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Review Article

A Review of Spinal Cord Injury, Looking Into the Role of Closed Reduction, Timing of Surgery and Evidence For and Against the Use of Steroids in Acute Spinal Cord Injury

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Abstract

Cervical Spinal Cord Injury (SCI) can result as a consequence of traumatic cervical spine fractures or dislocations. Reduction of the dislocation would help to restore the deformity and would help maintain canal diameter and may lead to an improved neurological outcome. There have been several authors who feel that traumatic disc herniation with fracture-dislocation or a facet joint dislocation increases the risk of spinal cord herniation after reduction. In 2002, the American Association of Neurological Surgeons / Congress of Neurological Surgeons published guidelines on the efficacy of closed reduction. This review looked at the efficacy of acute cervical spine fracture dislocation injuries, looking at over 1200 patients who were treated with closed reduction. They noted that roughly 80% of these reductions were successful with a low transient or permanent complication rate. Patient with cervical facture dislocations who cannot be examined because of decreased conscious levels, cannot have post reduction neurology assessed. For these set of patients, an MRI before attempted reduction would be recommended as a treatment option.

Animal laboratory tests which demonstrate that the strength of neuroprotection seems to have an inverse correlation with time to decompression. STASCIS looked at early decompressive surgery being defined at 24 hours or earlier and late surgery being classified as after this. There was a significant improvement of at least grade 2 AIS for those who were operated on within 24 hours compared to delayed surgery. The Canadian cohort study published by Wilson et al also advocated early decompression. What is defined as early surgery is a topic of contention.

The third area of discussion is around the use of steroid in acute spinal cord injury. Many drugs have been utilized in experimental models and have been shown to improve outcome in rat models. Methyprednisolone is the most studied drug for spinal cord injury. The three prominent trials were the NASCIS (North American Spinal Cord Injury Studies). They did not demonstrate any additional benefit for the use of steroids in patients with acute spinal cord injury.

Introduction

Traumatic spinal cord injury is very common. In the US, over 200,000 people are affected with 10,000 new cases per year [1-3]. Approximately 4000 of these patients die before hospital, and 1000 die in hospital. In the rest of the world, he incidence id about 15-40 million cases per million with a prevalence of 800cases per million. The majority of patients are male, with some studies having quoted a proportion of 70-80%. The patient population is young, with the average age range being 4 years. Hence, the subsequent ramifications of the injury and the associated medical decisions result in a large economic burden and social cost [4].

In this essay, areas of controversy will be mentioned. Treatment practice within units, around the country and around the world varies. Most of these areas in controversy are due to a lack of a robust evidence base from which clinicians can make decisions. Within spinal cord injury, these are both controversy in the surgical management and in the medical management. These include:

The role of closed reduction, the timing of surgery and the evidence for and against the use of steroids in spinal cord injury.

Role of closed reduction

Cervical spinal cord injury (SCI) can result as a consequence of traumatic cervical spine fractures or dislocations. They occur as a result of narrowing of spinal canal narrowing or by direct displacement of one component of the cervical spine causing focal compression of the spinal cord [5]. Reduction of the dislocation would help to restore the deformity and would help maintain canal

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diameter. If this is carried out soon after injury, decompression of the spinal cord may lead to an improved neurological outcome. Up until 2001, there was much published data which promoted closed reduction, with minimal associated neurological complications. Soon after, there were descriptive case series which associated cervical disc herniation, which were identified on post reduction MRI scans [6]. They reported ventral compression of the spinal cord by displaced disc material.

There have been several authors who feel that traumatic disc herniation with fracture-dislocation or a facet joint dislocation increases the risk of spinal cord herniation after reduction [7,8]. These authors also advocate the use of MRI where possible. However, obtaining an MRI scan would involve transporting the patient, one with an unstable spine, to the MRI machine would cause a delay in spinal reduction. This would require adequate justification, especially as there is cumulative evidence which suggests that early reduction would influence outcome [9,10]. Therefore, where expertise is present, initial closed reduction is generally undertaken as an urgent bases for the treatment of acute traumatic SCI.

In 2002, the American Association of Neurological Surgeons/ Congress of Neurological Surgeons published guidelines on the efficacy of closed reduction [11]. This review looked at the efficacy of acute cervical spine fracture dislocation injuries, looking at over 1200 patients who were treated with closed reduction. They noted that roughly 80% of these reductions were successful with a low transient or permanent complication rate. O'connor et al reported a case series of 21 patients who underwent closed reduction of the sub-axial cervical facet injuries, with only patient suffering transient neurological deterioration [12]. Similar results were shows by Anderson et al who performed a retrospective series of 45 patients who underwent closed reduction of facet dislocation injuries, with no statistical deterioration in motor function post closed reduction [13]. There are numerous examples of class 3 evidence which support the concept of early closed reduction being beneficial to patient care, but there is a last of class 1 and class 2 evidence [6].

The risks of closed reduction

As mentioned earlier, the incidence of neurological deterioration after closed reduction remains low, with the complication rate being < 1.0% [6]. To date, there are no known cases in the literature of patients who has suffered new permeant neurological deficit after closed reduction. There are published cases of transient neurological deterioration. The American Association of Neurological Surgeons / Congress of Neurological Surgeons review found 20 patients (from 1200) with a transient neurological deficit. These deficits improved after reduction with weights, open reduction or resolved spontaneously [14,15]. The common causes of neurological impairment post reduction included: disc herniation, epidural haematoma, overdistraction, failure to recognize a more rostral noncontiguous lesion and spinal cord edema [6].

Pre-reduction MRI

As there have been some reports of neurological complications post reduction, some authors now advocate the use of pre-reduction MRI to assess for ventral cord compromise secondary to traumatic disc disruption. The main concern is extruded disc material causing further neurological impairment.

The pre-reduction MRI however is not a decision that should be taken lightly. Significant time is lost in the arranging of an MRI scan, transport to the facility and performing the scan itself, time potentially wasted when urgent closed reduction could have already been performed.

There have only been 2 documented cases of neurological deterioration secondary to closed cervical reduction due to cord compression from a disc herniation [10,16].

From the above presented information, the published literature favors the concenter of closed reduction by traction reduction being a safe option, with low risks of complications. Furthermore, the use or pre reduction MRI scan is unnecessary as it would provide unnecessary delays. However, there is a paucity of class 1 and class 2 data on this.

Patient with cervical facture dislocations who cannot be examined because of decreased conscious levels, cannot have post reduction neurology assessed. For these set of patients, an MRI before attempted reduction would be recommended as a treatment option [6].

Timing of surgery

The current concepts for spinal cord injury indicate that there seem to be primary and secondary mechanisms that lead to a neurological insult. The primary injury is caused by rapid spinal cord compression and contusion, and leads to a signaling cascade which causes the secondary injury. Preventing this signaling changes promotes neuroprotection and this where therapeutic intervention is aimed [17,18]. Animal laboratory tests which demonstrate that the strength of neuroprotection seems to have an inverse correlation with time to decompression [19]. The theory therefore stated that those who undergo early decompressive surgery, have less neural tissue destruction and have an improved clinical outcome.

Up until the Surgical Timing in Acute Spine Cord Injury Study (STASCIS), there has not been any clinical evidence to provide support to the above hypothesis. They looked at early decompressive surgery being defined at 24 hours or earlier and late surgery being classified as after this. There was a significant improvement of at least grade 2 AIS for those who were operated on within 24 hours compared to delayed surgery, The STASCIS study also looked into post operative complication and mortality rate, and there was no statistical difference between the groups.

The Canadian cohort study by Wilson et al also found that early surgery was statistically significant when predicting an enhanced motor recovery [20].

It is clear from the published data, that early surgery has shown to reduce the effects of secondary injury of spinal canal injury. What is defined as early surgery is a topic of contention. In animal studies, the timing of surgical decompression varies between 8 - 24 hours post injury, and there has shown to be a time dependent effect. (20.5). However, in the real world, factors such as time for pre hospital transport, trauma stabalisation, definitive diagnosis and then definitive surgery all take time. The previous accepted time for early surgery was 72 hours, however work by McKinlet et al [21] and Vaccaro et al [22] failed to find a benefit between early (<72 hour) and delayed (>72 hour) surgery. A recent systematic review showed that decompression within 24 hours of injury resulted in improved

outcomes compared to delayed decompression. (22.5) 24 hours therefore represents a cut off where practicality and clinical benefit are both seen.

Evidence for and against the use to steroids in acute SCI

The third area of discussion is around the use of steroid in acute spinal cord injury. Many drugs have been utalised in experimental models and have been shown to improve outcome in rat models. Methyprednisolone is the most studied drug for spinal cord injury. The three prominent trials were the NASCIS (North American Spinal Cord Injury Studies).

Methylprednisolone is thought to preserve the blood-spinal barrier to reduce vagogenis oedema, demonstrated to increase spinal blood flow and scavenge free radicals. The first NASCIS study compared 1000mg bolus and 1000mg daily thereafter for 10 days compared to a standard dose (100mg bolus and 100mg daily for 10 days. This demonstrated no significant difference in neurological recovery for 1 year after injury [23]. However, this study was criticized for using a too low dose of methylprednisolone.

In 1990, Bracken performed the second National Acute Spinal Cord Injury Study. Methylprednisolone was given as a bolus 30mg/kg followed by an infusion at 5.4 mg/kg for 23 hours. Those who were treated within 8 hours of their injury showed significant improvement versus placebo and naloxone, at 6 months of injury [24].

1997 hosted the publication of the third National Acute Spinal Cord Injury Study. They concluded that patients who had acute SCI, those treated with methylprednisolone within 3 hours of injury should have a treatment regime for 24 hours, whereas those who were treated with methylprednisolone between hours 3 and 8, should ben maintained on steroid therapy for 48 hours [25].

Matsumoto et al also performed a prospective randomized double-blinded study comparing high-dose methylprednisolone sodium succinate versus placebo fo acute cervical spinal cord injury. There was a significantly increased chance of developing gastrointestinal and pulmonary complications in the patient population [26].

NICE has published guidelines on the use of steroids for acute spinal injury in February 2016. The Guideline Development Group (GDG) looked at evidence which was presented from six RCTs with four additional subsidiary papers. All of their studies had methyprednisoline as one of the comparators. NICE looked at a number of parametres which are listed below:

Methylprednisolone *versus* placebo

→ No clear conclusion of benefit or harm for or against mortality could be ascertained

→ No appreciable, clinically important benefit to improve neurological benefit was noted

→ Methylprednisolone resulted in a clear and clinically important increase in rate of hyperglycaemia and pulmonary embolism versus placebo

Methylprednisolone moderate *versus* low

→ No clear conclusion of benefit or harm for or against mortality could be determined

→ No clear benefit for high dose methylprednisolone versus low dose to improve neurological function was noted.

→ Moderate dose methylprednisolone resulted in a clinically important increase in UTI and wound infection rates.

Methylprednisolone 48 hours *versus* 24 hours

→ No clinically important difference was noted in a 48 hour regme versus a 24 hour regime for mortality

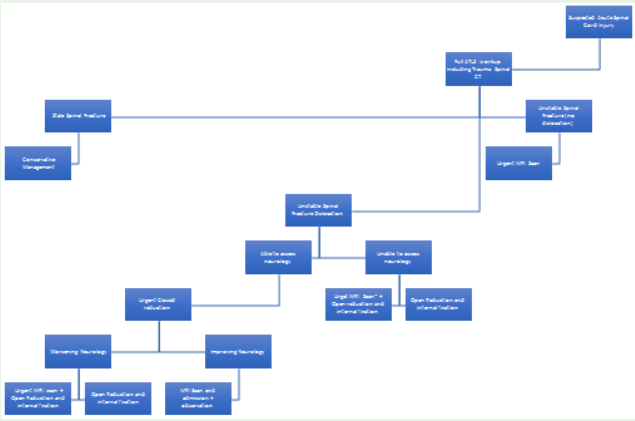
→ There was no benefit for a 48 our regime for improving neurological function.

→ Rates of side effects for 48 hours versus 24 hour regime were not large enough to be clinically appreciable.

NICE developed these conclusions after analysing multiple datasets and came to the conclusion that steroids would not be of benefit in the treatment of spinal cord injury [27].

Treatment algorithm and conclusion

From the information that we have presented above, we propose the following treatment algorithm as displayed below:



Flowchart : Algorithm.

We advocate that where an urgent plus operative intervention is needed, the MRI scan should only take place where there would not be a significant delay to surgical intervention.

We would try and advocate any open reduction and internal fixation surgery to be performed from 24 hours of the accident, where possible.

We also do not advocate the use of steroids in this patient population.

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References

1. Kraus JF, Franti CE, Riggins RS. "Incidence of traumatic spinal cord lesions." Journal of Chronic Diseases. 1975; 28: 471-492.
2. "Spinal cord injury facts and figures at a glance" Journal of Spinal Cord Medicine. 2010; 33: 439-440.
3. Go B, DeVivo M, Richards J, "The epidemiology of spinal cord injury," in Spinal Cord Injury: Clinical Outcomes From The Model Systems, S. Stover, J. Delisa, and G. Whiteneck, Eds. 1995; 21-55.
4. Ahmed M. Raslan, Andrew N. Nemecek. Controversies in the Surgical Management of Spinal Cord Injuries. 2012.
5. Hadley MN, Walters BC, Grabb BC, Oyesiku NM, Przybylski GJ, Resnick DK. initial closed reduction of cervical spine fracture - dislocation injured. Neurosurgery. 2002; 50(3 Suppl): S44-S50.
6. Lee AS, MacLean JC, Newton DA. "Rapid traction for reduction of cervical spine dislocations," Journal of Bone & Joint Surgery-British Volume. 1994; 76: 352-356.
7. Rizzolo SJ, Piazza MR, Cotler JM, Balderston RA, Schaefer D, Flanders A. "Intervertebral disc injury complicating cervical spine trauma," Spine. 1991; 16: S187-S189.
8. Brunette DD, Rockswol GL. "Neurologic recovery following rapid spinal realignment for complete cervical spinal cord injury," Journal of Trauma-Injury Infection & Critical Care. 1987; 27: 445-447.
9. Harrington JF, Likavec MJ, Smith AS. "Disc herniation in cervical fracture subluxation," Neurosurgery. 1991; 29: 374-379.
10. Initial closed reduction of cervical spinal fracture-dislocation injuries. In: Guidelines for the management of acute cervical spine and spinal cord injuries. Neurosurgery. 2002; 50: S51-S57.
11. O'Connor PA, McCormack O, Noël J, McCormack D, O'Bryne J. Anterior displacement correlates with neurological impairment in cervical facet dislocations. IntOrthop. 2003; 27: 190-193.
12. Anderson GD, Voets C, Ropiak R, et al. Analysis of patient variables affecting neurologic outcome after traumatic cervical facet dislocation. Spine J. 2004; 4: 506-512.
13. Farmer J, Vaccaro A, Albert TJ, Malone S, Balderston RA, Cotler JM. Neurologic deterioration after spinal cord injury. J Spinal Disord. 1998; 11: 192-196. 14.
14. Fehlings MG, Tator CH. An evidence-based review of decompressive surgery in acute spinal cord injury: rationale, indications, and timing based on experimental and clinical studies. J Neurosurg. 1999; 91(1 suppl): 1-11.
15. Maiman DJ, Barolat G, Larson SJ. Management of bilateral locked facets of the cervical spine. Neurosurgery. 1986; 18: 542-547.
16. Fehlings M, Sekhon L, eds. Cellular, ionic and biomolecular mechanisms of the injury process. Chicago: American Association of Neurologic Surgeons. 33-50.
17. Dimar J, Glassman S, Raque G, Zhang Y, Shields C. The influence of spinal canal narrowing and timing of decompression on neurologic recovery after spinal cord contusion in a rat model. Spine. 1999; 24: 1623-1633.
18. Furlan J, Noonan V, Cadotte D, Fehlings M. Timing of Decompressive Surgery of Spinal Cord after Traumatic Spinal Cord Injury: An Evidence-Based Examination of Pre-Clinical and Clinical Studies. J Neurotrauma. 2009; 27: 1-29.
19. http://journals.plos.org/plosone/article/file?id=10.1371/journal.pone.0032037&type=printable
20. McKinley W, Meade M, Kirshblum S, Barnard B. Outcomes of early surgical management versus late or no surgical intervention after acute spinal cord injury. Arch Phys Med Rehab. 2004; 85: 1818-1825.
21. Vaccaro AR, Daugherty RJ, Sheehan TP, Dante SJ, Cotler JM, Balderston RA, et al. Neurologic outcome of early versus late surgery for cervical spinal cord injury. Spine. 1997; 22: 609-612.
22. LaRosa G, Conti A, Cardali S, Cacciola F, Tomasello F. Does early decompression improve neurological outcome of spinal cord injured patients? Appraisal of the literature using a meta-analytical approach. Spinal Cord. 2004; 42: 503-512.
23. Bracken MB, Collins WF, Freeman DF, Shepard MJ, Wagner FW, Silten RM et al. Efficacy of methylprednisolone in acute spinal cord injury. JAMA. 1984; 251: 45-52.
24. Bracken MB, Shepard MJ, Collins WF, Holford TR, Young W, Baskin DS 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. New England Journal of Medicine. 1990; 322: 1405-1411.
25. Bracken MB, Shepard MJ, Holford TR, Leo-Summers L, Aldrich EF, Fazl M et al. Administration of methylprednisolone for 24 or 48 hours or tirilazadmesylate for 48 hours in the treatment of acute spinal cord injury. Results of the Third National Acute Spinal Cord Injury Randomized Controlled Trial. National Acute Spinal Cord Injury Study. JAMA. 1997; 277: 1597-1604.
26. Matsumoto T, Tamaki T, Kawakami M, Yoshida M, Ando M, Yamada H. Early complications of high-dose methylprednisolone sodium succinate treatment in the follow-up of acute cervical spinal cord injury. Spine. 2001; 26: 426-430.
27. https://www.nice.org.uk/guidance/ng41/evidence/full-guideline-pdf-2358425776

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