة التي تستهدف نقص التروية والالتهابات والسمية الخلوية إمكانات واعدة، ولكنها تتطلب أدلة سريرية أكثر قوة. يظل التدخل المبكر حاسماً في تحسين نتائج المرضى الذين يعانون من إصابات الحبل الشوكي الحادة .

الكلمات المفتاحية: إصابة الحبل الشوكي، الإدارة الحادة، إزالة الضغط المبكر، حماية الأعصاب، نقص التروية، الالتهابات، العلاجات التجريبية.