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Original Research Article

Effect of Surgical Decompression on Intrathecal Pressure Variation Proximal and Distal to the Site of Injury in Patients of Acute Spinal Cord Injury of Dorsal Spine

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Abstract

Object: Extradural decompression supplemented with stabilization of the injured vertebral segment is the gold standard for treating traumatic spines. Any effective surgical modality should not only be undoing the reversible harm caused by the primary cord injury but should also be contributing to reduce the potential deleterious effects of the secondary cord injury including the increased intra-thecal Pressure (ITP) inside relatively non-compliant intact dura. ITP has been reported to be increased after decompression but distal to the site of injury which actually may reduce vascular perfusion of the cord and be negating the likely benefits of the performed surgery. The objective of the present study was to evaluate the effect of surgical decompression on ITP both proximal and distal to the level of injury and exploring decompression's true efficacy in acutely injured thoracic spinal cord injury (SCI).

Methods: The present study comprised of twenty patients of thoracic spine injury presenting within 7 days of injury who underwent surgical decompression and posterior instrumentation. After giving general anaesthesia, all patients were put in prone position on the operating table keeping both the hips flexed to nearly 60 degrees using bolster under torso to open up interlaminar spaces. Lumbar puncture (LP) was performed through L4-5 space using 18 G epidural needle inserting a catheter and connecting it to pressure transducer to record intra thecal pressure. Midline posterior approach was utilized for exposing the injury site. Another epidural needle was inserted percutaneously and manoeuvred through an interlaminar window created through the inter-laminar space proximal to the site of injury. A second catheter was inserted through this needle into the intrathecal space and connected to another pressure transducer and the monitor. Decompression and posterior instrumentation were done in the standard manner.

Both catheters proximal and distal to site of injury recorded the ITP before, during and after the decompression and were maintained for 72 hours.

Results: Surgical decompression produced reduction in ITP proximal to the level of cord injury while it caused no change in ITP distal to the cord injury. Mean ITP proximal to the cord injury before and after decompression was 24.70± 6.78 and 19.10± 4.16. Mean ITP distal to the cord injury before and after decompression were 17.25 ± 5.36 and 17.15 ± 5.03 respectively. No adverse events related to pressure monitoring were noted in any patient.

Conclusions: Surgical decompression in acute SCI not only removes the extradural compression but also restores flow of the cerebrospinal fluid (CSF) and normalizes increased ITP all along the thecal sac. Decreased ITP both proximal and distal to the site of injury

brings improvement in the spinal cord perfusion pressure (SCPP) further enlarging benefits of the surgical decompression.

Abbreviations used in his paper: Intrathecal pressure (ITP), spinal cord perfusion pressure (SCPP), Mean arterial blood pressure (MABP), Lumbar puncture (LP), cerebrospinal fluid (CSF)

Keywords: Intrathecal Pressure, Cerebrospinal Fluid, Spinal Cord Injury, Extradural Decompression.

Introduction

Approximately 250 000 and 500 000 people suffer spinal cord injury (SCI) every year across the globe with more than 17000 new injuries reported annually in USA alone. [1-5] The dreadful and devastating consequences of SCI are attributable to the primary and secondary injury to the cord. Primary spinal cord injury results from the physical disruption of the spinal cord structure at the time of initial injury due to penetrating objects, bone fragments, hematoma, or other mechanisms. [6-12] The body reacts to SCI with complex cellular and extracellular cascades that serve protective and reparative roles but also exacerbate damage and contribute to secondary injury. The secondary mechanism sets in minutes after injury and lasts for weeks or months. [13] The secondary injury involves electrolyte shifts, oedema, and necrotic cell death besides the formation of free radicals, delayed calcium influx, immune system response, inflammation, and apoptotic cell death etc. [14-16]

While decompression of the extradural elements is the primary focus in the management of patients with a clinical spinal cord injury, little attention has been given to the potential deleterious effects of the secondary injury including the expanding volume inside relatively noncompliant intact dura. [17] Moreover, extradural decompression of the spinal cord is described to result in increased intra- and post operative ITP measured caudal to the injury site. [18] This increase in intrathecal pressure however should result in reduced spinal cord perfusion which may add to the insult inflicted by primary injury mechanism. It was postulated that there existed a pressure gradient across the site of injury (obstruction) and decompression results into re-establishment of CSF channel which caused distal ITP to rise though the study did not measure ITP proximal to the injury. Since such reported ITP variations appear to negate and undermine the time-tested benefits of decompression in SCI patients, we proposed to measure and reconfirm increase in ITP in SCI patients before, during and after decompression both proximal and distal to the site of injury. [19]

Methods: The present prospective study comprised of 20 consecutive patients of thoracic spine injury with the following inclusion criteria: 1) ASIA grade A and grade B SCI; 2) SCI between T-3 and T-12; 3) presentation within 7 days of injury; and 4) informed consent to participate in the study. Patients were excluded if suffered with concomitant head injury, chest injury or any major skeletal injury requiring surgical intervention and also if were less than 14 years of age. Clinical trial approval was obtained from the college ethical committee.

All patients underwent routine AP and lateral radiograph of the affected segment followed by detailed neurological examination. MRI of the confirmed

injured segment was carried out to determine the status of spinal cord, degree of compression, and to anticipate the potential problems during decompression. MRI was also utilized to see flow of CSF across the site of injury. After the routine pre-anaesthetic check up including all necessary blood, urine or radiological investigations, patients were taken for elective surgery at the earliest available time.

After giving general anaesthesia, patients were put prone on the operation table. Prone positioning bolsters were used under torso keeping abdomen and chest free for proper intra-operative ventilation. Under strict aseptic condition, lumbar puncture was performed at L3-4 or L4-5 level using 18G epidural needle. A catheter was advanced through the needle and inserted 4-5 cm intrathecally and secured to skin using adhesive tape. After confirming a free flow of CSF, the catheter was connected to the pressure transducer connected to a multichannel patient monitoring system. The transducer was then mounted on the side bar of operating table at the level of right atrium while patient was in prone position for surgery. After zeroing the transducer with atmospheric pressure, the intrathecal pressure was recorded. The height of the transducer with respect to the right atrium of the patient was kept fixed throughout the procedure.

After proper cleaning and draping, posterior midline incision was used. Exposure of the spine was done on either side till transverse process and facet joints. A window was created along inferior border of the lamina through the inter-laminar space proximal to the site of injury. An epidural needle was inserted percutaneously about 2 cm lateral to the midline incision and manoeuvred through the just created inter-laminar window into the underlying dura at the thoracic level. A second catheter was inserted through this needle to a depth of about 2-3 cm into the intrathecal space and fixed to the skin using sutures. This second catheter was then connected to the monitor via another pressure transducer after usual zeroing.

Depending upon the preoperative radiographic evaluation, degree and extent of fracture comminution and canal compromise etc., surgical decompression was performed involving hemi or complete laminectomy with or without facetectomy and partial corpectomy. Spine was stabilized using pedicle screw rod system using 4 screws spanning one above and below the injured level. ITP pressure was monitored continuously throughout the procedure both proximal and distal to site of injury. There was sinusoidal variation in ITP proximal to site of injury both before and after decompression. We took the mean baseline value to represent ITP in each case. Distal ITP showed sinusoidal variation only after decompression and not before. Wound closure was done in layers over gravity dependent drain. Both the catheters were kept in the post-operative period for 48 hours. Proximal catheter was dressed in the surgical wound. Intravenous antibiotics which included, ceftriaxone 1 gm and amikacin 500 mg twice daily was given for 5 days along with diclofenac and ranitidine. Both the catheters were removed after 48 hours using sterile technique checking blue tip to confirm the complete removal without any breakage. Patients were discharged at two weeks.

Results:

Age, Sex and Vertebral Level Distribution:

A total of 20 patients meeting the inclusion criteria were included in the study. The sample consisted of 17 male (85%) and 3 female (15%) patients between 18 to 45 yrs. of age. Vertebral level most commonly affected was D 12 (9) to be followed by D11(4), D5 (3), D9 (2) and one each at D7, D4. Thirteen out of 20 patients were in age group of 25 to 34 years (65%). Most common mechanism of injury was fall from height (65%) followed

by motor vehicular accident (20%) which is in accordance to epidemiological trends in developing countries.26 16 out of 20 patients were ASIA grade A and 4 were ASIA grade B.

Intrathecal Pressure:

The results were analyzed in relation to the effect of surgical decompression on the ITP which is a variable in SCPP or spinal cord vascularity. ITP before and after decompression both distal and proximal to the site injury is shown in Table 1.

Table 1: Mean ITP both proximal and distal to the site of injury before and after decompression

S.No.	ITP (mm Hg) proximal to injury		ITP (mm Hg) distal to injury	
	pre-decompression	post decompression	pre-decompression	post decompression
1	18	17	14	14
2	14	14	17	18
3	34	19	22	24
4	14	15	16	12
5	10	6	8	11
6	24	14	16	16
7	28	18	18	18
8	24	12	9	16
9	22	24	26	16
10	20	23	20	24
11	27	14	17	21
12	22	26	25	16
13	29	27	28	22
14	32	14	11	22
15	28	19	20	24
16	26	19	17	19
17	30	14	18	21
18	27	15	11	25
19	31	15	14	23
20	34	18	18	20

Table 2: Statistical analysis of ITP proximal and distal to injury before and after decompression

	Mean ± SD	Mean difference ± SD	p value
Proximal ITP before decompression	24.70 ± 6.78	-5.60± 4.70	0.0004
Proximal ITP after decompression	19.10 ± 4.16		
Distal ITP before decompression	17.25 ± 5.36	-0.10± 2.46	0.858
Distal ITP after decompression	17.15 ± 5.03		

ITP pressure was monitored continuously throughout the procedure for any variation. There was sinusoidal variation in ITP proximal to site of injury both before and after decompression. We took the mean baseline value to represent ITP in each case. Distal ITP showed sinusoidal variation only after decompression and not before.

ITP proximal to site of injury demonstrated measurable fall after surgical decompression in 16 of 20 patients while marginal insignificant increase in ITP was seen in 4 patients. Mean proximal ITP pressure before decompression was 24.70± 6.78 which after decompression fell down to 19.10 ± 4.16. The mean distal ITP before and after decompression was 17.25 ±5.36 and 17.15 ± 5.03 respectively. Fall in the ITP proximal to site of injury with decompression was statistically significant (P value 0.0004) while change in the ITP distal to the site of injury was not statistically significant (P value 0.858). (Table 2)

Complications-

Adverse events related to pressure measurement both proximally and distally were closely monitored. Intra-operatively, catheter insertion at both the sites did not lead to any change in pulse or blood pressure. There was no drop-in saturation at any point in time and all patients remained stable throughout the procedure. In the post-operative period, all patients were carefully checked with respect to the mild and self-limiting complications like persistent headache, nausea and vomiting,11 decreased vision, abducens nerve palsy,12 and tinnitus and severe complications like abnormal mentation, pupillary asymmetry, decerebrate posturing or pneumonia or deep vein thrombosis etc.1,14,25 No CSF leakage, probe dislodgement, pseudo-meningocoele, probe associated haematoma or meningitis etc.9,17 was seen in any patient as has been reported.

Discussion

Traumatic spinal cord injury is associated with significant morbidity and mortality. Surgeons over the years have had tough time tackling this difficult clinical situation. Many treatment modalities have been tried with very limited success. Extradural decompression supplemented with stabilization of the injured vertebral segment is the gold standard for treating such injuries. It helps not only undoing the reversible harm caused by the primary cord injury but is described to reduce the secondary cord injury also. [20]

Spinal cord perfusion pressure is one of the important outcome measure to affect functional recovery of the injured spinal cord and is calculated as difference between the MABP and ITP. [21] Most spinal injury care centres try to maintain MABP in the higher normal range to improve the SCPP ignoring the ITP completely. [22-25] CSF drainage has been the established way to reduce ITP in thoraco-abdominal aortic aneurysm surgery and has been suggested for treating SCI patients too. [26] Devastating complications like intracranial haemorrhage following CSF drainage or subdural intracranial haemorrhage if too large a volume of CSF is drained over a short period of time are deterrents to such a therapeutic approach in treating SCI patients.15,20,22 However CSF drainage was undertaken in treating SCI patients in a small randomized controlled trial.16 The limitations were a small sample size, broad inclusion criteria, lack of statistical power calculation and restricted drainage regimen (maximum 10 mL per hour). [27]

Same study also noted that ITP distal to the site of injury increased after decompression which authors reported as great surprise and contrary to their expectations. They commented that such increases in ITP lower SCPP and therefore may add to the contribution that parenchymal ischemia makes in the pathophysiology of secondary damage

after acute SCI. If one believes these observations truly to exist, the increased ITP after the surgical decompression would actually hamper recovery of the injured spinal cord thus downgrading and challenging the final benefit and credibility of the surgical decompression. [28]

They hypothesized a large pressure gradient across the occluded thecal sac at the injury site (with a very high-pressure system rostral and a low- pressure system caudal to the injury), and speculated that the apparent increase in distal ITP could actually be denoting the reduced ITP of the highly increased proximal ITP. These comments however without measuring ITP proximal to the injury site poses big question to such an interpretation. [19]

The present study evaluated ITP variations both proximal and distal to the site of injury intraoperatively before and after decompression. Pre-decompression ITP readings demonstrated that ITP proximal to the site of injury was higher (Mean 24.70 ± 6.78) than ITP distal to the site of injury (Mean 17.25 ± 5.36) in all patients. This pressure gradient across the site of injury could be explained by the possible obstruction to the flow of CSF due to the displaced osseo ligamentous elements, hematoma or cord edema. [30]

After decompression, ITP proximal to the site of injury reduced significantly (Mean 19.10 ± 4.16) though was still higher than ITP distal to the site of injury which remained nearly unaltered even after the decompression. Our findings are contrary to the earlier observation that ITP increases after the decompression both intra- and post operatively when measured caudal to the injury site. In such an event, surgical decompression resulting in increase in ITP would in turn reduce vascular perfusion of the cord and shall act deleterious to patient's clinical outcome than doing any benefit. This is the first study to have studied ITP proximal to the injury site and report reduced ITP after decompression both proximal and distal to the site of injury. [31]

Post decompression mean ITP proximal to site of injury was measured to be 19.10 ± 4.16 while it was 17.15 ± 5.03 distal to site of injury. These observations point out existence of a pressure gradient between any two sites of the thecal sac despite restoration of continuous flow showing ITP measurements with sinusoidal variations. From the primary site of secretion in the choroid plexus, CSF flows throughout the ventricular system of the brain and sub arachnoid space (SAS) in spinal canal.23,28 The high-pressure pulse waves and velocity of the CSF in the cranium and cervical SAS are reported to diminish significantly with caudal flow, with little or no flow in the relatively capacious SAS of the lumbar cistern.18 It is logical to have lesser ITP as the site of measurement moves away from the primary source of secretion. Pressure gradient between two ends of the restored unified column therefore is natural and rather essential to maintain the caudal flow in SAS. Not much change in ITP was seen in post decompression measurements at 24 or 48 hours at both proximal and distal to the site of injury. [32]

Introducing a catheter proximal to the site of obstruction was feared with complication of neuroherniation.16 In our study we inserted catheter in proximal part in all patients and recorded ITP both before and after decompression. Intraoperatively all the patients remained stable. There wasn't any episode of hemodynamic disturbance nor of fall in oxygen saturation. In the postoperative period, all the patients were examined for both mild and self-limiting complications like persistent headache, nausea and vomiting,11 decreased vision, abducens nerve palsy,12 and tinnitus and severe complications like abnormal mentation, pupillary asymmetry, and decerebrate posturing etc. [33] All the patients fared well without any complication pertaining

to proximal catheter insertion. Thus, through this study we support the safety of proximal catheter insertion for monitoring of ITP. This proximal ITP monitoring can also be useful adjunct in knowing the adequacy of decompression. [34]

Conclusions: A prospective study was conducted in acute ASIA grade A and B SCI patients in thoracic spine to evaluate the role of surgical decompression in preventing or negating the effects of secondary cord injury. ITP was measured in all patients before, during and after decompression both proximal and distal to the site of injury. Surgical decompression in acute SCI not only removed the extradural compression but also restored flow of the CSF and normalized increased ITP all along the thecal sac. Decreased ITP both proximal and distal to the site of injury was seen to bring improvement in the SCPP further enlarging benefits of the surgical decompression.

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