Efficacy and Mechanisms of Microscopic Intra- and Extramedullary Decompression and Irrigation Therapy for Chronic Cervical Spinal Cord Injury: Postprint

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Abstract

Objective: To investigate the clinical efficacy and possible mechanisms of intramedullary and extramedullary decompression combined with irrigation therapy for chronic cervical spinal cord injury. Methods: We followed up 57 patients with chronic cervical spinal cord injury in our hospital from January 2008 to January 2015. The etiologies included multilevel cervical disc herniation in 17 cases, long-segment ossification of the posterior longitudinal ligament (OPLL) in 25 cases, hypertrophy or calcification of cervical ligamentum flavum in 13 cases, and old cervical fracture in 2 cases. Thirty-one patients who underwent simple extramedullary decompression were designated as Group A, treated with posterior single-door laminoplasty, spinal canal decompression, and canaloplasty. Twenty-six patients who underwent intra- and extramedullary decompression were designated as Group B, treated with posterior single-door laminoplasty, dural incision and decompression, adhesion release, large-volume saline irrigation, and canaloplasty. In this group, cerebrospinal fluid was collected at the injury site after opening the dura mater and releasing adhesions for inflammatory factor level testing. During follow-up, Japanese Orthopaedic Association (JOA) scores were regularly assessed, preoperative and postoperative JOA scores were recorded periodically, and improvement rates were calculated, while X-ray, CT, or MRI were re-examined. Results: Imaging at 2 weeks postoperatively indicated that primary lesions were well resolved in both groups, with Group B showing better improvement in spinal cord high signal intensity than Group A. The JOA scores at each time point within 1 year postoperatively were higher than preoperative values in both Groups A and B ($P<0.05$). Comparison between the two groups showed that Group B's JOA scores and improvement rates at each time point within 1 year postoperatively were higher than Group A' s,

with statistically significant differences between groups (P<0.05). Preoperative JOA scores showed no statistical difference between the two groups (P>0.05), indicating that Group B achieved better short-term efficacy than Group A within 1 year postoperatively. In Group A, 3 cases developed cerebrospinal fluid leakage (all OPLL patients), 1 case developed epidural hematoma, and 1 case developed internal fixation loosening. In Group B, 1 case developed door hinge fracture. Group B had shorter postoperative recovery time than Group A, entering a plateau phase at 3 months. Cerebrospinal fluid test results showed that pro-inflammatory factors IFN- , IL-17F, IL-6, and sCD40L levels at the spinal cord injury site were significantly higher than normal cerebrospinal fluid, with IL-6 showing the most significant increase. Comparisons of increased pro-inflammatory factors with normal values were statistically significant in each group (P<0.05). Conclusion: For chronic cervical spinal cord injury, intra- and extramedullary decompression under microscope can achieve better efficacy than simple extramedullary decompression, which may be related to dural incision, adhesion release, relief of chronic compression, and reduction of inflammatory factor levels at the injury site under microscopy. Altering the local microenvironment can promote neurological function recovery, shorten the time to plateau phase, thereby improving functional outcomes and achieving better clinical efficacy.

Full Text

Efficacy of Intramedullary and Extramedullary Decompression and Lavage Therapy under Microscope for Treatment of Chronic Cervical Spinal Cord Injury

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Abstract

Objective: To investigate the clinical efficacy of combined intramedullary and extramedullary decompression with lavage therapy for chronic cervical spinal cord injury and explore its potential mechanisms.

Methods: Fifty-seven patients with chronic cervical spinal cord injury treated at our hospital between January 2008 and January 2015 were enrolled and followed up. The etiologies included multilevel cervical disc herniation (17 cases), long-segment ossification of the posterior longitudinal ligament (OPLL) (25 cases), hypertrophy or calcification of the cervical ligamentum flavum (13 cases), and old cervical fractures (2 cases). Thirty-one patients who underwent simple

extramedullary decompression via posterior single-door laminoplasty were designated as Group A, while 26 patients who underwent combined intramedullary and extramedullary decompression via posterior single-door laminoplasty with dural incision, adhesion release, and extensive saline lavage were designated as Group B. In Group B, cerebrospinal fluid (CSF) samples were collected at the injury site for inflammatory factor analysis. All patients were regularly followed up with JOA scoring, and preoperative and postoperative scores were recorded to calculate improvement rates. Radiographic examinations (X-ray, CT, or MRI) were performed periodically.

Results: Imaging at 2 weeks postoperatively showed satisfactory decompression of the primary lesions in both groups, with Group B demonstrating better resolution of intramedullary high signal intensity compared to Group A. The JOA scores at all time points within 1 year postoperatively were significantly higher than preoperative values in both groups ($P<0.05$). Group B exhibited higher JOA scores and improvement rates than Group A at all postoperative time points, with statistically significant intergroup differences ($P<0.05$). Preoperative JOA scores showed no significant difference between groups ($P>0.05$), indicating that Group B achieved better short-term outcomes. In Group A, 3 patients developed CSF leakage (all OPLL cases), 1 developed epidural hematoma, and 1 experienced internal fixation loosening. Group B had 1 case of hinge fracture. Group B demonstrated shorter recovery time, entering a plateau phase at 3 months. CSF analysis revealed significantly elevated levels of pro-inflammatory factors IFN- , IL-17F, IL-6, and sCD40L at the injury site compared to normal CSF, with IL-6 showing the most pronounced increase ($P<0.05$ for all comparisons).

Conclusion: For chronic cervical spinal cord injury, combined intramedullary and extramedullary decompression under microscopy yields superior clinical outcomes compared to simple extramedullary decompression. This may be attributed to dural incision and adhesion release under microscopy, which not only relieves chronic compression but also reduces local inflammatory factor levels. Modifying the local microenvironment can promote neurological recovery, shorten the time to plateau phase, and thereby improve functional prognosis and clinical efficacy.

Keywords: microscope; spinal cord injury; inflammatory factors; intramedullary decompression

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Introduction

Spinal cord injury (SCI) is a severe neurological condition with poor prognosis, characterized by high incidence, high disability rates, and substantial healthcare costs, representing a global medical challenge [1]. Based on disease duration, SCI can be classified as acute or chronic. While surgical treatment combining intramedullary and extramedullary decompression for acute SCI has been

reported to achieve certain clinical efficacy in China [2-3], its application to chronic cervical SCI caused by degenerative diseases remains under investigation, with limited in-depth studies and unclear mechanisms [4]. With societal aging, the incidence of cervical degenerative diseases is increasing [5-6], leading to a growing patient population with chronic cervical SCI. This presents an urgent challenge for spine surgeons to develop more effective treatment approaches.

Between January 2008 and January 2015, our hospital treated 57 patients with chronic cervical SCI, who were randomly divided into two groups based on surgical approach: Group A underwent simple extramedullary decompression, while Group B underwent combined intramedullary and extramedullary decompression under microscopy. In Group B, CSF was collected at the lesion site for inflammatory factor analysis [7-8]. Our observations indicated that Group B achieved superior outcomes compared to Group A, possibly related to dural incision, adhesion release, relief of chronic compression, and reduction of local inflammatory factors under microscopy. The detailed report follows.

1.1 Inclusion and Exclusion Criteria [9]

Inclusion criteria: (1) Primary diagnosis of chronic SCI (disease duration >3 months); (2) American Spinal Injury Association (ASIA) impairment scale grades A-D [10]; (3) No significant progression after >2 months of conservative treatment; (4) Abnormal high signal intensity on preoperative MRI T2WI (edema or necrotic foci); (5) Multilevel cervical disc herniation or OPLL involving 3 segments.

Exclusion criteria: (1) Penetrating SCI or complete transection; (2) Major spinal fracture within 2 months; (3) Infectious SCI; (4) Severe cardiopulmonary disease precluding surgery; (5) Concurrent brain or other neurological diseases.

Patient Characteristics

The 57 patients included 38 males and 19 females, aged 30-68 years, with disease duration ranging from 3 months to 10 years. Ninetety patients experienced symptom exacerbation after minor trauma. Etiologies included multilevel cervical disc herniation (17 cases), long-segment OPLL (25 cases), cervical ligamentum flavum hypertrophy or calcification (13 cases), and old cervical fractures (2 cases). Clinical manifestations included sensory deficits (limb numbness, hypoesthesia, band-like sensation) in 20 cases, motor weakness (limb weakness, gait instability) in 38 cases, urinary retention in 5 cases, and dysuria in 16 cases. Physical examination revealed positive Hoffmann' s sign, hyperactive knee and ankle reflexes, and positive ankle clonus in most patients, with 32 cases showing sensory abnormalities below the injury level. Preoperative imaging (cervical DR, CT) demonstrated degenerative changes including osteophyte formation, spinal stenosis, ligamentum flavum calcification, or intraspinal fracture fragments. MRI T2WI showed abnormal high signal intensity at the lesion level

with or without disc pathology (Figure 1 [Figure 1: see original paper]).

Surgical Methods

Patients were divided into Group A (n=31) and Group B (n=26). In Group A, etiologies included multilevel cervical disc herniation (9 cases), long-segment OPLL (15 cases), ligamentum flavum hypertrophy/calcification (6 cases), and old fracture (1 case). In Group B, etiologies included multilevel disc herniation (8 cases), long-segment OPLL (10 cases), ligamentum flavum hypertrophy/calcification (7 cases), and old fracture (1 case). Preoperative JOA scores were 5.93±1.06 and 6.34±0.38 for Groups A and B, respectively. All patients underwent preoperative and postoperative cervical DR, CT, and MRI examinations, and received 1000 mg methylprednisolone intravenously 30 minutes before decompression to prevent reperfusion injury.

Group A underwent posterior single-door laminoplasty with spinal canal decompression. Group B underwent posterior single-door laminoplasty with dural incision, local decompression, adhesion release, extensive ice-cold saline lavage, and spinal canal reconstruction. In Group B, after dural incision and adhesion release, CSF specimens were collected using a 5 mL syringe at the lesion site to measure pro-inflammatory factors IFN- , IL-17F, IL-6, and sCD40L [8, 11]. The criteria for adequate intramedullary decompression included restoration of spinal cord pulsation, free CSF flow, and obvious dilation of pial surface vessels (Figure 2 [Figure 2: see original paper]).

1.4 Follow-up and Statistical Analysis

Patients were followed up at 1, 6, and 12 months postoperatively. Neurological function was evaluated using the Japanese Orthopaedic Association (JOA) scoring system [12] to assess bone fusion and neurological recovery. JOA scores were recorded and improvement rates calculated. Statistical analysis was performed using SPSS 20.0 software. Multivariate repeated measures ANOVA was used, with paired t-tests comparing postoperative JOA scores and improvement rates with preoperative values at each time point. Intergroup differences were analyzed using two-sample t-tests, with $P<0.05$ considered statistically significant. The JOA scoring system has a maximum of 17 points: 4 points each for upper and lower limb function, 6 points for sensory function, and 3 points for bladder function. Neurological improvement rate = (postoperative score - preoperative score) / (17 - preoperative score) × 100%. CSF specimens were analyzed using liquid chip technology to detect four pro-inflammatory factors: IFN- , IL-17F, IL-6, and sCD40L [13]. SPSS 20.0 was used for variance homogeneity testing and F-test analysis of inflammatory factors compared to normal values, with $P<0.05$ considered statistically significant.

2.1 Imaging Findings

Postoperative imaging at 2 weeks showed satisfactory decompression of primary lesions in both groups. Group B demonstrated better resolution of abnormal intramedullary high signal intensity compared to Group A, with complete resolution in some cases requiring up to 3 months (Figure 3 [Figure 3: see original paper]).

2.2 JOA Scores and Improvement Rates

JOA scores at all postoperative time points were significantly higher than preoperative values in both groups ($P<0.05$). Group B showed higher JOA scores than Group A at all postoperative time points, with statistically significant intergroup differences ($P<0.05$). Preoperative JOA scores showed no significant difference between groups ($P>0.05$). Improvement rates at 1, 6, and 12 months were 39.98%, 47.45%, and 52.33% in Group A, and 52.18%, 58.38%, and 61.52% in Group B, respectively, with statistically significant intergroup differences ($P<0.05$). Both groups showed efficacy, with Group B demonstrating superior short-term outcomes (Table 1).

2.3 Complications

In Group A, 3 patients developed CSF leakage (all OPLL cases) that healed with conventional management, 1 developed epidural hematoma requiring reopening for hemostasis, and 1 experienced internal fixation loosening. Group B had 1 case of hinge fracture requiring revision and fixation.

2.4 Inflammatory Factor Analysis

Analysis of the four inflammatory factors revealed significantly elevated levels of pro-inflammatory cytokines IFN- , IL-17F, IL-6, and sCD40L in CSF at the chronic SCI site compared to normal CSF, with IL-6 showing the most prominent increase ($P<0.05$ for all comparisons) (Figure 4 [Figure 4: see original paper]).

3.1 Etiology and Pathogenesis of Chronic Cervical SCI

Chronic cervical SCI most commonly results from cervical degenerative diseases. However, untreated or inadequately treated cervical fractures with residual bone fragments causing persistent spinal cord compression also represent a significant cause [14-15]. Chronic SCI involves two sequential processes: primary (mechanical) and secondary (cellular) injury, leading to structural damage of neural tissue and corresponding neurological deficits. The primary injury results from compression by herniated discs, osteophytes, calcified soft tissue, or old bone fragments disrupting spinal canal integrity. Numerous studies have shown that this mechanical process can be effectively relieved by extramedullary surgical decompression. Secondary injury involves a cascade of physiological and biochem-

ical changes including oxidative stress, inflammatory responses, and excitatory amino acid release [16], causing autodestructive changes in surrounding tissues that deepen and expand the injury [17-18]. This secondary process represents an important mechanism exacerbating neurological dysfunction [19].

3.2 Superior Efficacy of Combined Decompression

Studies have demonstrated that surgical decompression significantly improves functional recovery in chronic SCI [20]. Our comparison of Groups A and B revealed that Group B achieved higher JOA scores at all time points ($P<0.05$), with improved scores at 12 months postoperatively compared to preoperative values, indicating better short-term outcomes with combined decompression. The main reasons include: (1) SCI involves compression from both bony structures and the dural sac, increasing intramedullary and extramedullary pressure —only dural incision can truly reduce intramedullary pressure; (2) Chronic SCI from degenerative disease or old fractures differs from acute SCI [2, 21], with longer disease duration and entry into a plateau phase, typically without significant hemorrhage or edema [22], but rather showing organized hematoma, tissue necrosis, dural adhesions, CSF blockage, and venous obstruction—confirmed intraoperatively. Microscopic release of dural adhesions and restoration of CSF flow and circulation better facilitates neurological recovery; (3) Intraoperative lavage reduces secondary injury from local chronic inflammation [23-24]. Inflammation has dual effects in SCI—while moderate inflammatory responses represent normal physiological vascular reactions and macrophages can clear necrotic tissue, excessive responses amplify inflammatory cascades, damage neurons and myelin, induce apoptosis and axonal degeneration, and impede neural repair and regeneration [11, 21].

All four measured inflammatory factors—IFN- , IL-17F, IL-6, and sCD40L—are pro-inflammatory cytokines [26-27]. Their elevated concentrations in lesion-site CSF compared to normal CSF ($P<0.05$) demonstrate persistent local chronic inflammation even in chronic SCI. Progressive chronic inflammation exacerbates subdural adhesions, which further compress the spinal cord, causing ischemic necrosis, myelomalacia, and syrinx formation [19, 23], creating a vicious cycle. While numerous drugs and cytokine inhibitors have been investigated for SCI-related inflammation, their mechanisms and protocols remain unconfirmed [18]. Although some agents show efficacy in animal models, human results are limited. Corticosteroids, long used for acute SCI, are rarely applied in chronic SCI due to side effects and uncertain efficacy. Therefore, surgical reduction of local inflammatory factors to improve the regenerative microenvironment represents the only effective approach to reduce secondary injury [28-29].

3.3 Indications and Contraindications for Intramullary Decompression

We propose the following indications: patients with varying degrees of SCI (ASIA grades A-D); MRI evidence of long-segment spinal cord compression

with abnormal intramedullary high signal; clinical signs consistent with SCI level; lack of improvement after >3 months of conservative treatment or previous extramedullary decompression; and primary pathologies requiring posterior approach surgery. Based on literature review and our clinical experience [30-31], contraindications include penetrating SCI, infectious spinal diseases, and neurological disorders from other medical conditions.

3.4.1 Surgical Technique

Under general anesthesia with endotracheal intubation, patients were placed prone. The lesion level was confirmed fluoroscopically. A posterior midline longitudinal incision was made, with paraspinal muscle dissection to expose the spinous processes, laminae, lateral masses, and pedicles. The less symptomatic side served as the hinge. High-speed burr or ultrasonic bone scalpel was used to open the lamina on the more compressed side, with partial fracture of the hinge-side lamina to open the spinal canal and remove compressive lesions. After removing portions of the opened lamina to fully expose the lesion area (intramedullary high signal region), the dura was longitudinally incised under microscopy and suspended with 0-silk sutures. A nerve dissector was used to release adhesions between dura and arachnoid, with gradual CSF release following separation of the arachnoid (CSF jet was observed in some high-pressure cases). CSF samples were collected for analysis, followed by extensive ice-cold saline irrigation. Adequate decompression was confirmed by restoration of spinal cord pulsation, free CSF flow, and dilation of pial vessels. The dura was closed continuously, the opened lamina was fixed with plates or lateral mass screws, and bone grafting was performed on the hinge side. After hemostasis, a drainage tube was placed and the incision closed.

3.4.2 Timing of Surgery

We believe that for chronic SCI from either old fractures or degenerative causes, surgery should be performed as early as possible if conservative treatment is ineffective and neurological deficits persist, as prolonged disease duration worsens subdural adhesions and reduces spinal cord microcirculation, increasing neurological damage. Patients with acute exacerbation after minor trauma should ideally undergo surgery within 12 hours. Batchelor et al.' s meta-analysis [32] suggests stable benefits from decompression within 12 hours post-injury, with relatively less benefit between 12-24 hours. Carlson et al.' s animal study [33] found the window for functional recovery in adult beagles was 3-6 hours post-injury. For patients with progressive onset and disease duration >1 month, even if plateau phase has been reached, surgery within 12 months remains optimal if compression, ischemia, and inflammatory injury persist with MRI-confirmed pathological changes—consistent with other researchers' views [34].

3.4.3 Criteria for Successful Decompression

Based on literature review and our experience, three criteria indicate adequate decompression: (1) restoration of spinal cord pulsation, (2) free CSF flow, and (3) obvious dilation of pial surface vessels.

3.4.4 Complication Prevention

Microscopic intramedullary decompression is a delicate procedure that avoids severe complications common in simple extramedullary or simple dural incision decompression (e.g., iatrogenic SCI, CSF leakage). The most common complication is hinge fracture, with occasional CSF leakage. Gentle technique without rough or repetitive movements generally prevents these. Hinge fracture causing spinal cord compression requires revision and fixation. CSF leakage requires enhanced wound care to prevent infection, with monitoring of blood counts, albumin, ESR, and electrolytes, and appropriate timing of drain removal. If CSF leakage persists for 3-5 days postoperatively, drain removal with gentle compression or lumbar drainage can prevent sinus formation [9], and CSF leakage generally heals.

3.5 Study Limitations

(1) Due to poor patient compliance, this study only compared short-term outcomes at 12 months postoperatively. Long-term neurological improvement beyond 1 year could not be evaluated, necessitating further multicenter studies on long-term efficacy to guide clinical practice.

(2) This study measured four common inflammatory factors in the intramedullary decompression group, confirming persistent local chronic inflammation in chronic SCI and demonstrating that dural incision reduces local inflammatory factors and secondary injury. This provides evidence for the superior outcomes in Group B. However, to avoid iatrogenic injury, invasive CSF sampling was not performed in the extramedullary decompression group.

Conclusion

For patients with confirmed chronic cervical SCI who meet surgical indications and have no contraindications, early surgical intervention is recommended. The preferred approach is microscopic dural incision decompression with adhesion release and ice-cold saline lavage, which achieves better clinical outcomes than simple extramedullary decompression. This may be related to relief of adhesion-induced secondary compression and reduction of chronic inflammatory responses mediated by local inflammatory factors. While this procedure is technically demanding with certain risks and complications, strict adherence to indications/contraindications and mastery of microsurgical techniques through appropriate learning curves can prevent procedure-related spinal cord injury.

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Note: Figure translations are in progress. See original paper for figures.