

Retrospective Study

Clinical Observation and Cause Analysis of Perioperative Superior Cluneal Neuralgia in Vertebral Augmentation

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Background: In our clinical practice, we observed that some osteoporotic vertebral compression fracture patients undergoing vertebral augmentation exhibited pain in the iliac crest region. This pain aligned with the diagnostic criteria for superior cluneal neuralgia (SCN) and affected treatment satisfaction.

Objective: This study aims to clinically observe patients undergoing vertebral augmentation in a hospital setting and analyze the etiology and risk factors associated with SCN.

Study Design: Retrospective cohort study.

Setting: Inpatient population of a single center.

Methods: We retrospectively analyzed clinical data from 630 patients who underwent vertebral augmentation in our hospital from March 2022 to March 2023. Fifty-two patients enrolled in the study experienced pain that met the diagnostic criteria for superior cluneal neuralgia during the perioperative period of the vertebral augmentation procedures. Those patients were divided into 2 subgroups according to the conditions involved in the occurrence of SCN: Group A (26 patients) had either no preoperative SCN but developed it postoperatively, or had preoperative SCN that worsened or did not alleviate postoperatively. Group B (26 patients) had preoperative SCN that was relieved postoperatively. Additionally, 52 consecutive patients in March 2022 to March 2023, who did not experience SCN during the perioperative period were selected as the control group (Group C). Variables such as surgical segment, age, height, weight, body mass index, duration of hospitalization, chronic low back pain (CLBP), duration of pain, anesthesia, surgical approach, fracture pattern, preoperative visual analog scale (pre-op VAS) score, intraoperative VAS score, one-day VAS score, one-month VAS score, lumbar sacral angle, and sacral tilt angle were statistically described and analyzed.

Results: In our hospital, the incidence of SCN during the perioperative period of vertebral augmentation procedures is 8.25% (52/630). Among all the segments of patients who developed SCN during the perioperative period, the L1 segment had the highest proportion, which was 29.03% and 35.14% in Groups A and B, respectively. Group B and Group C showed significant differences in duration of hospitalization ($P = 0.012$), pre-op VAS scores ($P = 0.026$), and CLBP ($P < 0.001$). Group A had significantly higher VAS scores preoperatively ($P = 0.026$) and intraoperatively ($P = 0.004$) and in CLBP ($P = 0.001$) than did Group C.

Limitations: This is a retrospective study. Single-center noncontrolled studies may introduce selection bias. The small sample size in each group might have also led to bias.

Conclusion: Perioperative SCN associated with vertebral augmentation is significantly correlated with preoperative VAS scores and CLBP. In addition, intraoperative VAS scores might be a factor contributing to the nonalleviation or exacerbation of postoperative SCN.

Key words: Vertebral augmentation, superior cluneal neuralgia, osteoporotic vertebral compression fracture

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The superior cluneal nerve is believed to be composed of cutaneous branches from the dorsal roots of nerves T11 to L4 (1). Due to variations in different populations, research has found branches of the superior cluneal nerve in the T11-L5 range (2-4). When one or more branches of the superior cluneal nerve are affected by various factors that lead to pain in the innervated areas, the resulting pain is referred to as superior cluneal neuralgia (SCN) (5-11). Osteoporotic vertebral compression fractures (OVCFs) are commonly seen injuries in spinal surgery. The typical clinical presentation includes localized pain at the fracture site and limited spinal mobility. Vertebral augmentation is a minimally invasive procedure often used to treat OVCFs. The main technique involves using a puncture needle to inject bone cement into the fractured vertebra's interior under x-ray guidance. Once the bone cement solidifies, it restores the stability of the fractured vertebra and provides effective pain relief at the fracture site (12,13).

In clinical practice, we have noticed a frequent association between SCN and OVCFs. This pain is commonly linked to specific trigger points. Compression of these trigger points leads to a notable increase in pain for patients, affecting both the trigger points and the regions innervated by the superior cluneal nerve. Local soft tissue blockades can offer relief from this pain (14). Patients and physicians alike may not pay much attention to preoperative SCN in people who have the condition eased postoperatively. However, there is frequent doubt about surgical efficacy in patients whose postoperative relief of SCN is not significant or who even develop SCN after surgery, which lowers patients' satisfaction with the course of treatment. Furthermore, some reports have suggested that pain in the lumbosacral region following compression fractures can be relieved through vertebral augmentation and corrective spinal deformity surgeries (15,16). The impact of vertebral augmentation procedures on SCN in patients remains uncertain. The aim of this study is to investigate the relationship between OVCFs and SCN, analyze their risk factors and pathogenesis, elucidate the occurrence of such pain, and thereby mitigate potential medical-legal disputes.

METHODS

Patient Population

The study cohort consists of patients who underwent vertebral augmentation procedures to treat OVCFs at our hospital between March 2022 and March 2023.

Those patients were divided into 2 subgroups according to the conditions involved in the occurrence of SCN: Group A (26 patients) had either no preoperative SCN but developed it postoperatively, or had preoperative SCN that worsened or did not alleviate postoperatively; Group B (26 patients) had preoperative SCN that was relieved postoperatively; additionally, 52 patients between March 2022 to March 2023 who did not experience SCN during the perioperative period were selected as the control group (Group C).

Selection Criteria

Inclusion criteria were as follows: (1) definitive diagnosis of OVCF, established through each patient's medical history and auxiliary examinations; (2) diagnosis of SCN before the procedure, after the procedure, or both.

Exclusion criteria were as follows: (1) various pathological fractures, including those caused by multiple myeloma, vascular malformations, bone tuberculosis, metastatic bone malignancies, etc.; (2) new-onset OVCFs or other ailments affecting outcome assessment within the follow-up period after discharge; (3) postoperative complications other than SCN that could impact outcome assessment (e.g., bone cement leakage, poor distribution of bone cement, spinal cord nerve injury, worsening of postoperative segmental pain); (4) missing information; (5) having been lost to follow-up.

Surgical Procedures

The patient was positioned in a prone posture, and standard disinfection and draping procedures were performed. C-arm fluoroscopy was used to locate the projection of the vertebral body fracture on the surface of the skin. After satisfactory anesthesia, the direction was adjusted under fluoroscopy, and guide needles were inserted from the pedicle projection points to the appropriate positions. Under C-arm guidance, bone cement was injected into the vertebral body. Fluoroscopy confirmed good distribution of bone cement within the vertebral body, with no evident cement leakage. Postoperatively, the patient reported improved pain in the lower back, with no changes in muscle strength or sensation in the lower limbs. The operating cannulas were then taken out, and each incision site underwent surgical dressing.

Postoperative Management

The hospital information management system was

used to retrieve patients' basic inpatient information, including VAS scores, age, gender, fractured segments, etc. Imaging parameters were measured using the hospital's imaging management system.

Ethical Consideration

This is a retrospective cohort study, and patients' identities remain confidential. Consequently, the hospital ethics committee's clearance was not needed for this investigation.

Data Analysis

SPSS® Version 26.0 (IBM® Corporation) was used for all statistical tests. The mean ± SD was used to represent continuous variables. If the data did not fit the normal distribution, the Kruskal-Wallis test was expressed by a 50% percentile (25%, 75%) for continuous variables. The Student's t-test or Mann-Whitney U test was used for independent samples, and the paired t-test or Wilcoxon signed-rank test was used for paired samples. Categorical variables were analyzed using the chi-square test or Fisher's exact test. Repeated measures ANOVA was utilized if the variance was homogeneous and the data were normally distributed. P-values under 0.05 were regarded as significant.

RESULTS

From March 2022 to March 2023, a total of 630 patients underwent vertebral augmentation procedures at our hospital. Their incidence rate of perioperative

SCN was 8.25% (52/630). Among the 52 patients who experienced perioperative SCN, there were 8 men and 44 women, with an average age of 75.59 ± 10.00. Ten patients had fractures in 2 vertebral bodies, and 4 patients had fractures in 3 or more vertebral bodies. The distribution of fractured segments in different subgroups can be found in Table 1. Table 2 displays the patients' fundamental characteristics, such as age, gender, duration of pain, amount of anesthesia used, lumbar sacral angle, and more.

Among patients who experienced SCN, Group A had VAS scores of 8 (7.00–8.25) preoperatively and 4 (2–6) intraoperatively. When compared to Group C, the VAS scores in these two time periods were significantly higher in Group A, with statistical significance (P = 0.026, 0.004). Additionally, the occurrence of CLBP was significantly higher in Group A than in Group C (P = 0.001). Group B had a longer duration of hospitalization (P = 0.019) and higher preoperative VAS scores (P = 0.012) and incidence of CLBP (P < 0.001) than did Group C. Other parameters did not show significant differences.

We established a logistic regression model to predict the probability of postoperative persistent SCN (PPP-SCN) (Fig. 1). Using the regression analysis, a nomogram was constructed that incorporated the 3 significant risk factors for predicting PPP-SCN (Fig. 2). On the point scale axis, a score was assigned to each of these variables' values. Each individual score could be added quickly to get the overall score, and we could estimate the PPP-SCN by projecting the total score to the

Table 1. Number and proportion of fractured vertebrae (T6-L5) in each group.

Group A			Group B			Group C		
Segment	Number	Percentage	Segment	Number	Percentage	Segment	Number	Percentage
L1	9	29.03%	L1	13	35.1%	L1	14	21.54%
T12	5	16.13%	T12	7	18.9%	T12	10	15.38%
L2	5	16.13%	L2	6	16.2%	L4	10	15.38%
L3	4	12.90%	T7	4	10.8%	L3	9	13.85%
T11	3	9.68%	L3	4	8.1%	L2	8	12.31%
L4	3	9.68%	L6	1	2.7%	T11	4	6.15%
T6	1	3.23%	T9	1	2.7%	T8	3	4.62%
L5	1	3.23%	T10	1	2.7%	L5	3	4.62%
T7	0	0.00%	T11	1	2.7%	T9	2	3.08%
T8	0	0.00%	T8	0	0.0%	T7	1	1.54%
T9	0	0.00%	L4	0	0.0%	T10	1	1.54%
T10	0	0.00%	L5	0	0.0%	T6	0	0.00%
Total	31	100%	Total	37	100%	Total	65	100%

Table 2. Comparison of various indicators among Groups A, B, and C as well as the results of their P-values.

Characteristic	Group A	Group B	Group C	P-total		
				P-ab	P-ac	P-bc
Mean age (yrs) (x ± s)	77.89±10.14	73.31±9.50	74.08±10.43		0.204	
Female gender (%)	84.62%	84.62%	84.62%		1	
Height (cm)	155 (155.50-158)	156 (150-159.25)	160 (153.5-168)		0.656	
Weight (kg)	55 (50-63.50)	55 (48-63.25)	54 (49.25-65)		0.999	
BMI	22.75 (20.81-25.65)	24.01 (19.88-26.60)	23.02 (20.81-25.87)		0.818	
Duration of hospitalization (days)	5.5 (3.75-8.00)	5.5 (4-11.25)	4 (3-6)	0.563	0.067	0.012*
Pre-op VAS	8 (7.00-8.25)	8 (7-8)	7 (6.25-8)	0.996	0.026*	0.026*
Intra-op VAS	4 (2-6)	2.5 (2-3.25)	2 (1-3)	0.061	0.004*	0.488
1-day Postoperative VAS	3.5 (2-5)	3 (2-4)	3 (2-4)		0.231	
1-month Postoperative VAS	2 (1.00-3.00)	1 (0-3)	1 (0-2)		0.147	
Duration of pain (days)	10.5 (5.50-16.25)	16 (8-25.25)	12 (7.25-21)		0.791	
Lumbar Angle						
Lumbosacral Angle	32.46±8.51	28.37±7.78	31.64±8.75		0.172	
Sacral Tilt Angle	36.17 (33.45-40.38)	35.89 (30.94-43.53)	36.01 (31.75-45.74)		0.204	
Fracture pattern						
Single segmental fracture	21	16	43		0.234	
Double segmental fracture	4	7	5			
Multiple segmental fracture	1	3	4			
Surgical Approach						
Left	2	1	3		0.243	
Right	7	11	11			
Bilateral	17	14	38			
Anesthesia Method						
Intravenous Anesthesia	17	12	32		0.310	
Local anesthesia	9	14	20			
History of CLBP						
Yes	19	21	17	0.743	0.001*	<0.001*
No	7	5	35			

Body mass index: BMI. Preoperative visual analog scale: pre-op VAS. Intraoperative visual analog scale: intra-op VAS. Duration of pain (days): The patient experienced significant pain in the lumbar and thoracic regions following an OVCF, up until the time of undergoing vertebral augmentation. Chronic lower back pain: CLBP. (Preoperatively, the patient had a history of chronic lower back pain lasting 3 months or longer, and the condition was distinct from SCN.)

Notes: * represents $P < 0.05$ between the 2 groups. Data are presented as mean ± SD or 50% (25%-75%). All VAS scores in the table refer to the pain scores caused by OVCF, not SCN. Therefore, even in Group A, it can be observed that postoperative pain caused by OVCF is generally relieved. When drawing comparisons using the appropriate statistical methods corresponding to the data types, the P value will be denoted as P -total if there is no statistically significant difference in pairwise comparisons among the 3 groups. If there is a statistically significant difference between any 2 groups, further comparisons of their P values are conducted. P-ab represents the P value obtained from comparing Group A with Group B, while P-ac and P-bc follow the same principle.

lower total point scale. A calibration curve is presented to evaluate the accuracy of this model (Fig. 3).

In our study, we found no significant differences in the lumbosacral angle and sacral tilt angle between the control group and the study group ($P > 0.05$) (Table 2).

DISCUSSION

The onset of SCN is attributed to various factors that affect the nerve. These causes include mechanical compression of the superior cluneal nerve within the

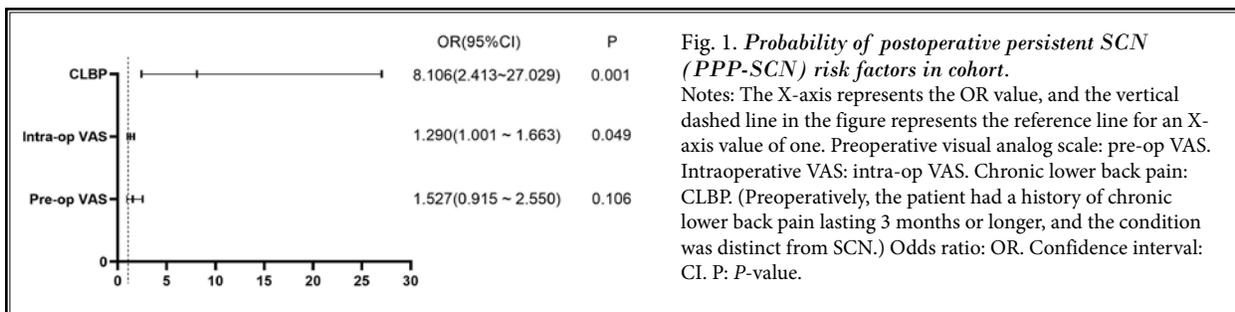


Fig. 1. Probability of postoperative persistent SCN (PPP-SCN) risk factors in cohort.

Notes: The X-axis represents the OR value, and the vertical dashed line in the figure represents the reference line for an X-axis value of one. Preoperative visual analog scale: pre-op VAS. Intraoperative VAS: intra-op VAS. Chronic lower back pain: CLBP. (Preoperatively, the patient had a history of chronic lower back pain lasting 3 months or longer, and the condition was distinct from SCN.) Odds ratio: OR. Confidence interval: CI. P: P-value.

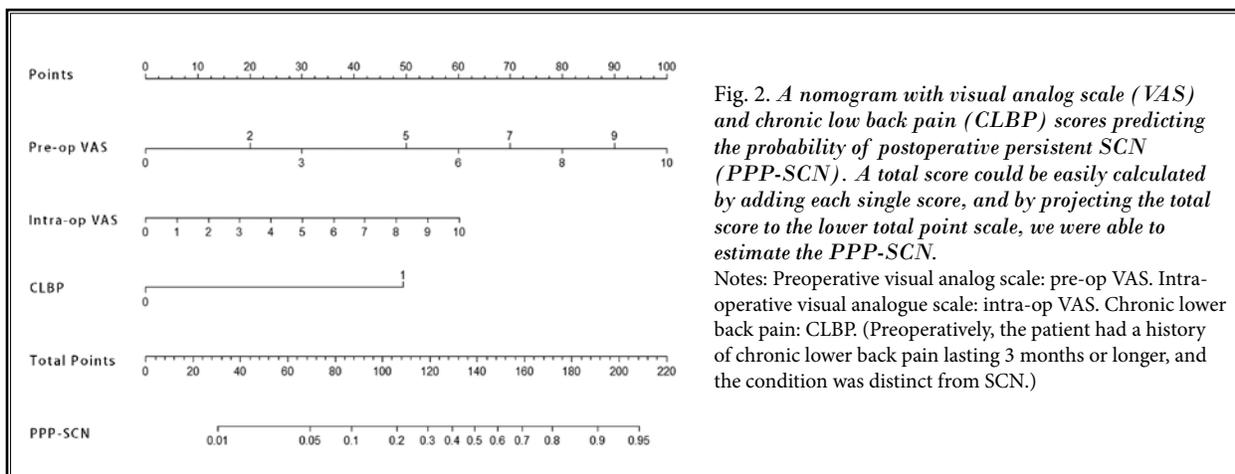


Fig. 2. A nomogram with visual analog scale (VAS) and chronic low back pain (CLBP) scores predicting the probability of postoperative persistent SCN (PPP-SCN). A total score could be easily calculated by adding each single score, and by projecting the total score to the lower total point scale, we were able to estimate the PPP-SCN.

Notes: Preoperative visual analog scale: pre-op VAS. Intraoperative visual analogue scale: intra-op VAS. Chronic lower back pain: CLBP. (Preoperatively, the patient had a history of chronic lower back pain lasting 3 months or longer, and the condition was distinct from SCN.)

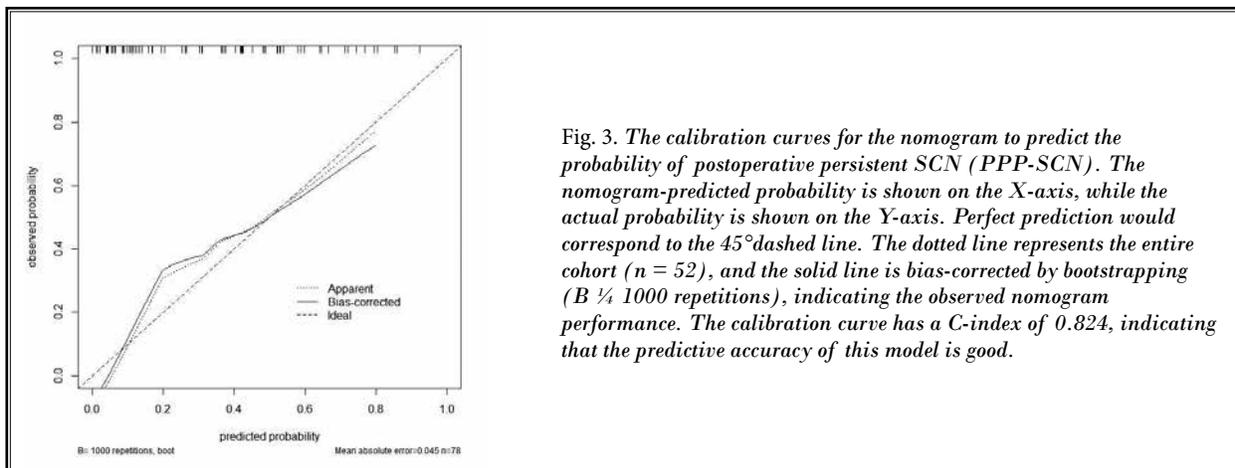


Fig. 3. The calibration curves for the nomogram to predict the probability of postoperative persistent SCN (PPP-SCN). The nomogram-predicted probability is shown on the X-axis, while the actual probability is shown on the Y-axis. Perfect prediction would correspond to the 45°dashed line. The dotted line represents the entire cohort (n = 52), and the solid line is bias-corrected by bootstrapping (B ¼ 1000 repetitions), indicating the observed nomogram performance. The calibration curve has a C-index of 0.824, indicating that the predictive accuracy of this model is good.

fibrous tunnel during its passage through the thoracolumbar fascia, mechanical compression due to adipose tissue, stretching of the buttock and back muscles to the point of mechanical injury to the superior cluneal nerve, potential nerve damage during iliac crest bone harvesting, and pathological influences like compression and inflammatory stimulation of the superior cluneal nerve's higher-level branches (2,7,17-19). We

can confirm the morphology of the superior cluneal nerve through ultrasound examination (20-22). Using angiography to assess blood flow perfusion also makes it possible to determine whether the superior cluneal nerve is experiencing compression indirectly (23).

In our study, physical examination was performed on all patients, resulting in confirmed diagnoses of SCN. Patient One was a 66-year-old female. When we

inquired about her medical history, it was revealed that she did not have a history of SCN before the OVCF occurred. Typical clinical symptoms of SCN appeared after the onset of OVCF. Preoperative lumbar vertebral anteroposterior and lateral x-ray examination suggested that the patient had an L1 vertebral compression fracture. Through a physical examination, we could pinpoint 2 trigger points on Patient One's left side, precisely over the posterior iliac crest, aligning with the compression zone of the superior cluneal nerve (Fig. 4).

Patient 2 was a 75-year-old woman with a T11 OVCF who did not experience SCN before surgery but developed left-sided SCN postoperatively. A postoperative x-ray reassessment indicated a satisfactory distribution of bone cement (Fig. 5). We performed an ultrasound examination of the superior cluneal nerve, revealing significant swelling of the left-sided superior cluneal nerves (Fig. 6), implying potential compression and resultant nerve edema.

SCN can be alleviated through local soft tissue injections, radiofrequency ablation, or nerve decompression procedures (14,24-28). For discussion, we divided

the incidences of SCN related to vertebral augmentation procedures into categories:

Group A comprises patients for whom surgery is considered a potentially triggering and exacerbating factor for SCN. We believe that intraoperative pain stimulation and psychological factors may be the primary factors contributing to this outcome in these patients. Intraoperative pain stimulation and psychological factors can lead to the contraction of a patient's back muscles (29,30). If a patient's superior cluneal nerve branches have a tortuous course through the back muscles or if the bone fiber tunnel itself is narrow, it is likelier to induce SCN in the patient.

Pain masking may also be a contributing factor to the postoperative discovery of SCN in some patients. In certain cases, patients simultaneously experience SCN and pain at the site of the vertebral body fracture before surgery. The intensity of pain associated with vertebral body fracture is greater than that associated with SCN, and this fracture-related pain may mask the pain originating from the superior cluneal nerve. Once the pain at the fracture site is alleviated through vertebral augmentation, the issue of SCN becomes more apparent.

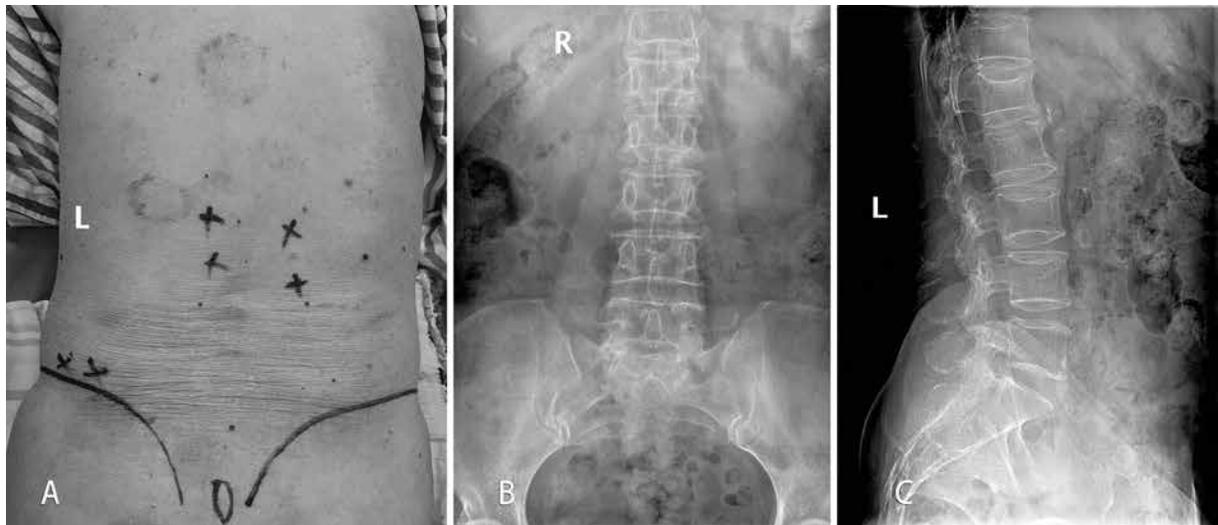


Fig. 4. Patient One is a 66-year-old with an L1 osteoporotic vertebral compression fracture (OVCF) accompanied by superior cluneal neuralgia (SCN). **A:** The patient is placed in a prone position. The 2 longest symmetrical black lines represent the surface projections of the iliac crest and the posterior superior iliac spine. The circle in the middle represents the surface projection of the uppermost edge of the sacral median crest. The black crosses indicate the patient's back's tender points, with the 2 crosses above the left iliac crest representing the trigger points aggravated by SCN. The 4 crosses beside the central spine represent facet joint tender points, which are related to the vertebral fracture. **B and C** are images of the preoperative lumbar vertebral anteroposterior and lateral x-ray examinations of the patient, showing a wedge-shaped deformity of the L1 vertebra, indicating an L1 vertebral compression fracture.

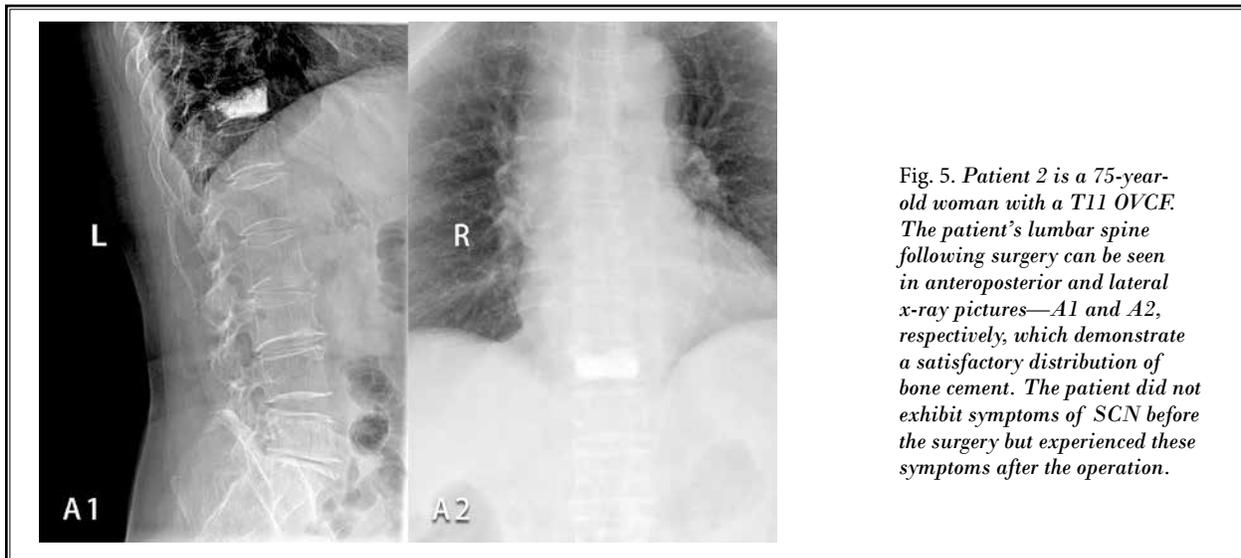


Fig. 5. Patient 2 is a 75-year-old woman with a T11 OVCF. The patient's lumbar spine following surgery can be seen in anteroposterior and lateral x-ray pictures—A1 and A2, respectively, which demonstrate a satisfactory distribution of bone cement. The patient did not exhibit symptoms of SCN before the surgery but experienced these symptoms after the operation.

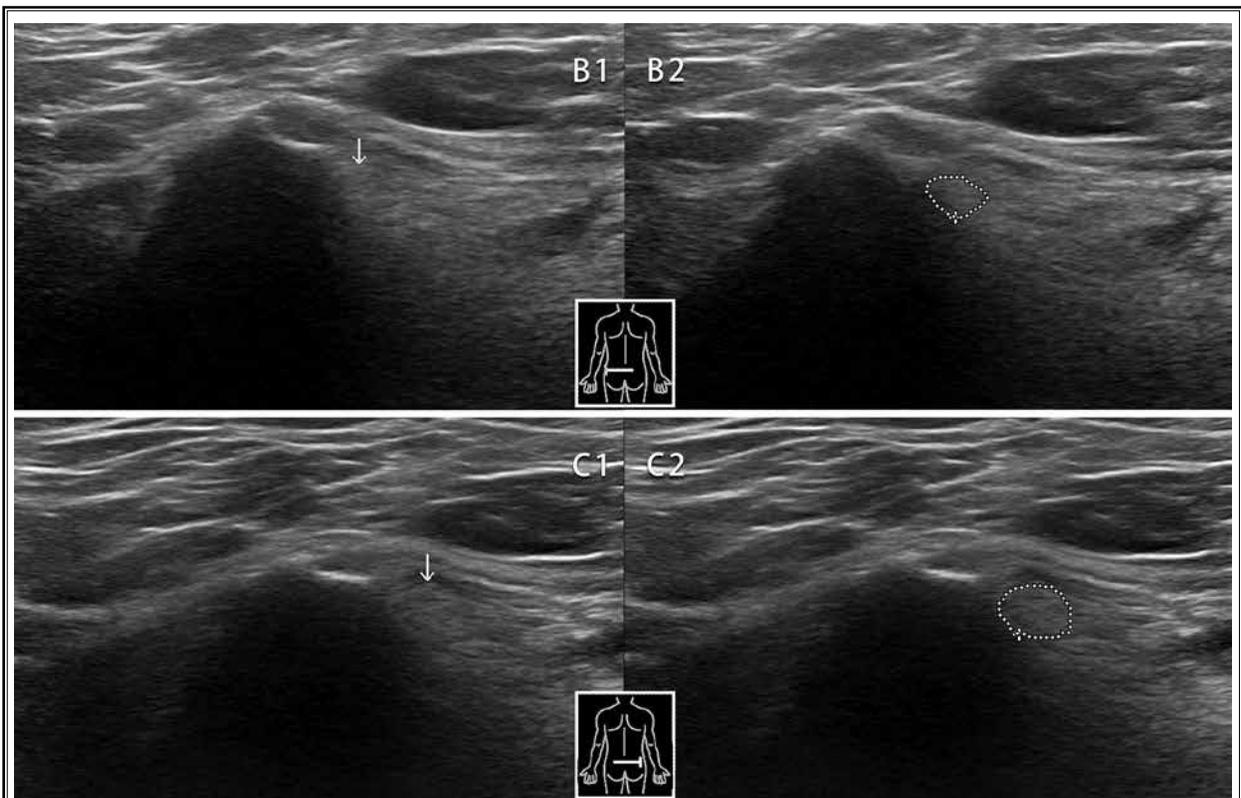


Fig. 6. We conducted a postoperative ultrasound examination of the bilateral superior cluneal nerves in Patient 2 and observed that the right superior cluneal nerve had a significantly thicker medial branch than the left side did. B1: Normal position of the medial branch of the left superior cluneal nerve (indicated by the white arrow). B2: Cross-sectional contour of the normal medial branch of the left superior cluneal nerve (outlined in white dashes, with a circumference of 1.28 cm and a cross-sectional area of 0.112 cm²). C1: Thickened position of the medial branch of the right superior cluneal nerve (indicated by the white arrow). C2: Cross-sectional contour of the thickened medial branch of the right superior cluneal nerve (outlined in white dashes, with a circumference of 1.68 cm and a cross-sectional area of 0.201 cm²).

During the operation, physicians need to establish an external channel for injecting bone cement into the vertebral body. The channel may come close to the nerve's articular branches, and both intraoperative trauma and postoperative local edema may affect articular branches of the nerve behind the vertebrae. While being established, the channel may also pass near the superior cluneal nerve, potentially impacting the development of SCN. The increase in intravertebral pressure is primarily due to the injection of bone cement during vertebral augmentation, which can lead to tissue trauma and inflammation in the surrounding area. This inflammation may irritate the superior cluneal nerve, resulting in SCN. Vertebral augmentation alters the structural integrity of the fractured vertebra, potentially affecting spinal alignment and biomechanics. Changes in alignment may put additional stress or tension on the surrounding nerves, including the superior cluneal nerve, leading to entrapment or irritation.

When the physician performs the unilateral puncture and injects the bone cement, only one channel connects the vertebral body to the outside. By contrast, bilateral puncture offers an additional channel connecting to the outside world. Therefore, bilateral puncture procedures allow for more channels permitting the release of intravertebral pressure, which reduces the likelihood of pressure-related discomfort. Since the degree of lower back pain is also a risk factor for SCN, we hypothesize that a single-channel approach may be associated with a higher probability of SCN caused by intravertebral factors (30).

When patients experience CLBP for a longer duration, their superior cluneal nerves are also subjected to prolonged stretching and compression, resulting in more nerve damage and symptoms of ischemia. Although local soft tissue injections can relieve lower back pain in these patients, this relief is only temporary and provides a short-lived neural blockade. After a certain period, the patient's SCN has a high probability of recurring. The efficacy of conservative treatment for such patients is often unsatisfactory (31). Simultaneously, some patients in the present study had preoperative SCN that showed no significant changes after surgery, indicating that their SCN was not significantly related to vertebral augmentation surgery and was likely caused by other factors. For instance, some patients with Parkinson's disease have chronically tense muscles, leading to prolonged compression of the superior cluneal nerve. In these patients, Parkinson's disease appears to be the predominant factor causing their SCN (32). The

pathogenesis of SCN can also be analogized to post-vertebral augmentation surgery pain in other areas, such as pain in the intercostal region after vertebral augmentation surgery, which involves related neural pain transmission mechanisms as well (33).

In some patients, SCN is triggered during lumbar spine movement. Chiba et al (18) suggest that symptoms of SCN worsen during lumbar hyperextension and when standing or walking. This issue is also related to the superior cluneal nerve's anatomical structure. Changes in limb position that trigger SCN involve the stretching of back muscles and alterations in the superior cluneal nerve's morphology. Therefore, this type of SCN often involves compression of the superior cluneal nerve. The bony structure of the ilium can to some extent affect the superior cluneal nerve and the arrangement of the muscles in the lumbar region. For this reason, we suspect that abnormal lumbar spine angles may influence the occurrence of SCN (34). When Erdem et al reported a case of SCN, they suggested that SCN was related to the posture of the lumbar spine, which could potentially affect the arrangement of muscles in the lumbar region and increase the risk of compression of the superior cluneal nerve (35). Therefore, we conducted a statistical analysis of the lumbosacral angle and sacral tilt angle between our study group and the control group. However, in our study results, we found no significant differences in the lumbosacral angle or sacral tilt angle between the 2 groups ($P > 0.05$). Given the limited sample size of this study, it cannot be ruled out that abnormal lumbosacral angles may be associated with the occurrence of SCN. Further research using a larger sample size is needed to investigate this relationship more comprehensively.

We believe that the onset of SCN in patients has a certain relationship to the tension and contraction of the back muscles caused by lower back pain. Therefore, SCN may be triggered by vertebral augmentation surgery for OVCF or other conditions or lumbar spine surgeries that cause lower back pain (36).

CONCLUSION

SCN could be a complication during the perioperative period of vertebral augmentation procedures. The occurrence of perioperative SCN in vertebral augmentation procedures shows a significant correlation with pre-op VAS scores and the presence of CLBP. Furthermore, the intra-op VAS scores could potentially be a contributing factor to the lack of relief or exacerbation of postoperative SCN.

Author Contributions

Fu-Kuan Zhu was responsible for the conception and writing of the entire article. Fei-Fei Cheng was responsible for the statistical design and selection of statistical methods in the article. Juan Cheng and Si-Yu Wang were responsible for the collection and analysis of patients' ultrasound and x-ray images. Hui Zheng was responsible for the data analysis and cre-

ation of the statistical tables in the article. Xi-Zi Miao iwa responsible for the data collection in the article. Zhong-Liang Deng was responsible for supervising the standardization of the article-writing process and the authenticity of various data collections. Lei Shi and Lei Chu conducted literature retrieval, summarized relevant previous work, and finally revised the article.

REFERENCES

- Kuniya H, Aota Y, Saito T, et al. Anatomical study of superior cluneal nerve entrapment. *J Neurosurg Spine* 2013; 19:76-80.
- Konno T, Aota Y, Kuniya H, et al. Anatomical etiology of "pseudosciatica" from superior cluneal nerve entrapment: A laboratory investigation. *J Pain Res* 2017; 10:2539-2545.
- Iwanaga J, Simonds E, Patel M, Oskouian RJ, Tubbs RS. Anatomic study of superior cluneal nerves: Application to low back pain and surgical approaches to lumbar vertebrae. *World Neurosurg* 2018; 116:e766-e768.
- Iwanaga J, Simonds E, Schumacher M, Oskouian RJ, Tubbs RS. Anatomic study of superior cluneal nerves: Revisiting the contribution of lumbar spinal nerves. *World Neurosurg* 2019; 128:e12-e15.
- Gill B, Cheng DS, Buchanan P, Lee DW. Review of interventional treatments for cluneal neuropathy. *Pain Physician* 2022; 25:355-363.
- Mizumoto J. Superior cluneal nerve entrapment syndrome: A common but often overlooked cause of low back pain. *J Gen Fam Med* 2022; 23:183-184.
- Isu T, Kim K, Morimoto D, Iwamoto N. Superior and middle cluneal nerve entrapment as a cause of low back pain. *Neurospine* 2018; 15:25-32.
- Maigne JY, Doursounian L. Entrapment neuropathy of the medial superior cluneal nerve. Nineteen cases surgically treated, with a minimum of 2 years' follow-up. *Spine (Phila Pa 1976)* 1997; 22:1156-1159.
- Karl HW, Helm S, Trescot AM. Superior and middle cluneal nerve entrapment: A cause of low back and radicular pain. *Pain Physician* 2022; 25:E503-e521.
- Iwanaga J, Simonds E, Schumacher M, Yilmaz E, Altafulla J, Tubbs RS. Anatomic study of the superior cluneal nerve and its related groove on the iliac crest. *World Neurosurg* 2019; 125:e925-e928.
- Tubbs RS, Levin MR, Loukas M, Potts EA, Cohen-Gadol AA. Anatomy and landmarks for the superior and middle cluneal nerves: Application to posterior iliac crest harvest and entrapment syndromes. *J Neurosurg Spine* 2010; 13:356-359.
- Hadjipavlou AG, Tzermiadianos MN, Katonis PG, Szpalski M. Percutaneous vertebroplasty and balloon kyphoplasty for the treatment of osteoporotic vertebral compression fractures and osteolytic tumours. *J Bone Joint Surg Br* 2005; 87:1595-1604.
- Parreira PCS, Maher CG, Megale RZ, March L, Ferreira ML. An overview of clinical guidelines for the management of vertebral compression fracture: A systematic review. *Spine J* 2017; 17:1932-1938.
- Lin F, Zhang Y, Wu T, et al. Local anesthetic and steroid injection to relieve the distal lumbosacral pain in osteoporotic vertebral compression fractures of patients treated with kyphoplasty. *Pain Physician* 2022; 25:E581-E587.
- Fang YP, Lu YJ, Gan MF, Shen X, Lu D. Percutaneous kyphoplasty for a patient of thoracolumbar osteoporotic vertebral compression fractures with distal lumbosacral pain: A case report. *Ann Palliat Med* 2021; 10:4944-4949.
- Niu J, Song D, Gan M, et al. Percutaneous kyphoplasty for the treatment of distal lumbosacral pain caused by osteoporotic thoracolumbar vertebral fracture. *Acta Radiol* 2018; 59:1351-1357.
- Mahli A, Coskun D, Altun NS, Simsek A, Ocal E, Kostekci M. Alcohol neurolysis for persistent pain caused by superior cluneal nerves injury after iliac crest bone graft harvesting in orthopedic surgery: Report of four cases and review of the literature. *Spine (Phila Pa 1976)* 2002; 27:E478-E481.
- Chiba Y, Isu T, Kim K, et al. Association between intermittent low-back pain and superior cluneal nerve entrapment neuropathy. *J Neurosurg Spine* 2016; 24:263-267.
- Knezevic NN, Candido KD, Vlaeyen JWS, Van Zundert J, Cohen SP. Low back pain. *Lancet* 2021; 398:78-92.
- Wu WT, Mezian K, Nanka O, Chang KV, Özçakar L. Ultrasonographic imaging and guided intervention for the superior cluneal nerve: A narrative pictorial review. *Pain Physician* 2022; 25:E657-E667.
- Chang KV, Hsu SH, Wu WT, Özçakar L. Ultrasonographic technique for imaging and injecting the superior cluneal nerve. *Am J Phys Med Rehabil* 2017; 96:e117-e118.
- Ricci V, Özçakar L. Ultrasound imaging of the superior cluneal nerve: Sonoanatomy of the osteo-fibrous tunnel revisited. *Clin Anat* 2019; 32:466-467.
- Kim K, Isu T, Chiba Y, et al. The usefulness of ICG video angiography in the surgical treatment of superior cluneal nerve entrapment neuropathy: Technical note. *J Neurosurg Spine* 2013; 19:624-628.
- Visnjevac O, Pastrak M, Ma F, Visnjevac T, Abd-Elsayed A. Radiofrequency ablation of the superior cluneal nerve: A novel minimally invasive approach adopting recent anatomic and neurosurgical data. *Pain Ther* 2022; 11:655-665.
- Herring A, Price DD, Nagdev A, Simon B. Superior cluneal nerve block for treatment of buttock abscesses in the emergency department. *J Emerg Med* 2010; 39:83-85.
- Kim K, Shimizu J, Isu T, et al. Low back pain due to superior cluneal nerve

- entrapment: A clinicopathologic study. *Muscle Nerve* 2018; 57:777-783.
27. Morimoto D, Isu T, Kim K, et al. Long-term outcome of surgical treatment for superior cluneal nerve entrapment neuropathy. *Spine (Phila Pa 1976)* 2017; 42:783-788.
 28. Morimoto D, Isu T, Kim K, et al. Surgical treatment of superior cluneal nerve entrapment neuropathy. *J Neurosurg Spine* 2013; 19:71-75.
 29. Martinez-Calderon J, Flores-Cortes M, Morales-Asencio JM, Luque-Suarez A. Pain-related fear, pain intensity and function in individuals with chronic musculoskeletal pain: A systematic review and meta-analysis. *J Pain* 2019; 20:1394-1415.
 30. Kallmes DF, Comstock BA, Heagerty PJ, et al. A randomized trial of vertebroplasty for osteoporotic spinal fractures. *N Engl J Med* 2009; 361:569-579.
 31. Vaillancourt PD, Langevin HM. Painful peripheral neuropathies. *Med Clin North Am* 1999; 83:627-642, vi.
 32. Iwamoto N, Isu T, Kim K, et al. Low back pain caused by superior cluneal nerve entrapment neuropathy in patients with Parkinson disease. *World Neurosurg* 2016; 87:250-254.
 33. Lin F, Zhang Y, Song X, et al. Percutaneous kyphoplasty to relieve the rib region pain in osteoporotic thoracic vertebral fracture patients without local pain of fractured vertebra. *Pain Physician* 2023; 26:53-59.
 34. Nakipoğlu GF, Karagöz A, Ozgirgin N. The biomechanics of the lumbosacral region in acute and chronic low back pain patients. *Pain Physician* 2008; 11:505-511.
 35. Erdem HR, Koçak FA, Kurt EE, Tuncay F. Superior cluneal nerve entrapment neuropathy due to lower crossed syndrome: A case with low back pain. *Agri* 2022; 34:311-315.
 36. Iwamoto N, Isu T, Kim K, et al. Treatment of low back pain elicited by superior cluneal nerve entrapment neuropathy after lumbar fusion surgery. *Spine Surg Relat Res* 2017; 1:152-157.