

Percutaneous decompression combined with basivertebral nerve radiofrequency ablation for intractable pain in brucellar spondylitis: a retrospective cohort study

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Abstract

Background

Brucellar spondylitis frequently causes intractable low back pain that responds poorly to conventional drug therapy. This study aimed to evaluate the clinical efficacy of percutaneous decompression combined with basivertebral nerve radiofrequency ablation for intractable pain in lumbar brucellar spondylitis.

Methods

A retrospective cohort study included 54 patients with lumbar BS treated from January 2022 to June 2024, allocated into three groups (n = 18 each): drug-only (Group A), decompression plus drug (Group B), and decompression plus RFA plus drug (Group C). Perioperative indicators, intravertebral pressure, inflammatory cytokines, pain relief, complications, and clinical efficacy were compared.

Results

Surgical groups (B, C) showed superior VAS and ODI improvement compared to Group A ($P < 0.05$), with Group C outperforming Group B at immediate, 1-week, and 3-month follow-ups ($P < 0.05$). Group C exhibited lower postoperative bone marrow TNF- α and IL-6 than Group B ($P < 0.05$). Both surgical groups demonstrated significant intravertebral pressure reduction ($P < 0.05$). Among 36 patients with pressure measurement, pressure reduction did not correlate with VAS reduction (Pearson $r = -0.09$, $P = 0.60$).

Conclusions

Percutaneous decompression combined with RFA is a minimally invasive, safe, and effective strategy for rapid pain relief in lumbar BS-associated intractable pain, targeting both mechanical and neuroinflammatory pain drivers.

Introduction

Brucellar spondylitis (BS) is an infectious spondylitis caused by Brucella infection, with an incidence ranging from 2% to 60% [1–2]. It most commonly affects the lumbar spine, followed by the thoracic and cervical regions [3–4]. Its main symptoms include low back pain radiating to both hips, with more than 55% of patients experiencing severe pain [5]. Notably, while drug treatment effectively controls infection [6], it fails to rapidly alleviate persistent low back pain in refractory cases. Approximately 60% of BS patients continue to experience moderate to severe pain post-infection control, significantly impairing quality of life [7]. While surgical intervention can rapidly alleviate lumbar pain symptoms, it carries significant risks including extensive trauma, high likelihood of spinal cord or nerve injury, and multiple

complications. Consequently, there is a need to explore minimally invasive treatment approaches capable of providing early, rapid pain relief to enhance patients' quality of life and shorten treatment duration.

The superior and inferior endplates of the vertebral body, being richly supplied with blood vessels, become the primary sites of initial invasion and retention by *Brucella* bacteria. When these endplates are infected by *Brucella*, discitis and vertebral osteomyelitis develop, causing severe pain characteristic of vertebral-origin pain [8]. Research indicates that non-infectious vertebral-origin lumbar pain correlates with increased intravertebral pressure, vertebral degeneration, chronic aseptic inflammatory irritation of the endplates, and pain transmission via the vertebral basilar nerves innervating the vertebral body [9, 10]. Radiofrequency ablation combined with percutaneous vertebroplasty (PVP) or percutaneous kyphoplasty (PKP) can effectively alleviate pain in fractured vertebrae and enhance spinal stability. However, clinical benefits are limited for patients with spinal cord or nerve root compression [11]. Treatment using percutaneous vertebral base nerve radiofrequency ablation can effectively improve pain and functional impairment [12]. This method is effective primarily because radiofrequency ablation first involves percutaneous puncture of the vertebral body at the site where vertebral base nerves are concentrated, releasing high intraosseous pressure within the vertebral body and reducing the stimulation of vertebral base nerves caused by this high pressure [13–15]. Secondly, radiofrequency ablation disrupts the widespread transmission of pain signals by the vertebral basilar nerves. Considering that BS-induced low back pain is vertebrogenic, while chemical-inflammatory aetiologies have been documented in the literature, physical factors remain unreported. We therefore hypothesise that the mechanism of BS-induced low back pain resembles that of chronic low back pain, anticipating that vertebral decompression combined with percutaneous vertebral radiofrequency ablation may rapidly alleviate symptoms. Consequently, this study analysed clinical data from three treatment modalities for lumbar brucellosis spondylitis: unmodified drug therapy alone; drug therapy supplemented with percutaneous vertebral decompression; and percutaneous decompression combined with radiofrequency ablation, to evaluate the clinical efficacy of these three approaches in managing refractory pain associated with lumbar brucellosis spondylitis. This aims to clarify the analgesic effect of combined percutaneous decompression and radiofrequency ablation in patients with intractable low back pain due to BS, thereby providing novel therapeutic strategies and theoretical support for clinical management of BS.

Materials and Methods

1.1 General Clinical Data

This retrospective study analysed clinical data from 54 patients with single-segment lumbar brucellosis spondylitis treated in our department between January 2022 and June 2024. Among the 54 patients, 33 were male and 21 female, with a mean age of 52.6 ± 11.3 years; Affected segments: L3 (18 cases), L4 (21 cases), L5 (15 cases). Mean disease duration was 14.3 ± 5.7 months. All patients completed follow-up, with a mean follow-up period of 6.8 ± 1.2 months. This study protocol was approved by the Ethics

Committee of Zhangye People's Hospital Affiliated to Hexi University (ZY20230107), and all patients signed informed consent forms.

1.2 Inclusion and exclusion criteria

1.2.1 Inclusion Criteria

(1) Complete clinical documentation; (2) Compliance with the expert consensus and diagnostic criteria for brucellosis-associated spondylitis [16] ; (3) Early-stage, treatment-naïve or treatment-failure cases of BS; (4) Chronic cases with acute exacerbations, having undergone standard treatment for ≥ 2 weeks; (5) Cases with intractable pain due to disc inflammation, requiring oral analgesics $\geq 2-3$ times daily with minimal pain relief and VAS ≥ 6 points; (6) Vertebral destruction $< 1/3$, cavitation ($< 2\text{cm}$), paravertebral abscess ($< 2\text{cm}$) ; (7) Lesions affecting a single motion unit (not exceeding two vertebrae) with preserved spinal stability; (8) Minimal intravertebral abscesses or inflammatory granulomas without spinal cord, cauda equina, or nerve root compression.

1.2.2 Exclusion Criteria

(1) Uncertain diagnosis or concomitant infectious or neoplastic diseases; (2) Vertebral destruction $> 1/3$, cavitation ($> 2\text{cm}$), paravertebral abscess ($> 2\text{cm}$); (3) Patients with lesions involving multiple segments; (4) Follow-up duration < 6 months or incomplete follow-up data; (5) Concurrent severe underlying medical conditions.

1.3 Grouping and Pharmacotherapy

Patients were categorised by treatment modality: Conventional drug therapy group (Group A, 18 cases), Percutaneous decompression + drug therapy group (Group B, 18 cases), Percutaneous decompression with radiofrequency ablation+ drug therapy group (Group C, 18 cases). All patients received standard dual anti-brucellosis medication for 2 weeks preoperatively (Rifampicin 0.6 g/day; Doxycycline 0.2 g/day), alongside symptomatic treatment including antipyretics, analgesics, and hepatoprotective agents. Surgical intervention may be considered when follow-up tests show an erythrocyte sedimentation rate $\leq 30\text{mm/h}$ and C-reactive protein $\leq 45\text{mg/L}$ with a downward trend. A six-week course constitutes one treatment cycle, with a minimum treatment duration of six months. The treatment regimen may be adjusted or discontinued based on clinical presentation, laboratory findings (normalisation of ESR and CRP), and imaging assessment. Treatment cycles may be extended to 18 months if necessary.

1.4 Preoperative Examination and Assessment

All patients undergo preoperative Rose Bengal plate agglutination test (RBPT). Preoperative assessments include the Oswestry Disability Index (ODI), Visual Analogue Scale (VAS) for lumbar pain, C-reactive protein (CRP), erythrocyte sedimentation rate (ESR), and peripheral serum TNF- α and IL-6. Lumbar spine X-ray, CT, and MRI examinations were performed to confirm the responsible segment and extent of pathology.

1.5 Intravertebral pressure measurement equipment, calibration of accuracy and precision of pressure measurement devices, connection methods

1.5.1 Measurement Equipment

Pressure sensor (Sainuo Measurement & Control SCYG410, range 0–30 kPa, 24V supply, 4-20mA output, accuracy: $\pm 0.1\%$, connection: G1/8 external thread); Analogue Data Acquisition Module DAM3158A (Beijing Altair Technology), G1/8 internal thread Luer connector (Huataiyu Electronics), 15G straight-tip needle, length 150mm, outer diameter 1.78mm, inner diameter 1.3mm (Jiangsu Suzhou Hi-Tech Zone Shishan Jiaqi Trading), three-way switch $\times 1$, Sterile syringe 10ml $\times 1$, Saline solution 100ml $\times 1$ bottle, PKP percutaneous cannula (Shanghai Kailite) and sealing cap (Jiangsu Keyuan), Inflation valve (Ningbo Xingxin Gas Equipment Co., Ltd.), Constant pressure gauge (Ningbo Xingxin Gas Equipment Co., Ltd.).

1.5.2 Accuracy Verification

Take a 1000ml graduated cylinder (specification: 1000ml, height 440mm, inner diameter 68mm) and fill with approximately 500ml of deionised water. Position the fully assembled pressure measurement apparatus at the 100ml mark on the cylinder base. Once the liquid surface stabilises and the pressure gauge reading remains constant, record the liquid level height and the pressure gauge reading. Inflate a latex balloon to a constant pressure of 5–30 kPa (approximately 50–300 mm Hg) using a filling valve with a constant pressure gauge. Secure the puncture site with medical tape to prevent balloon rupture. Subsequently, puncture the balloon using the connected pressure measurement and puncture apparatus, reading the maximum pressure displayed on the pressure gauge as the measurement value.

1.5.3 Connection Method

Place the cryogenic plasma sterilised pressure sensor, Luer connector, straight-tip puncture needle, sterile three-way connector, and sterile syringe on the surgical instrument table. Connect as illustrated, inject saline equivalent to the volume of the pressure measurement circuit (straight-tip puncture needle, three-way connector, and continuous sensor circuit), expel air, and clamp the three-way connector for standby. a PKP Preoperative Preparation and Pressure Measurement Device Connection b PKP Intravertebral Pressure Measurement Screen During Surgery Computer PC Pressure Display Software Interface: Intraosseous Pressure Recording Area and Pressure Curve Display Area.

1.6 Surgical Procedure

Intravertebral Pressure Monitoring: The patient is positioned prone. Following routine disinfection and draping, perform local infiltration anaesthesia with 5-10ml of 2% lidocaine at the marked puncture site, covering the skin, subcutaneous tissue, fascia, and the area surrounding the pedicle. Under C-arm fluoroscopic guidance, locate the injured vertebra and enter laterally through the pedicle shadow (at the 1–2 or 4–5 o'clock

position). When lateral fluoroscopy confirms the needle tip has passed the posterior vertebral margin, and the needle tip does not extend beyond the medial border of the pedicle shadow on anteroposterior fluoroscopy, connect the pressure sensing system. Withdraw the core of the percutaneous cannula, swiftly insert the pressure sensor probe into the cannula, and record the initial intravertebral pressure when fluoroscopy confirms the probe has reached the vertebral midline or the anterior two-thirds of the vertebral body. If the initial pressure exceeds 50 mmHg, employ the "stepwise decompression method": First aspirate approximately 2ml of inflammatory fluid. After a 30-second interval, measure pressure again. If the pressure decrease is < 20%, aspirate a further 1-2ml. Repeat the procedure until the pressure drops below 25mmHg or decreases by $\geq 50\%$. Following simple decompression, irrigate the affected area with 5-10ml of physiological saline.

Bone marrow aspiration from the affected vertebra: Retain 5–8 ml of the aspirated bone marrow for testing TNF- α and IL-6 concentrations. If the aspirate is purulent fluid, concurrent bacterial culture is performed.

Radiofrequency ablation: Insert the radiofrequency ablation electrode through the working channel. Confirm via lateral fluoroscopy that the electrode tip is positioned within the residual lesion area, avoiding the dural sac and nerve roots. Connect the radiofrequency ablation device and set parameters: temperature 70–80°C, power 20–25W, ablation duration 60 seconds per session. Monitor electrode impedance throughout; if impedance suddenly exceeds 300 Ω , pause ablation and inject 0.5ml saline to reduce impedance. Adjust ablation duration and number of sessions based on intraoperative findings. Initiate radiofrequency ablation. During each ablation cycle, enquire whether the patient experiences discomfort such as lower limb numbness or pain, while closely monitoring vital signs. Upon completion of ablation, withdraw the radiofrequency ablation electrode. If no significant active bleeding is observed, reinsert the pressure sensor probe into the percutaneous needle cannula and record the intravertebral pressure within the affected vertebra. Slowly withdraw the working channel sheath. Apply sterile gauze to the puncture site and maintain local pressure with the

palm for 5–10 minutes. After confirming no oozing, disinfect the puncture site again with povidone-iodine, cover with sterile transparent dressing, and apply moderate pressure bandaging to prevent haematoma formation.

Postoperative management: Instruct the patient to maintain strict bed rest for 24 hours postoperatively. Thereafter, ambulation with a lumbar support belt is permitted. Routine antibiotic prophylaxis is administered for 3 days. Bed rest remains the primary regimen for 3 weeks.

1.7 Evaluation Criteria

(1) Surgery-related indicators, intraoperative changes in affected vertebral disc pressure, and postoperative complications; (2) Assess clinical efficacy via VAS pain scores and ODI indices at immediate postoperative, 1-week, 3-month, and final follow-up (> 6 months) assessments; (3) Laboratory tests: Comparison of changes in ESR and CRP at 1 week, 1 month, 3 months, and final follow-up; and

changes in TNF- α and IL-6 levels in pre-treatment peripheral blood, pre-operative bone marrow blood, post-decompression bone marrow blood, peripheral blood at 1 week, and peripheral blood at 3 months; (4) Clinical efficacy evaluation and complications.

1.8 Statistical Analysis

Analysis was conducted using SPSS 25.0 software. Normally distributed quantitative data were expressed as mean $\pm$ standard deviation ($\bar{x} \pm s$). Normally distributed quantitative data underwent one-way analysis of variance (ANOVA); non-normally distributed data used the Kruskal-Wallis H non-parametric test. Categorical data were analysed using chi-square tests (X^2). Pearson correlation analysis was used to assess the relationship between intravertebral pressure reduction and VAS score reduction. $\alpha = 0.05$; $P < 0.05$ indicated statistical significance.

Results

2.1 Perioperative Evaluation Indicators

Complete follow-up data were obtained for all patients, with follow-up exceeding 6 months in all three groups. Preoperative VAS and ODI scores showed no statistically significant differences among the three groups ($P > 0.05$ for all). No significant spinal cord or nerve injury symptoms were observed postoperatively.

2.2 Laboratory Findings Evaluation

Prior to treatment, all patients tested positive in the Rose Bengal plate agglutination test (RBPT). At the final follow-up, all patients tested negative in the RBPT. The ESR and CRP results for patients before treatment, at 1 week, 1 month, 3 months post-treatment, and at the final follow-up are presented in Tables 1 and 2. Results indicated no significant differences between groups before and after treatment ($P > 0.05$). At final follow-up, infection indicators in all groups remained within normal ranges with no statistically significant differences ($P > 0.05$). This demonstrates that vertebral decompression and radiofrequency ablation exerted no appreciable effect on infection control.

Table 1
Comparison of ESR before and after treatment ($\bar{x} \pm s$)

Group	Cases	Before treatment	1 Week	1 Month	3-Month	Last follow-up
Group A	18	43.50 $\pm$ 12.29	37.94 $\pm$ 6.33	25.33 $\pm$ 3.34	15.00 $\pm$ 11.33	4.72 $\pm$ 2.85
Group B	18	46.44 $\pm$ 10.83	38.44 $\pm$ 4.74	25.11 $\pm$ 2.22	13.00 $\pm$ 7.76	4.42 $\pm$ 2.37
Group C	18	47.72 $\pm$ 9.20	37.33 $\pm$ 3.46	23.00 $\pm$ 4.06	10.94 $\pm$ 5.89	4.94 $\pm$ 2.51
<i>F</i>		0.72	0.22	2.75	1.00	0.04
<i>p</i>		> 0.05	> 0.05	> 0.05	> 0.05	> 0.05

Normal ESR range: male: 0–15 mm/h, female: 0–20 mm/h.

Table 2
Comparison of CRP before and after treatment ($\bar{x} \pm s$)

Group	Cases	Before treatment	1 Week	1 Month	3-Month	Last follow-up
Group A	18	34.05 $\pm$ 14.62	25.12 $\pm$ 6.96	19.03 $\pm$ 4.12	4.31 $\pm$ 1.79	2.87 $\pm$ 2.22
Group B	18	37.85 $\pm$ 14.96	28.76 $\pm$ 12.81	19.71 $\pm$ 4.12	5.38 $\pm$ 2.10	2.30 $\pm$ 1.84
Group C	18	33.59 $\pm$ 15.89	23.61 $\pm$ 6.90	20.74 $\pm$ 6.41	5.83 $\pm$ 2.54	3.54 $\pm$ 1.64
<i>F</i>		0.43	1.45	0.53	1.95	1.88
<i>p</i>		> 0.05	> 0.05	> 0.05	> 0.05	> 0.05

CRP Normal value range: 0–10 mg/L.

2.3 Evaluation of improvement in VAS and ODI

Results indicated that within one week post-treatment, pain had alleviated and independent living capacity improved across all groups ($P < 0.05$). By the final follow-up, the surgical treatment group demonstrated statistically significant superiority in VAS scores at all time points compared to the pharmacological treatment group ($P < 0.05$). Comparisons of VAS scores at 1 week and 3 months post-treatment between the simple decompression group and the decompression plus radiofrequency ablation group showed statistically significant differences ($P < 0.05$). However, comparisons at the final follow-up showed no statistically significant differences in VAS scores among the three groups ($P > 0.05$ for all). At 1 week, 1 month, and 3 months post-treatment, the combined radiofrequency ablation group

demonstrated superior improvement in ODI scores compared to the puncture decompression alone group ($P < 0.05$). At the final follow-up, no statistically significant differences in ODI scores were observed among the three groups ($P > 0.05$ for all comparisons).

Table 3
Comparison of VAS scores before and after treatment ($\bar{x} \pm s$)

Group	Cases	Before treatment	Immediate postoperative	1 Week	3-Month	Last follow-up
Group A	18	8.22 $\pm$ 0.55	5.28 $\pm$ 0.96	3.94 $\pm$ 1.06	3.72 $\pm$ 1.13	1.14 $\pm$ 0.62
Group B	18	8.44 $\pm$ 0.71	4.44 $\pm$ 0.86* [#]	2.83 $\pm$ 0.51* [#]	1.78 $\pm$ 0.55* [#]	1.33 $\pm$ 0.77
Group C	18	8.22 $\pm$ 0.81	3.22 $\pm$ 0.73*	1.89 $\pm$ 0.68*	1.39 $\pm$ 0.50*	0.94 $\pm$ 0.54
<i>F</i>		0.61	26.40	31.13	46.28	2.96
<i>p</i>		> 0.05	< 0.01	< 0.01	< 0.05	> 0.05
*: Compared with the A group, $P < 0.05$; #: Compared with the C group, $P < 0.05$						

Table 4
Comparison of ODI scores before and after treatment ($\bar{x} \pm s$)

Group	Cases	Before treatment	1 Week	1 Month	3-Month	Last follow-up
Group A	18	75.33 $\pm$ 3.07	71.50 $\pm$ 3.96	54.56 $\pm$ 2.33	32.44 $\pm$ 7.45	5.00 $\pm$ 1.75
Group B	18	75.39 $\pm$ 2.45	47.28 $\pm$ 2.78* [#]	27.72 $\pm$ 4.80* [#]	16.56 $\pm$ 4.90* [#]	4.78 $\pm$ 2.13
Group C	18	73.06 $\pm$ 2.39	42.06 $\pm$ 5.33*	26.33 $\pm$ 4.38*	14.61 $\pm$ 3.62*	4.28 $\pm$ 1.67
<i>F</i>		4.5	257.18	265.94	39.33	0.71
<i>p</i>		> 0.05	< 0.01	< 0.01	< 0.01	> 0.05
*: Compared with the A group, $P < 0.05$; #: Compared with the C group, $P < 0.05$						

2.4 TNF- α and IL-6 concentrations Results evaluation

Results indicated that there were no statistically significant differences in peripheral blood TNF- α and IL-6 concentrations among the three groups prior to treatment ($P > 0.05$ for all). One week post-treatment, peripheral blood TNF- α and IL-6 concentrations decreased across all groups. Preoperatively, no significant difference existed in bone marrow concentrations of these two inflammatory factors between the two surgical groups ($P > 0.05$). However, postoperatively, the radiofrequency ablation group (C) exhibited significantly lower TNF- α and IL-6 than the decompression-only group (B) ($P < 0.05$). At 1 week and 3 months post-treatment, inflammatory factor levels in the surgical group were consistently superior to those in the medical group at all time points, consistent with changes in the patients' VAS scores.

Table 5
Comparison of TNF- α concentration before and after treatment ($\bar{x} \pm s$)

Group	Cases	peripheral blood before treatment	Preoperative bone marrow blood	Bone marrow blood after decompression	Peripheral blood after 1 week	Peripheral blood after 3- week
Group A	18	30.67 $\pm$ 6.26	-	-	23.06 $\pm$ 5.30	13.00 $\pm$ 2.22
Group B	18	27.28 $\pm$ 7.08	84.78 $\pm$ 7.14	43.56 $\pm$ 10.99 [#]	18.22 $\pm$ 5.06 ^{*#}	9.72 $\pm$ 3.53 [*]
Group C	18	25.89 $\pm$ 5.73	82.06 $\pm$ 7.17	34.17 $\pm$ 7.50	14.50 $\pm$ 2.96 [*]	9.94 $\pm$ 2.60 [*]
<i>F/t</i>		2.67	1.30	8.97	15.93	7.50
<i>p</i>		> 0.05	> 0.05	< 0.05	< 0.05	< 0.05
*: Compared with the A group, $P < 0.05$; #: Compared with the C group, $P < 0.05$						

Table 6
Comparison of IL-6 concentration before and after treatment ($\bar{x} \pm s$)

Group	Cases	peripheral blood before treatment	Preoperative bone marrow blood	Bone marrow blood after decompression	Peripheral blood after 1 week	Peripheral blood after 3- week
Group A	18	44.94 ± 6.12	-	-	21.72 ± 7.17	14.94 ± 2.80
Group B	18	44.00 ± 7.32	110.17 ± 17.29	57.78 ± 8.71 [#]	20.00 ± 5.02 [#]	5.94 ± 1.80 [*]
Group C	18	43.22 ± 7.13	111.06 ± 23.08	49.61 ± 6.41	14.61 ± 3.94 [*]	4.89 ± 1.94 [*]
<i>F/t</i>		0.28	0.01	10.26	8.07	111.39
<i>p</i>		> 0.05	> 0.05	< 0.05	< 0.05	< 0.05

*: Compared with the A group, $P < 0.05$; #: Compared with the C group, $P < 0.05$

2.5 Changes in Intravertebral Pressure Before and After Surgery

Percutaneous decompression produced a significant reduction in intravertebral pressure from pre- to post-procedure in groups B and C ($P < 0.05$). However, among the 36 patients with intraoperative pressure measurement, the percentage reduction in intravertebral pressure did not correlate significantly with the percentage reduction in VAS score at final follow-up (Pearson $r = -0.09$, $P = 0.60$), suggesting that mechanical decompression alone accounts for only a partial component of the observed analgesic effect.

Table 7
Changes in Intravertebral Pressure Between Pre- and Post-Decompression Groups

Group	Cases	Pre-decompression	post-decompression	<i>t</i>	<i>p</i>
Group B	18	41.22 ± 3.41	18.72 ± 2.97	18.59	< 0.01
Group C	18	40.67 ± 4.73	19.11 ± 3.01	16.47	< 0.01

2.6 Complications

At the final follow-up, all 54 patients were assessed for clinical efficacy. Among them, 38 patients (70.4%) had excellent outcomes (VAS ≤ 2 points), 14 patients (25.9%) had good outcomes (VAS 3–4 points), and the remaining 2 patients (3.7%) had poor outcomes (VAS ≥ 5 points). The overall effective rate was 96.3% (52/54). Pain recurrence was observed in 4 patients (7.4%) at a mean postoperative period of 4.2 ± 1.3 months, all of whom underwent repeat radiofrequency ablation for symptom relief.

Notably, no major severe complications associated with the ablation procedure were detected in any case, such as spread of brucellar infection, permanent nerve injury, or progressive spinal instability.

Discussion

Based on a review of current literature and our experimental research on the biomechanics and histopathology of brucellosis spondylitis, this study innovatively proposes a combined treatment model of mechanical decompression and neural modulation. This approach achieves precise intervention targeting the dual underlying mechanisms of intractable pain in lumbar brucellosis spondylitis: increased intravertebral pressure and cytokine-mediated neurosensitisation.

The normal vertebral bone marrow cavity exists within a relatively sealed mechanical environment where pressure is maintained within a stable range. The acute inflammatory response triggered by *Brucella* infection significantly disrupts this equilibrium, potentially leading to increased intravertebral pressure through the following pathways. Firstly, inflammation-activated cells release vasoactive substances, causing vasodilation and increased permeability in the vertebral periosteum and intramedullary vessels. Substantial plasma components then infiltrate the interstitial spaces, forming localised oedema, while concurrent inflammatory cell infiltration further increases the vertebral body's internal volume. Research confirms that early-stage infected vertebrae exhibit elevated intravertebral pressure compared to normal levels, showing significant positive correlation with inflammatory mediators such as TNF- α and IL-6 [17]. This validates the accuracy of our measurements, demonstrating that such pressure elevation directly stimulates persistent dull pain within the vertebra, establishing an inflammation-pressure cycle. This also explains why antibiotics, while controlling infection, cannot eliminate established pressure accumulation and neural sensitisation, thus failing to rapidly alleviate severe low back pain. Secondly, abscess formation further exacerbates intravertebral pressure. Approximately 30% of BS patients develop paravertebral or epidural abscesses. These accumulations of pus within enclosed spaces generate high pressure [18]. Research indicates that undrained abscesses maintain elevated intravertebral pressure and significantly reduce antibiotic penetration efficiency [19]. Our clinical observations also reveal that BS patients experience reduced pain while bedridden but worsening pain upon ambulation. This may relate to increased spinal loading from postural changes, further elevating intravertebral pressure and stimulating nerve endings. Finally, proteases and inflammatory mediators released by *Brucella* bacteria progressively disrupt the trabecular bone structure of vertebrae, forming worm-eaten bone defects. Finite element analysis models reveal pronounced "stress concentration" phenomena at the undamaged vertebral margins surrounding the bone destruction zones, with local pressure loads increasing by 2.8-fold [20]. This abnormal pressure distribution not only exacerbates bone destruction but also induces radiating pain by traction on surrounding soft tissues and nerve roots. Advanced vertebral collapse alters spinal alignment, subjecting adjacent vertebrae to additional stress and creating a vicious cycle of pain. The precise vertebral pressure measurement and decompression system employed in this study are critical for evaluating decompression efficacy and pain relief. Our customised vertebral decompression system demonstrated excellent reliability, with advantages including: (1) 15G fine-gauge needles minimising tissue trauma; (2) pressure readings taken after 30 seconds of stabilisation to ensure

equilibrium and enhance measurement consistency; (3) preoperative pressure readings are consistent with elevated intradiscal pressure observed in animal models of disc degeneration[21], validating the system's measurement accuracy. Mechanical decompression achieved substantial intravertebral pressure reduction (Table 7), confirming pressure off-loading as a key analgesic component. This aligns with clinical evidence that decompression alleviates spinal infection pain[22].

In the pathogenesis of BS, TNF- α and IL-6, as key inflammatory mediators, synergise with mechanical stress to jointly contribute to the generation and maintenance of pain. Research indicates that TNF- α primarily initiates inflammatory responses and pain signals during the acute phase. When vertebral bodies undergo bacterial infection, activated macrophages release TNF- α . This cytokine activates the NF- κ B signalling pathway, promoting the synthesis and release of numerous pro-inflammatory mediators, thereby establishing a vicious cycle of inflammation [23]. Animal studies confirm elevated TNF- α concentrations in vertebral tissue of BS model mice compared to controls, showing significant correlation with pain behavioural scores [24, 25], which underscores the pivotal role of TNF- α in BS-associated pain. Concurrently, TNF- α directly acts upon dorsal root ganglia, heightening their sensitivity to mechanical and chemical stimuli and lowering pain thresholds. As a multifunctional cytokine, IL-6 sustains pain states and promotes neural remodelling during the subacute and chronic phases of infection. Studies indicate markedly elevated IL-6 levels in both serum and cerebrospinal fluid of BS patients, persisting longer than TNF- α . Furthermore, IL-6 induces sensory neurons to express increased capsaicin receptors (TRPV1) and sodium channels, enhancing neural impulse conduction efficiency [26–28]. This neurosensitisation effect causes patients to exhibit intense pain responses to minor stimuli even after infection control, manifesting as allodynia.

In this study, preoperative TNF- α and IL-6 levels in the bone marrow of affected vertebrae were markedly elevated compared to peripheral blood. The significant difference in cytokine levels between bone marrow and peripheral blood ($P < 0.05$) suggests that abnormal inflammatory factors are primarily confined to the vertebral lesion area. This explains why systemic anti-inflammatory treatment yields limited efficacy, whereas percutaneous decompression achieves rapid pain relief by directly aspirating inflammatory bone marrow fluid, thereby enabling targeted clearance of inflammatory factors. Notably, although decompression reduced intravertebral pressure by a mean of 53%, the percentage reduction in pressure did not correlate with VAS score reduction at final follow-up (Pearson $r = -0.09$, $P = 0.60$; see Results). This dissociation indicates that mechanical off-loading accounts for only a partial component of the analgesic response—the substantial and rapid pain relief (mean VAS reduction 86%) implies additional contributions from neurogenic and inflammatory pathways that decompression alone cannot address. This provides the mechanistic rationale for combining decompression with basivertebral nerve radiofrequency ablation. However, bone marrow cytokine levels after decompression remained elevated relative to peripheral blood. This disparity highlights the distinct kinetic profiles of these mediators and underpins the complementary roles of mechanical and thermal therapies. Extending the clinical observation that IL-6 persists longer than TNF- α , the disproportionately greater IL-6 reduction after RFA (vs. TNF- α) aligns with IL-6's role as a sustained-output cytokine in chronic infection, whereas TNF- α peaks acutely and is more readily cleared by mechanical aspiration. Consequently, while decompression

effectively evacuates pre-formed inflammatory pools, RFA is crucial for suppressing the ongoing cytokine synthesis that drives chronic pain.

During percutaneous vertebroplasty (PVP), the process of puncturing the pedicle and subsequently withdrawing the needle core causes bleeding within the vertebral body while simultaneously reducing vertebral pressure. Upon injection of bone cement, we observed a marked exacerbation of the patient's pain, confirming that increased vertebral pressure induces pain. Previous studies have demonstrated that the exothermic reaction during bone cement polymerisation elevates temperatures beyond 50°C, causing thermal necrosis and chemical neurotoxicity in adjacent tissues, potentially ablating nerves surrounding fractured vertebrae [29, 30]. Unlike PVP, where uncontrolled, sustained exothermic cement polymerization (> 50°C) may cause inadvertent thermal necrosis, RFA employs precise temperature control (70–80°C, 60 s) to achieve selective basivertebral nerve ablation while minimizing collateral damage. On one hand, it destroys the neural roots and posterior pain-sensing nerve endings to block pain signal transmission. On the other hand, local thermotherapy inhibits the activity of inflammatory enzymes (such as matrix metalloproteinase-9), further reducing cytokine release [31]. This "destruction followed by blockade" strategy aligns closely with the pathological progression of BS. Furthermore, in this study, radiofrequency ablation achieved additional pain relief beyond decompression alone. The VAS score decreased to 1.89 ± 0.68 at the 1-week follow-up and remained stable at the final follow-up (> 6 months), indicating the long-term efficacy of neural modulation. The ODI score decreased from 73.06 ± 2.39 preoperatively to 4.28 ± 1.67 at the final follow-up. This improvement in ODI scores reflects not only pain relief but also restoration of spinal function. Following decompression alone, both TNF- α and IL-6 in bone marrow blood decreased substantially, with TNF- α showing a marginally greater reduction. However, after radiofrequency ablation, the further decrease in IL-6 was more pronounced. This aligns with the functional characteristics of the two cytokines: decompression primarily eliminates pre-formed inflammatory factors, whereas radiofrequency ablation focuses more on reducing the ongoing synthesis of inflammatory factors, thereby achieving long-term control of chronic pain. Notably, the intravertebral pressure recorded immediately after RFA remained comparable to the post-decompression level, with no observable rebound. This finding may alleviate previous concerns that "radiofrequency ablation could exacerbate tissue oedema and increase pressure" [32], providing biomechanical evidence for the safety of combined procedures.

Current approaches for treating BS pain include surgical methods such as vertebral lesion resection with bone graft fusion and percutaneous abscess drainage. The combined procedure in this study offers the following advantages: (1) Decompression provides immediate pain relief, while radiofrequency ablation maintains long-term efficacy. This explains why patients with inadequate decompression response still benefit from radiofrequency ablation, highlighting the value of combined therapy; (2) Broader indications: Applicable without requiring concomitant abscesses or fractures, provided refractory pain is present; (3) More comprehensive mechanism of action: Simultaneously addresses the three primary pain sources: pressure, cytokines, and neural sensitisation; (4) Superior efficacy: minimal postoperative complications with excellent clinical outcomes. Furthermore, this study pioneered the establishment of a vertebral compression measurement system, providing quantitative guidance for minimally invasive procedures.

This eliminates blind operation and further enhances treatment precision. The limitations of this study include its short research period and intravertebral small sample size, necessitating comparative analysis with larger cohorts.

Abbreviations

BS
Brucellar spondylitis
CT
Computed tomography
MRI
Magnetic resonance imaging
VAS
Visual Analogue Scale
ODI
Oswestry Disability Index
CRP
C-reactive protein
ESR
Erythrocyte sedimentation rate
PVP
Percutaneous vertebroplasty
RFA
Radiofrequency ablation

Declarations

Ethics approval and consent to participate

This study was performed in line with the principles of the Declaration of Helsinki.

Consent for publication

Written informed consent was obtained from the patient for publication of this report and accompanying images.

Competing interests

The authors declare that they have no competing interests.

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Author Contribution

CL and HL contributed to conceptualization, data curation, formal analysis, and writing—original draft. BL contributed to conceptualization, data curation, formal analysis, writing—original draft, and writing—review and editing. HL, JL, and ZY contributed to formal analysis, methodology, and software. JW, QS, ZQ, and BR contributed to conceptualization, data curation, formal analysis, investigation, and methodology. YW contributed to conceptualization, supervision, data curation, formal analysis, and funding acquisition. All authors read and approved the final manuscript.

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Not applicable.

Data Availability

Data supporting the findings of this study are available from the corresponding author upon reasonable request.

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33. Case. 1.

Figures

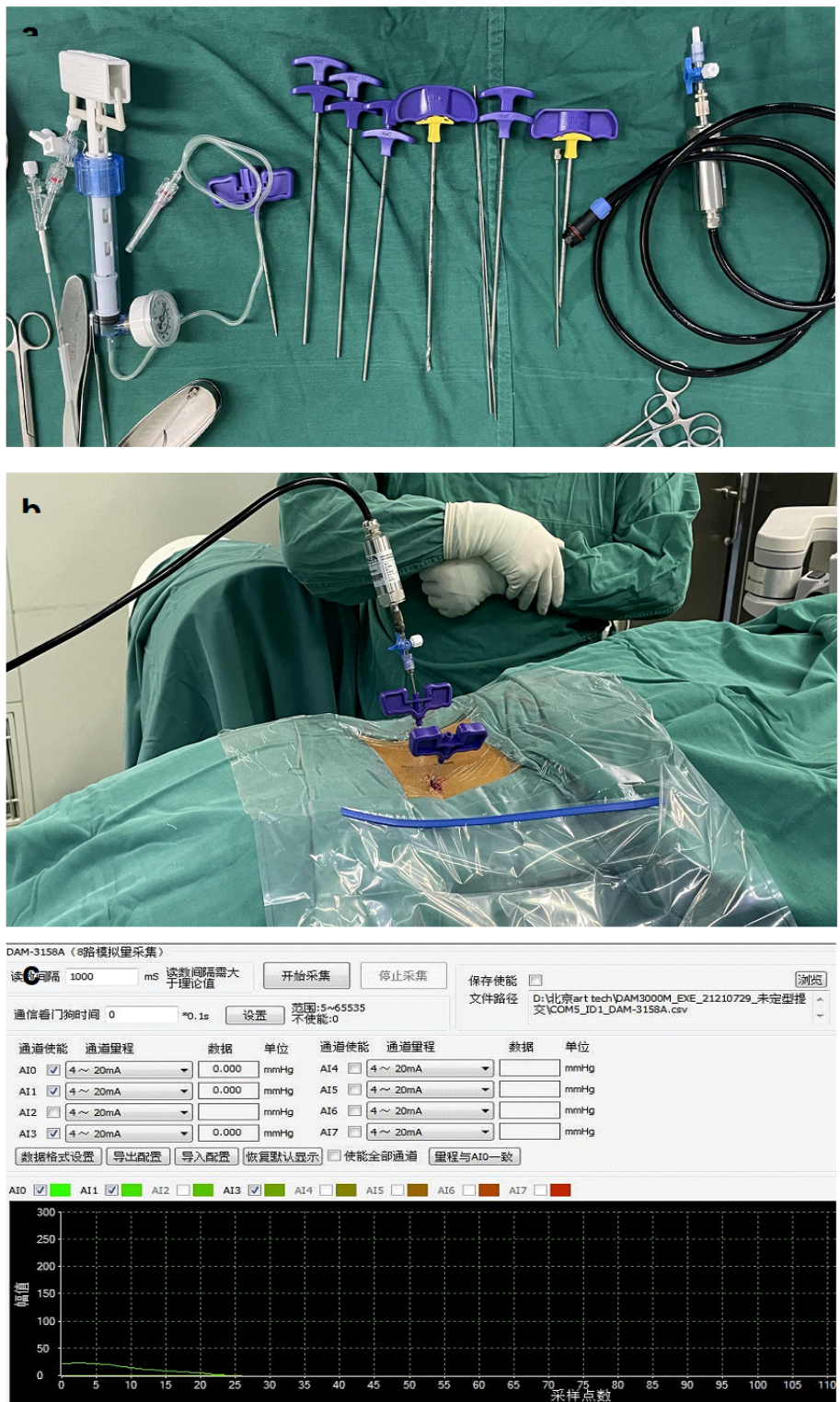


Figure 1

(a) Preoperative preparation and connection of the pressure measurement device; (b) Intraoperative measurement of intravertebral pressure; (c) PC software interface: intravertebral pressure recording area and pressure curve display area

Supplementary Files

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