Chinese Journal of Traumatology 26 (2023) 8–13

Contents lists available at ScienceDirect

Chinese Journal of Traumatology

journal homepage: http://www.elsevier.com/locate/CJTEE

Review

Clinical characteristics and treatment of spinal cord injury in children and adolescents

Jin-Zuo Wang [a], Ming Yang [a], Meng Meng [a], Zhong-Hai Li [a, b, *]

[a] Department of Orthopedics, First Affiliated Hospital of Dalian Medical University, Dalian, 116000, Liaoning Province, China
[b] Key Laboratory of Molecular Mechanism for Repair and Remodeling of Orthopedic Diseases, Dalian, 116000, Liaoning Province, China

ARTICLE INFO

Article history:
Received 30 November 2021
Received in revised form
24 February 2022
Accepted 29 March 2022
Available online 16 April 2022

Keywords:
Spinal cord injury
Spinal cord injury with no radiographic abnormality
Children and adolescents
Treatment

ABSTRACT

Pediatric and adult spinal cord injuries (SCI) are distinct entities. Children and adolescents with SCI must suffer from lifelong disabilities, which is a heavy burden on patients, their families and the society. There are differences in Chinese and foreign literature reports on the incidence, injury mechanism and prognosis of SCI in children and adolescents. In addition to traumatic injuries such as car accidents and falls, the proportion of sports injuries is increasing. The most common sports injury is the backbend during dance practice. Compared with adults, children and adolescents are considered to have a greater potential for neurological improvement. The pathogenesis and treatment of pediatric SCI remains unclear. The mainstream view is that the mechanism of nerve damage in pediatric SCI include flexion, hyperextension, longitudinal distraction and ischemia. We also discuss the advantages and disadvantages of drugs such as methylprednisolone in the treatment of pediatric SCI and the indications and timing of surgery. In addition, the complications of pediatric SCI are also worthy of attention. New imaging techniques such as diffusion tensor imaging and diffusion tensor tractography may be used for diagnosis and assessment of prognosis. This article reviews the epidemiology, pathogenesis, imaging, clinical characteristics, treatment and complications of SCI in children and adolescents. Although current treatment cannot completely restore neurological function, patient quality of life can be enhanced. Continued developments and advances in the research of SCI may eventually provide a cure for children and adolescents with this kind of injury.

Introduction

Spinal cord injury (SCI) is a disabling and irreversible condition. SCI in children and adolescents is rare and should be treated differently due to differences in anatomy compared to adults.[1] The patterns of SCI also differ among adult, children and adolescents. Unfortunately, the treatment of SCI does not fully restore neurological function.

Regardless of patient age, SCI is divided into two stages: primary and secondary.[2] The initial mechanism is considered primary lesion, and the following biochemical and cellular processes is considered secondary lesion. Traction and compression forces from the initial mechanical trauma can cause vertebral fractures and dislocations. The resulting bone fragments and disrupted intervertebral discs and ligaments can then compress the spinal cord and nerve roots, causing damages to the central and peripheral nervous system, e.g. injuring blood vessels, and disrupting axons and broken neuron cell membranes. Spinal cord hemorrhage, necrosis and edema occur in the first hours following trauma.[3] Within minutes of injury, the spinal cord swells to fill the spinal canal in the injured segment. Spinal cord ischemia occurs when spinal cord swelling exceeds venous blood pressure, triggering a chain of biochemical reactions that lead to cell death. In addition, injury-induced neural membrane disruption produces toxic chemicals and causes electrolyte disturbances, leading to secondary injury cascades that exacerbate the initial injury by injuring or killing adjacent cells. Glutamate released from injured spinal neurons and astrocytes causes excitotoxicity,[4] which leads to secondary injury through calcium ion overload and free radical production. Current treatments mainly target at these primary and secondary injuries. This review aims to provide clinical guidance for the diagnosis and treatment of children and adolescents with SCI.

* Corresponding author. Department of Orthopedics, First Affiliated Hospital of Dalian Medical University, Dalian, 116000, Liaoning Province, China.
E-mail address: lizhonghai@dmu.edu.cn (Z.-H. Li).
Peer review under responsibility of Chinese Medical Association.

https://doi.org/10.1016/j.cjtee.2022.04.007

Epidemiology

The incidence of SCI in children and adolescents is generally low, but varies by country and geographic region, ranging from 2.9 to 27 per million in European countries.[5,6] In the United States, 3%–5% of SCIs occur in individuals under the age of 15, and approximately 20% occurs under the age of 20.[7]

In general, the incidence of SCI is roughly equal in male and female aged 5 years and younger, but in older age groups, SCI is more common in male.[7] This may be related to risky behavior in male children and adolescents. However, a survey conducted in Beijing revealed that 71% of pediatric SCI patients are female and the main cause of injury was performing backbends during dance practice.[8]

Overall, the most common site of SCI in children is the cervical spine (87.19%), followed by the thoracic spine (9.61%) and lumbar spine (1.48%).[9] Sports are the most common cause of injury (39.83%), followed by falls (24.18%) and motor vehicle-related injuries (23.18%). In contrast, thoracic injuries are most common in Chinese children and adolescents (77%), followed by cervical (10%), multiple levels (5%) and lumbar (4%).[8] And in China, the main causes of injury are sports (41%), falls (27%), traffic accidents (10%) and violence (8%).[8] Most sports-associated injuries are caused by performing backbends during dance practice.[8]

Injury mechanism

Approximately 55% – 65% of children and adolescents experiencing SCI have no evidence of spinal fracture or instability on plain radiography or CT,[4] which is called spinal cord injury without radiographic abnormality (SCIWORA) and first described by Pang et al., in 1982.[1] SCIWORA is common in children and adolescents, accounting for 74.1% of all pediatric SCIs.[8] The incidence of SCIWORA decreases with age in children and seldom occurs in adulthood. Pang et al.[1] considered that flexion, hyperextension, longitudinal distraction and ischemia were the mechanisms of neural damage in SCIWORA. With the advent of MRI, diagnosis of SCIWORA based on X-ray or CT appears to be obsolete. MRI can show the significant injury of spinal cord, spinal soft tissue components (ligament, dural sac or muscle) or vertebral endplate. Freigang et al.[10] considered that "real SCIWORA" should be redefined as the absence of any spinal abnormality.

The spine in children is more elastic than adults. The facet joints in children are shallow and horizontal, allowing a certain degree of sliding, and the ligaments and joint capsules are more flexible. However, the neck muscles of children are weak, the vertebral body is anterior wedge-shaped, and the spinous process, transverse process and uncinate process are less developed. The intervertebral disc is composed of nucleus pulposus and annulus fibrosus, and the nucleus pulposus is rich in water content, which allows longitudinal expansion when the spine is pulled. Because of this, SCIWORA often occurs in children and adolescents due to the high flexibility of the spine. Still, even though the spine of an infant can stretch by 5 cm, the infant spinal cord ruptures after only 5 – 6 mm of traction.[4]

Before the age of 8, the head is larger and heavier than the rest of the body. When the head moves, the fulcrum of movement is in the upper cervical spine and the maximum movement is at the $C_{2/3}$ segment. With age, the weight and size of the head decreases relative to the rest of the body and the fulcrum moves down to $C_{5/6}$ segment, which continues into adulthood. This explains the high incidence of upper cervical spine injury in younger children, while older children and adults usually experience subaxial cervical spine injury.[11,12] Temporary dislocation occurs when the child's spine overstretched and flexed, and then recovers naturally due to spinal elasticity; however, SCI can still occur.

SCIWORA is less common in the thoracic spine injury. Most thoracic SCIs are caused by car accidents that involves a direct collision and belt-related injury.[13] However, thoracic SCIs caused by performing backbends during dance practice is frequently reported in China. Ren et al.[14] hypothesized that the mechanism of primary SCI in these patients is longitudinal distraction of the thoracic spine, which results in spinal cord distraction and injury to nerve cells, axons and small vessels. In China, dance learning is very common among girls, which explains the higher incidence of SCI among Chinese girls. Surfer myelopathy is another SCIWORA syndrome that can occur in novice surfers and is caused by repeated hyperextension of the spine, similar to backbends. The injury mechanism is not well-known. Previous studies show that it may be related to arterial insufficiency, spinal cord infarction, avulsion of perforation vessels, fibrocartilaginous embolization.[15–18] In 2021, Wang et al.[19] reviewed 31 children diagnosed with surfer myelopathy and concluded that it is caused by spinal venous hypertension.

Clinical characteristics and imaging

Children and adolescents with SCI often have delayed neurological dysfunction. Among children and adolescents with SCIWORA, 22% – 54% of cases experienced symptoms between 30 min and 4 days after injury.[1,20] Common symptoms include transient clumsiness, paresthesias, total paralysis and lower back or leg pain. Younger patients may have difficulty in expressing symptoms of nerve injury and cooperating with the sensorimotor examination, and some children may even ignore transient paresthesias after injury. Thus diagnosis in the early stage of this injury may be difficult.

After SCI, the spinal cord should be evaluated, and its neurological status is typically assessed using the International Standards for Neurological Classification of SCI (ISNCSCI). Although the ISNCSCI assessment validated for adult SCI patients may not be accurate in young children, imaging assessment should be performed.

MRI is the most efficient way to assess the spinal cord as the gold standard for diagnosis of SCI.[21] Acute SCI manifests as single level edema, hematoma, multilevel edema and a mix of hemorrhage and edema. T1-weighted imaging provides data regarding cord morphology and anatomy, which should be conducted first. T2-weighted imaging is the best method for identifying hemorrhage because of low T2 signal intensity due to deoxyhemoglobin in acute hematoma. Subacute hematoma appears hyperintense on both T1- and T2-weighted imaging because of conversion of deoxyhemoglobin to methemoglobin. Later, chronic hematoma shows hypointense on T2-weighted imaging because of hemosiderin-laden macrophages. Spinal cord edema shows hyperintense T2 signal against a background of normal nervous tissue.[22] MRI can indicate patient prognosis: patients with spinal cord hemorrhage always have a poor outcome, and patients with single level edema always have a better outcome.[23]

Diffusion-weighted MRI (DWI) can assess white matter tract integrity within the spinal cord. Shen et al.[24] performed MRI with DWI on 5 children with thoracic SCIWORA, in which 4 children exhibited abnormal T1 or T2 signal in the spinal cord. Even the patient with a normal spinal cord on T1- and T2-weighted imaging exhibited abnormal signal on DWI. DWI is likely to offer important data in SCI patients. In diffusion tensor imaging, the diffusion of water molecules is quantified in directions parallel and transverse to the plane of neuronal axons. This technique can examine and separate the white matter of the spinal cord from the gray matter to

evaluate structural damage. Mulcahey et al.[25] found that diffusion tensor imaging values have a better association with ISNCSCI examination than conventional MRI. Diffusion tensor tractography (DTT) can measure the volume of spinal white matter fibers. Zhu et al.[26] performed DTT on 20 adults with thoracolumbar complete SCI and found that the number of patients with improved American Spinal Injury Association (ASIA) grade had correlation with the measurement of DTT. Similarly, Zhu et al.[27] performed DTT on 24 patients with acute cervical SCI, and the results showed that the prognosis of complete SCI was related to the partial preservation of spinal white matter fibers, which can predict neurological outcomes. DTT has the potential to improve the diagnosis and prognostic evaluation of SCI.

Treatment

Treatment of SCI in children and adolescents is controversial. Current standard treatment includes conservative treatment and surgery. Conservative treatment mainly includes medications and spinal immobilization (cervical collar or brace). However, neither conservative nor surgical treatment can result in full recovery of neurological function. Cell therapy is a new strategy currently being studied and developed.

Medication

Medical therapy plays a considerable role in SCI treatment. Clinical trials have shown that it is effective in treating secondary injuries. Steroids and GM-1 ganglioside are currently approved for use in humans.[3] Methylprednisolone (MP) is the most common drug used in SCI and is the only drug that has been tested in controlled multicenter trials. However, the use of MP in children and adolescents is controversial, especially the dose. In 1990, Bracken et al.[28] first reported the efficacy of MP in treating SCI in the National Acute Spinal Cord Injury Study II (NASCIS II). In this study, MP was administered orally at 30 mg/Kg followed by an hourly infusion of 5.4 mg/Kg over 23 h. Although this therapy has been widely used in children and adolescents with SCI, the youngest person enrolled in NASCIS II was 13 years old. In 2013, the Congress of Neurological Surgeons updated their guidelines to recommendation against the use of MP. However, in 2017, AO spine updated guidelines to support the use of 24 h high-dose MP within 8 h after SCI.[29] Several recent studies explored the effectiveness of MP in SCI. Liu et al.[30] conducted a meta-analysis of 16 studies of high-dose MP in the treatment of SCI, and the results showed that high-dose MP was not associated with improved outcomes. Similarly, Sultan et al.[31] reviewed 12 studies, including 5 randomized controlled trials and 7 observational studies and reported that MP was not associated with improvement in motor or neurological scores.

Another controversy involves MP complications. Complications of high-dose steroids in adult with SCI have been well documented. Several relevant retrospective studies have been conducted in children and adolescents. In 2011, Arora et al.[32] reported a case series of 15 children with SCI aged 8–16 years who were treated with MP according to NASCIS II. High-dose steroids did not lead to any serious infections. In a retrospective case-control study conducted in 2015 on pediatric SCI patients, Cage et al.[33] found no notable difference in gastrointestinal or wound complications between the high-dose steroid group and control group. In fact, the rate of infection was higher in the control group. Caruso et al.[34] examined 36 patients with SCI below 17 years old and found that those who received steroids had a higher rate of complications than those who did not. Although current views differ on the use of high-dose steroids in children and adolescents with SCI, steroids remain the first choice.[32]

Gangliosides are sialic acid derivatives of endogenous glycolipids present in neuron cell membranes.[35] In vitro, they increase the formation and growth of neurites, induce neuronal regeneration, and promote neuroplasticity.[3] The use of GM-1 ganglioside to treat SCI in children and adolescents has not been reported. In a randomized study of adult SCI patients, GM-1 ganglioside treatment was associated with better improvement in pain and touch sensitivity compared to placebo. However, there was no significant difference in the degree of improvement in motor function between the treatment and placebo groups.[35] A randomized controlled trial of 760 patients with SCI showed that although GM-1 ganglioside promoted neurological recovery and improved bladder and rectal function, the differences compared with the control group were not significant.[36] GM-1 ganglioside trials have not been conducted in pediatric SCI patients.

Surgery

Like adults, children and adolescents with SCI and spinal fracture or dislocation should undergo surgery. According to the 2021 clinical guidelines, surgical indications for SCI is determined by the Sub-Axial Injury Classification and Severity Scale and Thoracolumbar Injury Classification and Severity score.[37] Surgical treatment is recommended when the score is $\geq$ 5. At a score of 4, surgery and conservative treatment are available. Spinal cord decompression with fusion is the most common surgical procedure. Spinal cord decompression improves the microenvironment of the spinal cord and promotes recovery of neurological function.[38] Depending on the severity of injury, the decompression method is different.[37] Anterior or posterior local decompression is used for the local edema which is result from the presence of spinal cord compression. Extensive laminectomy plus durotomy is feasibly performed for decompression of extensive spinal cord edema. Myelotomy can be considered in the presence of spinal cord hematoma or necrotic foci. In those patients with spinal instability, fusion may prevent recurrent SCI. However, the treatment of “real SCIWORA” is conservatively.[10] Surgical options for the other SCIWORA include halo and internal fixation.[39,40] Many reports suggest that conservative treatment can also achieve good results, however, most of which are case reports.[23,41–55] Seventeen articles reporting treatment of SCIWORA in children and adolescents are summarized in Table 1. Zhu et al.[56] studied adult patients with SCIWORA who underwent early (< 72 h) posterior laminectomy and durotomy with duroplasty decompression, and found that durotomy helps to thoroughly decompress the spinal cord and improve cerebrospinal fluid circulation. Although the safety and efficacy of this procedure in children and adolescents have not been proven, it may be an option.

In studies of adult with SCI, timing of surgery is associated with neurological prognosis.[57] Fehlings et al.[58] found that performance of decompression was safe within 24 h after injury, which was associated with improved neurologic outcome at 6 months follow-up. However, does the earlier the surgery, the better the prognosis? Biglari et al.[59] reported that decompression within the first 4 h after injury was not associated with better neurological outcome than decompression between 4 h and 24 h. Furlan et al.[60] recommend surgical intervention within 8 – 24 h when surgery is indicated, as early decompression may improve neurological function, reduce cost of care and lead to earlier engagement in rehabilitation exercises.

Cell therapy

Cell-based regenerative therapy has shown promise in treating SCI. Schwann cells, neural stem or progenitor cells, olfactory ensheathing cells, oligodendrocyte precursor cells and

Table 1
Treatment and outcome of SCIWORA in children and adolescents.

Study (year)	*n*	Male	Mean age (year)	Surgery	Conservative treatment	Outcome
Brauge et al.[41] (2019)	30	-	9.78		Glucocorticoid	The rate of at least partial neurologic recovery was 20/30.
Campbell et al.[23] (2018)	1	0	2		External immobilization	At the 3-month follow-up she was paraplegia.
Bansal et al.[42] (2016)	1	0	0.6		Glucocorticoid and cervical collar	The patient restored strength in upper and lower limbs.
Kim et al.[43] (2016)	1	0	1		Cervical collar	At the 12-month follow-up, cervical MRI was normal and neurological recovery was achieved at 24-month follow-up.
Mahajan et al.[39] (2013)	69	54	8.9	Halo and Internal fixation	Glucocorticoid, Cervical collar, Brace	There are 10 children with persistent deficits.
Trigylidas et al.[44] (2010)	3	2	2.5			Two patients suffered permanent disabilities and 1 had a transient deficit.
Grubenhoff et al.[45] (2008)	1	1	7		Glucocorticoid	The left hand recovered some flexion and extension functions.
Lee et al.[46] (2006)	1	1	1.16		Methylprednisolone	All neurological functions recovered completely.
Buldini et al.[47] (2005)	2	1	1.4		Methylprednisolone	At the 1-year follow-up, he was able to walk without support, but cannot raise the left arm.
Wang et al.[48] (2004)	6	-	7.7		Methylprednisolone, Philadelphia collar	Neurological function improved in all 6 cases, and neurological function was completely recovered in 3 cases.
Carreon et al.[49] (2004)	5	-	5.4		Glucocorticoid	Four cases had complete cord injuries. A 17-year-old girl who presented with a central cord lesion that resolved.
Ergun et al.[50] (2003)	1	0	12		Glucocorticoid	During a 2-year follow-up, the low extremity force was slightly lower than normal.
Yamaguchi et al.[51] (2002)	1	0	14		Glucocorticoid and Hyperbaric oxygen therapy	The patient was able to walk independently, and sensory disturbances were almost normal.
Brown et al.[40] (2001)	39	28	10.8	Halo	Cervical collar and Brace	Of them, 21 patients presented with short-lived paresthesias or motor weakness, and 8 with more severe and neurologic deficits.
Beck et al.[52] (2000)	1	0	16		Methylprednisolone	Patient recovered completely without any continuing neurological or motor deficits.
Pollina et al.[53] (1999)	1	1	3		Methylprednisolone, Guilford and TLSO orthosis	Patient regained the tricep and hand function to near baseline, but remained paraplegic.
Bondurant et al.[54] (1993)	1	1	2.25		Cervical collar	Motor function continued to improve and fecal continence was regained.

SCIWORA: spinal cord injury without radiographic abnormality, TLSO: thoracolumbosacral orthosis.
-: not mentioned.

mesenchymal stem cells have all been studied. Postulated mechanisms of action include neuroprotection, immunomodulation, axon regeneration, neuronal relay generation and myelin reproduction.[61] Several recent clinical trials of cell therapy for both adults and children have shown benefit. Kakabadze et al.[62] performed intrathecal injection of bone marrow mesenchymal stem cells in 18 patients with thoracolumbar SCI and 9 of them showed neurological improvement. Saberi et al.[63] studied 33 patients with cervical and thoracic SCI who underwent intramedullary Schwann cell transplantation, and found partial restoration of sensorimotor function with no adverse effects. Lima et al.[64] transplanted autologous olfactory sheath cells in 7 patients suffering from complete SCI and found that all of them showed neurological improvement.

Few cell transplantation trials for SCI in children and adolescents have been conducted. In a study of Jarocha et al.,[65] 5 children with chronic complete SCI underwent a total of 19 autologous bone marrow nucleated cell transplants, and only one was associated with an adverse event (transient bradycardia). Their results showed that children with chronic complete SCI who receive multiple transplants can experience improvement in neurological function and quality of life.

Despite the promise, cell therapy still faces challenges, such as ethical concerns regarding the source of transplanted cells and the correct direction of axonal regeneration. Furthermore, long-term safety and efficacy of the therapy have yet to be determined. Children and adolescents with SCI are a special patient group. With the progress of related research, more experimental results will be applied to the clinic, providing hope for the future.

Complications

Complications of SCI in children and adolescents are essentially the same as those in adults. However, continuing growth and development of children and adolescents and their unique anatomy may result in problems such as scoliosis, hip dislocation and hypercalcemia. Especially for very young patients, they have difficulty expressing their symptoms, which can lead to a hard diagnosis of complications such as autonomic dysreflexia.

Spinal deformity

Spinal deformity is common in children with SCI, especially those with skeletal hypoplasia. Scoliosis develops in 97% of children with SCI before growth spurt.[66] Patient age below 14.6 years at the time of SCI is a predictor of scoliosis.[67] Therefore, injured children before growth spurt should be closely monitored due to the high risk of scoliosis and curve progression. If bracing fails, deformity should be treated with surgical correction. In general, Cobb angle $> 40^{\circ}$ in a patient over the age of 10 years is an indication for corrective surgery.

Hip dislocation

Hip dislocation is also a common complication of SCI in children. McCarthy et al.[68] reported that hip subluxation or dislocation occurs in > 90% of children with SCI under 10 years of the age, and the incidence declined significantly thereafter. They speculate that it

may be related to the severity of injury. In their study, most patients (≤ 10 years old) had thoracic injuries, while older patients had mostly cervical injuries. However, they found no evidence of association between severity of injury and hip subluxation or dislocation. Hip dislocation may interfere the use of a standing frame. Therefore, close surveillance of the hip joint is necessary, as surgical intervention is usually required eventually.[69]

Hypercalcemia

Hypercalcemia occurs in 10% – 23% of SCI patients, with adolescents and young adult males most affected.[7] Spinal immobilization after SCI is thought to promote bone resorption, which can lead to hypercalcemia. Pediatric SCI patients are susceptible to hypercalcemia because of an increase of bone turnover and metabolically active bone mass in growing children and adolescents. The classic symptoms of hypercalcemia include pain, nausea, vomiting, malaise, lethargy, polyuria, polydipsia, dehydration and behavioral changes. Hypercalcemia sometimes manifesting as an acute abdomen, can lead to unnecessary surgery and is usually treated by aggressive hydration with furosemide diuresis and bisphosphonates.[69]

Autonomic dysreflexia

Schottler et al.[70] reported that approximately 51% of children with SCI at T6 or higher lesions develop autonomic dysreflexia. The pathophysiology, symptoms, and diagnostic criteria of autonomic dysreflexia are the same in children and adults. As with adults, children and adolescents with SCI have lower baseline blood pressure. Therefore, it is important to measure blood pressure on a regular basis. When the usual baseline is exceeded by > 20 mmHg, autonomic dysreflexia should be diagnosed.[71] As mentioned previously, younger children may not be able to articulate their symptoms. Therefore, autonomic dysreflexia should be suspected in such patients if they are unusually sleepy, irritable or crying.[72] Treatment for autonomic dysreflexia involves removing the potential irritants, e.g. the bowel and bladder should be emptied, and the tight clothing or accessories should be removed. Nifedipine can be considered if conservative treatment is not effective. Recurrent autonomic dysreflexia can be treated with oral prazosin or terazosin.

Conclusion

Pediatric and adult SCI differ in terms of pathogenesis, clinical characteristics and treatment. Treatment of SCI in children and adolescents remains controversial. Although current treatment cannot completely restore neurological function, patient quality of life can be enhanced. Continued developments and advances in the research of SCI may eventually provide a cure for children and adolescents with this kind of injury.

Funding

This study was supported by the LiaoNing Revitalization Talents Program (XLYC1807131).

Ethical statement

Not applicable.

Declaration of competing interest

All authors declare that there is no conflict of interests.

Author contributions

All authors contributed to the conception and design, writing and editing the manuscript. Zhong-Hai Li takes responsibility for the integrity of the work as a whole, from inception to finished article.

Acknowledgments

We thank Liwen Bianji (Edanz) (https://www.liwenbianji.cn) for editing the language of a draft of this manuscript.

References

1. Pang D, Wilberger Jr JE. Spinal cord injury without radiographic abnormalities in children. *J Neurosurg*. 1982;57:114–129. https://doi.org/10.3171/jns.1982.57.1.0114.
2. McDonald JW, Sadowsky C. Spinal-cord injury. *Lancet*. 2002;359:417–425. https://doi.org/10.1016/S0140-6736(02)07603-1.
3. Cristante AF, Barros Filho TEP, Marcon RM, et al. Therapeutic approaches for spinal cord injury. *Clinics*. 2012;67:1219–1224. https://doi.org/10.6061/clinics/2012(10)16.
4. Kriss VM, Kriss TC. SCIWORA (spinal cord injury without radiographic abnormality) in infants and children. *Clin Pediatr*. 1996;35:119–124. https://doi.org/10.1177/000992289603500302.
5. Augutis M, Abel R, Levi R. Pediatric spinal cord injury in a subset of European countries. *Spinal Cord*. 2006;44:106–112. https://doi.org/10.1038/sj.sc.3101793.
6. Canosa-Hermida E, Mora-Boga R, Cabrera-Sarmiento JJ, et al. Epidemiology of traumatic spinal cord injury in childhood and adolescence in Galicia, Spain: report of the last 26-years. *J Spinal Cord Med*. 2019;42:423–429. https://doi.org/10.1080/10790268.2017.1389836.
7. Vogel LC, Anderson CJ. Spinal cord injuries in children and adolescents: a review. *J Spinal Cord Med*. 2003;26:193–203. https://doi.org/10.1080/10790268.2003.11753682.
8. Zou ZW, Teng AN, Huang LY, et al. Pediatric spinal cord injury without radiographic abnormality: the Beijing experience. *Spine (Phila Pa*. 2021;46:1083–1088. https://doi.org/10.1097/BRS.0000000000004030, 1976.
9. Carroll T, Smith CD, Liu X, et al. Spinal cord injuries without radiologic abnormality in children: a systematic review. *Spinal Cord*. 2015;53:842–848. https://doi.org/10.1038/sc.2015.110.
10. Freigang V, Butz K, Seebauer CT, et al. Management and mid-term outcome after "real SCIWORA" in children and adolescents. *Global Spine J*. 2021. https://doi.org/10.1177/2192568220979131, 2192568220979131.
11. Cirak B, Ziegfeld S, Knight VM, et al. Spinal injuries in children. *J Pediatr Surg*. 2004;39:607–612. https://doi.org/10.1016/j.jpedsurg.2003.12.011.
12. Alas H, Pierce KE, Brown A, et al. Sports-related cervical spine fracture and spinal cord injury: a review of nationwide pediatric trends. *Spine (Phila Pa*. 2021;46:22–28. https://doi.org/10.1097/BRS.0000000000003718, 1976.
13. Achildi O, Betz RR, Grewal H. Lapbelt injuries and the seatbelt syndrome in pediatric spinal cord injury. *J Spinal Cord Med*. 2007;30:21–24. https://doi.org/10.1080/10790268.2007.11753964.
14. Ren J, Zeng G, Ma YJ, et al. Pediatric thoracic SCIWORA after back bend during dance practice: a retrospective case series and analysis of trauma mechanisms. *Childs Nerv Syst*. 2017;33:1191–1198. https://doi.org/10.1007/s00381-017-3407-0.
15. Thompson TP, Pearce J, Chang G, et al. Surfer's myelopathy. *Spine*. 2004;29:353–356. https://doi.org/10.1097/01.brs.0000134689.84162.e7.
16. Freedman BA, Malone DG, Rasmussen PA, et al. Surfer's myelopathy: a rare form of spinal cord infarction in novice surfers: a systematic review. *Neurosurgery*. 2016;78:602–611. https://doi.org/10.1227/NEU.0000000000001089.
17. Chang CW, Donovan DJ, Liem LK, et al. Surfers' myelopathy: a case series of 19 novice surfers with nontraumatic myelopathy. *Neurology*. 2012;79:2171–2176. https://doi.org/10.1212/WNL.0b013e31827595cd.
18. Gandhi J, Lee MY, Joshi G, et al. Surfer's myelopathy: a review of etiology, pathogenesis, evaluation, and management. *J Spinal Cord Med*. 2021;44:2–7. https://doi.org/10.1080/10790268.2019.1577057.
19. Wang YL, Zhu FZ, Zeng L, et al. Surfer myelopathy in children: a case series study. *World Neurosurg*. 2021;148:227–241. https://doi.org/10.1016/j.wneu.2020.12.135.
20. Rozzelle CJ, Aarabi B, Dhall SS, et al. Spinal cord injury without radiographic abnormality (SCIWORA). *Neurosurgery*. 2013;72:227–233. https://doi.org/10.1227/NEU.0b013e3182770ebc.
21. Smith AC, Albin SR, O'Dell DR, et al. Axial MRI biomarkers of spinal cord damage to predict future walking and motor function: a retrospective study. *Spinal Cord*. 2021;59:693–699. https://doi.org/10.1038/s41393-020-00561-w.
22. Szwedowski D, Walecki J. Spinal cord injury without radiographic abnormality (SCIWORA) - clinical and radiological aspects. *Pol J Radiol*. 2014;79:461–464. https://doi.org/10.12659/PJR.890944.

23. Iaconis Campbell J, Coppola F, Volpe E, et al. Thoracic spinal cord injury without radiologic abnormality in a pediatric patient case report. *J Surg Case Rep*. 2018;10:250. https://doi.org/10.1093/jscr/rjy250.
24. Shen HY, Tang Y, Huang L, et al. Applications of diffusion-weighted MRI in thoracic spinal cord injury without radiographic abnormality. *Int Orthop*. 2007;31:375–383. https://doi.org/10.1007/s00264-006-0175-y.
25. Mulcahey MJ, Samdani A, Gaughan J, et al. Diffusion tensor imaging in pediatric spinal cord injury: preliminary examination of reliability and clinical correlation. *Spine (Phila Pa*. 2012;37:797–803. https://doi.org/10.1097/BRS.0b013e3182470a08, 1976.
26. Zhu FZ, Zeng L, Gui S, et al. The role of diffusion tensor imaging and diffusion tensor tractography in the assessment of acute traumatic thoracolumbar spinal cord injury. *World Neurosurg*. 2021;150:23–30. https://doi.org/10.1016/j.wneu.2021.01.146.
27. Zhu FZ, Liu Y, Zeng L, et al. Evaluating the severity and prognosis of acute traumatic cervical spinal cord injury: a novel classification using diffusion tensor imaging and diffusion tensor tractography. *Spine (Phila Pa*. 2021;46:687–694. https://doi.org/10.1097/brs.0000000000003923, 1976.
28. Bracken MB, Shepard MJ, Collins WF, et al. A randomized, controlled trial of methylprednisolone or naloxone in the treatment of acute spinal-cord injury. Results of the Second National Acute Spinal Cord Injury Study. *N Engl J Med*. 1990;322:1405–1411. https://doi.org/10.1056/nejm199005173222001.
29. Fehlings MG, Tetreault LA, Wilson JR, et al. A clinical practice guideline for the management of acute spinal cord injury: introduction, rationale, and scope. *Global Spine J*. 2017;7:84s–94s. https://doi.org/10.1177/2192568217703387.
30. Liu ZY, Yang Y, He L, et al. High-dose methylprednisolone for acute traumatic spinal cord injury: a meta-analysis. *Neurology*. 2019;93:841–850. https://doi.org/10.1212/wnl.0000000000007998.
31. Sultan I, Lamba N, Liew A, et al. The safety and efficacy of steroid treatment for acute spinal cord injury: a Systematic Review and meta-analysis. *Heliyon*. 2020;62, 03414. https://doi.org/10.1016/j.heliyon.2020.e03414.
32. Arora B, Suresh S. Spinal cord injuries in older children: is there a role for high-dose methylprednisolone? *Pediatr Emerg Care*. 2011;27:1192–1194. https://doi.org/10.1097/PEC.0b013e31823b4d06.
33. Cage JM, Knox JB, Wimberly RL, et al. Complications associated with high-dose corticosteroid administration in children with spinal cord injury. *J Pediatr Orthop*. 2015;35:687–692. https://doi.org/10.1097/bpo.0000000000000364.
34. Caruso MC, Daugherty MC, Moody SM, et al. Lessons learned from administration of high-dose methylprednisolone sodium succinate for acute pediatric spinal cord injuries. *J Neurosurg Pediatr*. 2017;20:567–574. https://doi.org/10.3171/2017.7.Peds1756.
35. Barros Jr TE, De Araujo FF, Higino LDP, et al. The effect OF MONOSIALOGANGLYOSIDE (GM-1) administration IN spinal cord injury. *Acta Ortopédica Bras*. 2016;24:123–126. https://doi.org/10.1590/1413-785220162403160032.
36. Geisler FH, Coleman WP, Grieco G, et al. The Sygen multicenter acute spinal cord injury study. *Spine*. 2001;26:87–98. https://doi.org/10.1097/00007632-200112151-00015.
37. Guo X, Feng Y, Sun T, et al. Clinical guidelines for neurorestorative therapies in spinal cord injury (2021 China version). *J Neurorestoratol*. 2021;9:31–49. https://doi.org/10.26599/jnr.2021.9040003.
38. Proctor MR. Spinal cord injury. *Crit Care Med*. 2002;30:489–499. https://doi.org/10.1097/00003246-200211001-00014.
39. Mahajan P, Jaffe DM, Olsen CS, et al. Spinal cord injury without radiologic abnormality in children imaged with magnetic resonance imaging. *J Trauma Acute Care Surg*. 2013;75:843–847. https://doi.org/10.1097/TA.0b013e3182a74abd.
40. Brown RL, Brunn MA, Garcia VF. Cervical spine injuries in children: a review of 103 patients treated consecutively at a level 1 pediatric trauma center. *J Pediatr Surg*. 2001;36:1107–1114. https://doi.org/10.1053/jpsu.2001.25665.
41. Brauge D, Plas B, Vinchon M, et al. Multicenter study of 37 pediatric patients with SCIWORA or other spinal cord injury without associated bone lesion. *Orthop Traumatol Surg Res*. 2020;106:167–171. https://doi.org/10.1016/j.otsr.2019.10.006.
42. Bansal KR, Chandanwale AS. Spinal cord injury without radiological abnormality in an 8 Months old female child: a case report. *J Orthop Case Rep*. 2016;6:8–10. https://doi.org/10.13107/jocr.2250-0685.363.
43. Kim SH, Yoon SH, Cho KH, et al. Spinal cord injury without radiological abnormality in an infant with delayed presentation of symptoms after a minor injury. *Spine (Phila Pa*. 2008;33:792–794. https://doi.org/10.1097/BRS.0b013e3181878719, 1976.
44. Trigylidas T, Yuh SJ, Vassilyadi M, et al. Spinal cord injuries without radiographic abnormality at two pediatric trauma centers in Ontario. *Pediatr Neurosurg*. 2010;46:283–289. https://doi.org/10.1159/000320134.
45. Grubenhoff JA, Brent A. Case report: Brown-Séquard syndrome resulting from a ski injury in a 7-year-old male. *Curr Opin Pediatr*. 2008;20:341–344. https://doi.org/10.1097/MOP.0b013e3282ff62d6.
46. Lee CC, Lee SH, Yo CH, et al. Complete recovery of spinal cord injury without radiographic abnormality and traumatic brachial plexopathy in a young infant falling from a 30-feet-high window. *Pediatr Neurosurg*. 2006;42:113–115. https://doi.org/10.1159/000090466.
47. Buldini B, Amigoni A, Faggin R, et al. Spinal cord injury without radiographic abnormalities. *Eur J Pediatr*. 2006;165:108–111. https://doi.org/10.1007/s00431-005-0004-0.
48. Wang MY, Hoh DJ, Leary SP, et al. High rates of neurological improvement following severe traumatic pediatric spinal cord injury. *Spine (Phila Pa*. 2004;29:1493–1497. https://doi.org/10.1097/01.brs.0000129026.03194.0f, 1976.
49. Carreon LY, Glassman SD, Campbell MJ. Pediatric spine fractures: a review of 137 hospital admissions. *J Spinal Disord Tech*. 2004;17:477–482. https://doi.org/10.1097/01.bsd.0000132290.50455.99.
50. Ergun A, Oder W. Pediatric care report of spinal cord injury without radiographic abnormality (SCIWORA): case report and literature review. *Spinal Cord*. 2003;41:249–253. https://doi.org/10.1038/sj.sc.3101442.
51. Yamaguchi S, Hida K, Akino M, et al. A case of pediatric thoracic SCIWORA following minor trauma. *Childs Nerv Syst*. 2002;18:241–243. https://doi.org/10.1007/s00381-002-0560-9.
52. Beck A, Gebhard F, Kinzl L, et al. Spinal cord injury without radiographic abnormalities in children and adolescents: case report of a severe cervical spine lesion and review of literature. *Knee Surg Sports Traumatol Arthrosc*. 2000;8:186–189. https://doi.org/10.1007/s001670050212.
53. Pollina J, Li V. Tandem spinal cord injuries without radiographic abnormalities in a young child. *Pediatr Neurosurg*. 1999;30:263–266. https://doi.org/10.1159/000028808.
54. Bondurant CP, Oró JJ. Spinal cord injury without radiographic abnormality and Chiari malformation. *J Neurosurg*. 1993;79:833–838. https://doi.org/10.3171/jns.1993.79.6.0833.
55. Konovalov N, Peev N, Zileli M, et al. Pediatric cervical spine injuries and SCIWORA: WFNS spine committee recommendations. *Neurospine*. 2020;17:797–808. https://doi.org/10.14245/ns.2040404.202.
56. Zhu FZ, Yao S, Ren ZW, et al. Early durotomy with duroplasty for severe adult spinal cord injury without radiographic abnormality: a novel concept and method of surgical decompression. *Eur Spine J*. 2019;28:2275–2282. https://doi.org/10.1007/s00586-019-06091-1.
57. Wutte C, Klein B, Becker J, et al. Earlier decompression (< 8 hours) results in better neurological and functional outcome after traumatic thoracolumbar spinal cord injury. *J Neurotrauma*. 2019;36:2020–2027. https://doi.org/10.1089/neu.2018.6146.
58. Fehlings MG, Vaccaro A, Wilson JR, et al. Early versus delayed decompression for traumatic cervical spinal cord injury: results of the Surgical Timing in Acute Spinal Cord Injury Study (STASCIS). *PLoS One*. 2012;7:32037. https://doi.org/10.1371/journal.pone.0032037.
59. Biglari B, Child C, Yildirim TM, et al. Does surgical treatment within 4 hours after trauma have an influence on neurological remission in patients with acute spinal cord injury? *Therapeut Clin Risk Manag*. 2016;12:1339–1346. https://doi.org/10.2147/tcrm.S108856.
60. Furlan JC, Noonan V, Cadotte DW, et al. Timing of decompressive surgery of spinal cord after traumatic spinal cord injury: an evidence-based examination of pre-clinical and clinical studies. *J Neurotrauma*. 2011;28:1371–1399. https://doi.org/10.1089/neu.2009.1147.
61. Assinck P, Duncan GJ, Hilton BJ, et al. Cell transplantation therapy for spinal cord injury. *Nat Neurosci*. 2017;20:637–647. https://doi.org/10.1038/nn.4541.
62. Kakabadze Z, Kipshidze N, Mardaleishvili K, et al. Phase 1 trial of autologous bone marrow stem cell transplantation in patients with spinal cord injury. *Stem Cell Int*. 2016, 6768274. https://doi.org/10.1155/2016/6768274.
63. Saberi H, Firouzi M, Habibi Z, et al. Safety of intramedullary Schwann cell transplantation for postrehabilitation spinal cord injuries: 2-year follow-up of 33 cases. *J Neurosurg Spine*. 2011;15:515–525. https://doi.org/10.3171/2011.6.Spine10917.
64. Lima C, Pratas-Vital J, Escada P, et al. Olfactory mucosa autografts in human spinal cord injury: a pilot clinical study. *J Spinal Cord Med*. 2006;29:191–203. https://doi.org/10.1080/10790268.2006.11753874.
65. Jarocha D, Milczarek O, Kawecki Z, et al. Preliminary study of autologous bone marrow nucleated cells transplantation in children with spinal cord injury. *Stem Cells Transl Med*. 2014;3:395–404. https://doi.org/10.5966/sctm.2013-0141.
66. Dearolf 3rd WW, Betz RR, Vogel LC, et al. Scoliosis in pediatric spinal cord-injured patients. *J Pediatr Orthop*. 1990;10:214–218.
67. Kulshrestha R, Kuiper JH, Masri WE, et al. Scoliosis in paediatric onset spinal cord injuries. *Spinal Cord*. 2020;58:711–715. https://doi.org/10.1038/s41393-020-0418-6.
68. McCarthy JJ, Chafetz RS, Betz RR, et al. Incidence and degree of hip subluxation/dislocation in children with spinal cord injury. *J Spinal Cord Med*. 2004;27:80–83. https://doi.org/10.1080/10790268.2004.11753423.
69. Powell A, Davidson L. Pediatric spinal cord injury: a review by organ system. *Phys Med Rehabil Clin*. 2015;26:109–132. https://doi.org/10.1016/j.pmr.2014.09.002.
70. Schottler J, Vogel LC, Sturm P. Spinal cord injuries in young children: a review of children injured at 5 years of age and younger. *Dev Med Child Neurol*. 2012;54:1138–1143. https://doi.org/10.1111/j.1469-8749.2012.04411.x.
71. Consortium for Spinal Cord Medicine. Acute management of autonomic dysreflexia: individuals with spinal cord injury presenting to health-care facilities. *J Spinal Cord Med*. 2002;25:67–88.
72. Hickey KJ, Vogel LC, Willis KM, et al. Prevalence and etiology of autonomic dysreflexia in children with spinal cord injuries. *J Spinal Cord Med*. 2004;27:54–60. https://doi.org/10.1080/10790268.2004.11753786.