


Impact of Paravertebral Muscle Degeneration on Residual Low Back Pain Following Percutaneous Kyphoplasty for Osteoporotic Vertebral Fractures: A Retrospective Study

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Background: Residual low back pain (LBP) is frequently reported after percutaneous kyphoplasty (PKP) for osteoporotic vertebral fractures (OVFs), yet its underlying mechanisms remain unclear. Paravertebral muscles (PVMs) degeneration, particularly fat infiltration and atrophy may contribute to persistent postoperative pain.

Objective: To evaluate the association between PVMs degeneration and residual LBP after PKP and identify imaging-based predictors for risk stratification.

Methods: This retrospective cohort study included 213 patients (mean age 70.88 ± 8.58 years; 82.2% female) with single-level OVFs who underwent PKP between January 2021 and June 2023. Patients with multiple-level fractures, chronic LBP, neurological deficits, prior spinal surgery, incomplete imaging, or inadequate follow-up were excluded. Fat infiltration percentage (FI%) and cross-sectional area of the multifidus (MF), erector spinae (ES), and psoas major (PS) were measured at the L4 level using transverse T2-weighted MRI. Residual LBP was defined as postoperative VAS ≥ 3.5 at 12-month follow-up. Logistic regression and ROC analyses were conducted and appropriate univariate tests (*t*-test or Mann–Whitney *U*-test) were performed.

Results: Residual LBP occurred in 13.6% of patients and was associated with higher VBQ scores (3.14 ± 0.38 vs 2.57 ± 0.25 , $P=0.001$), greater postoperative kyphosis ($16.03 \pm 6.69^\circ$ vs $6.70 \pm 4.80^\circ$, $P=0.001$), increased FI% of ES/MF ($57.28 \pm 5.63\%$ vs $43.40 \pm 14.93\%$, $P=0.001$), reduced PS area (10.74 ± 4.23 cm² vs 16.15 ± 3.71 cm², $P=0.001$), and concentrated cement distribution (11.5% vs 73.6%, $P=0.001$). Independent predictors included elevated VBQ (OR=85.2, 95% CI 7.006–1036.458), kyphosis (OR=1.14, 95% CI 1.017–1.276), FI% of ES/MF (OR=1.082, 95% CI 1.008–1.160), and PS area (OR=0.509, 95% CI 0.285–0.910). ROC analysis identified FI% $\geq 49.78\%$ and PS area ≤ 11.937 cm² as optimal cutoffs.

Conclusion: Preoperative magnetic resonance imaging assessment of paravertebral muscle may help identify patients at risk for residual low back pain after kyphoplasty. Incorporating preoperative imaging and postoperative physical therapy referral may improve patient outcomes.

Keywords: paravertebral muscle degeneration, low back pain, osteoporotic vertebral fracture, percutaneous kyphoplasty

Introduction

With the aging population, the prevalence of osteoporosis and associated fragility fractures has been steadily increasing. Osteoporotic vertebral fractures (OVFs), commonly result from low-energy trauma or may even occur spontaneously in the absence of any identifiable injury, are associated with severe pain, impaired mobility, and increased mortality. Epidemiological data indicate that the incidence of OVFs is approximately 15% among women aged 50 to 59 years, rising sharply to 50% in women over 85 years of age.¹ Timely and effective treatment is therefore crucial.

The primary objectives of treating OVF are to alleviate pain, prevent further deterioration, minimize the risk of deformity and immobility, and facilitate early mobilization. Conservative management, including medication, bed rest, and orthotic bracing, has limited efficacy in pain control and mobility restoration.² Although some studies have explored the role of dynamic orthoses, their impact on clinical outcomes remains inconclusive.³ In contrast, minimally invasive procedures such as percutaneous vertebroplasty (PVP) and percutaneous kyphoplasty (PKP) have shown superior results in pain relief and functional improvement. A meta-analysis by Halvachizadeh et al⁴ found both PVP and PKP to be more effective than nonoperative treatment in relieving pain and improving quality of life. These findings support the role of cement augmentation in appropriately selected patients.

PKP, introduced in the early 2000s, has gained widespread acceptance as an effective intervention for OVFs by restoring vertebral height, alleviating pain and promoting early mobilization.⁵ Although PKP is a minimally invasive procedure that generally induces a milder postoperative inflammatory response,⁶ residual LBP remains a relatively common and clinically challenging complication, affecting up to 15.6% of patients.⁷ Residual LBP following PKP surgery can substantially compromise the procedure's outcomes and diminish patients' quality of life. A variety of risk factors have been reported to be strongly associated with residual LBP, including bone mineral density (BMD),^{7,8} posterior fascia injury,⁹ cement diffusion rate,⁸ nonunion,¹⁰ multiple vertebral augmentation and injected cement volume.⁷

Recent studies suggest that degeneration of paravertebral muscles (PVMs)—specifically the psoas major (PS), erector spinae (ES), and multifidus (MF)—may play a critical role in spinal stability, sagittal alignment, and pain modulation.^{11–13} Fat infiltration percentage (FI%) and muscle atrophy have been linked to spinal pathologies and poorer recovery after vertebral fractures.¹⁴ In contrast, well-preserved muscles by certain physical therapy may help stabilize the spine, reduce chronic pain, and improve quality of life.¹⁵ Nakamura et al¹⁶ further identified that the incidence of additional OVFs in the thoracolumbar region and persistent LBP in the lumbar region was significantly correlated with the FI% of the PVMs. Nonetheless, few studies have investigated PVMs degeneration in post-PKP residual LBP using magnetic resonance imaging (MRI), and many have not differentiated the functional roles of specific muscles, such as the lumbar stabilizer, PS and the thoracic extensors, ES and MF, despite recognized differences in fat infiltration patterns.^{14,16}

Understanding the role of PVMs degeneration is crucial for optimizing clinical outcomes and developing more effective treatment strategies for OVFs. This study aimed to assess the association between PVMs degeneration, particularly fat infiltration and muscle atrophy, and residual LBP after PKP. We further sought to identify potential predictors and estimate clinically relevant cutoff values for use in preoperative evaluation and postoperative planning. We hypothesized that greater PVMs degeneration may be associated with a higher risk of residual LBP following PKP. The findings may provide insights into underlying mechanisms and inform future clinical and rehabilitative strategies.

Methods

Patients diagnosed with single-level OVFs (ICD-10 code M80) and treated with PKP between January 2021 and June 2023 at our institution were retrospectively reviewed. The detailed inclusion and exclusion criteria were listed as follows. Inclusion criteria: (1) Bone mineral density (BMD) consistent with osteoporosis, as confirmed by dual-energy X-ray absorptiometry (DEXA). (2) A single-level vertebral fracture confirmed by MRI, with corresponding clinical symptoms of acute back pain. (3) Underwent PKP rather than conservative or alternative surgical treatment. (4) Preoperative MRI imaging available for assessing PVMs degeneration. (5) Complete follow-up data for at least 12 months postoperatively. Exclusion criteria: (1) Pathological fractures due to primary/metastatic tumors or spinal infections. (2) Presence of neurological deficit including muscle weakness, numbness et al (3) History of chronic LBP prior to current OVFs. (4) Cognitive impairment or other conditions precluding reliable communication or follow-up. (5) Inadequate MRI quality insufficient for quantitative analysis. (6) History of prior spinal surgery. Comorbidities such as hypertension, diabetes mellitus, smoking and alcohol consumption were not used as inclusion or exclusion criteria but were recorded and analyzed. All diagnoses of OVFs were made and confirmed through radiological assessment (MRI and DEXA) by senior attending spine surgeons.

Surgical Technique

All procedures were performed by two senior spine surgeons, each with over 10 years of experience in PKP. Patients underwent general anesthesia and were placed prone. The fractured vertebra was located by C-arm and skin was routinely disinfected. The bilateral pedicle approach was adopted. After establishing working channel, the balloon was inserted and inflated to restore the height of the vertebra. The balloon was removed after reaching certain pressure. Bone cement was then injected into the vertebra. After curing, working channel was removed and the incision was then closed. Patients ambulated on postoperative day and were discharged 2–3 days postoperatively.

Postoperative Treatment and Follow-Up

All patients received 1000 mg of calcium and 2000 IU of vitamin D supplement daily and were followed up for 12 months.

Identifying Residual Low Back Pain

Residual LBP after PKP is defined using a visual analog scale (VAS). According to a previous study,¹⁷ VAS scores ≤ 3.4 were described as mild pain, 3.5–7.4 as moderate pain, and ≥ 7.5 as severe pain. Patients presented VAS scores over 3.5 12-month post-operation without new fractures were considered as residual LBP.

Patient Clinical Data Collection

The basic information of patients is exported from the medical record system of our institution including sex, age, diagnose, surgical and medication history. Radiological images were collected and analyzed by two independent surgeons. Patients were divided into two groups (VAS ≥ 3.5 and VAS < 3.5) according to last follow-up VAS scores. VAS scores were recorded pre-operation (Pre-operation VAS), one day post-operation (Post-operation VAS) and 12-month post-operation (Last follow-up VAS). Risk factors include sex, age, body mass index (BMI), vertebral bone quality (VBQ), hypertension, diabetes, smoking, drinking, trauma severity, fascia injury, affected vertebra [thoracic spine (T5–T9), thoracolumbar spine (T10–L2), and lumbar spine (L3–L5)], cement volume, cement distribution, local kyphotic angle, total area of PVMs, muscle area of PVMs, FI% of PVMs. Prior research discovered that fat infiltration in the lumbar PVMs was more pronounced at the lower lumbar levels, with measurements at the L4 level serving as a reliable indicator of overall lumbar muscle status.¹⁸ FI%, total area and muscle area of PVMs were assessed using transverse T2-weighted MRI images at the L4 level. Manual segmentation of was performed by two independent observers blinded to clinical outcomes.

FI% of PVMs Measurement

FI% of PVMs was quantified using the Advanced Weka Segmentation plugin (v3.3.4) within the ImageJ platform (NIH v1.54f). This machine learning-based tool executes automated segmentation of adipose tissue in transverse T2-weighted MRI sequences. To normalize against inter-individual heterogeneity, patient-specific segmentation models were calibrated using subcutaneous adipose tissue as an internal reference standard. This approach enabled enhanced precision in identification of intramuscular fat deposition and improved the overall reliability of the FI% measurements.

Statistical Analysis

Normality of continuous variables was assessed using the Shapiro–Wilk test. For comparisons between the residual LBP group and the non-residual LBP group, continuous variables were analyzed using the Mann–Whitney *U*-test or independent samples *t*-test, depending on data distribution. Categorical variables were compared using the Chi-square test. A binary logistic regression model was utilized to identify independent risk factors for residual LBP. Statistical significance was defined as $P < 0.05$. All statistical analyses were carried out using SPSS 26.0 (IBM Corp, USA).

Ethics Statement

This study was approved by the Medical Research Ethics Committee of the First Affiliated Hospital of Soochow University and granted a consent waiver as this study exclusively analyzed pre-existing medical records and imaging data without modifying patient care pathways. To ensure patient confidentiality, access to clinical data was restricted to institution-approved workstations via password-protected Excel files. Following data compilation, comprehensive de-identification procedures were implemented to permanently remove all personal identifiers from analytical datasets. This research was conducted in full compliance with the codes of ethical conduct from the Declaration of Helsinki.

Results

The demographic characteristics of the study cohort were presented in [Table 1](#). Our analysis included 213 consecutive patients (mean age 70.88 ± 8.58 years; 82.2% female) who underwent percutaneous kyphoplasty for OVFs ([Figure 1](#)). The population demonstrated a mean body mass index of 23.64 ± 3.02 kg/m². Vertebral fractures showed distinct anatomical predilection, with the majority localized to the thoracolumbar region (T10-L2, 83.6%), followed by lumbar (L3-L5, 10.8%) and thoracic (T5-T9, 5.6%) regions.

Postoperative residual LBP was identified in 13.6% of the enrolled patients ([Table 2](#)). Comparative analysis revealed significant differences between groups: patients with residual LBP demonstrated elevated VBQ scores (3.14 ± 0.38 vs 2.57 ± 0.25 , $P=0.001$) and greater postoperative kyphotic deformity ($16.03 \pm 6.69^\circ$ vs $6.70 \pm 4.80^\circ$, $P=0.001$) compared to pain-free counterparts. Notably, the residual LBP group exhibited a threefold higher incidence of fascial injury (41.4% vs 17.9%, $P=0.004$). Musculoskeletal parameters showed marked intergroup disparities, including reduced total PS cross-sectional area (10.74 ± 4.23 cm² vs 16.15 ± 3.71 cm², $P=0.001$), diminished PS muscle area (8.49 ± 4.08 cm² vs 13.07 ± 3.86 cm², $P<0.001$), and exacerbated fatty infiltration in ES and MF ($57.28 \pm 5.63\%$ vs $43.40 \pm 14.93\%$, $P=0.001$). Furthermore, cement volume and distribution patterns significantly differed between groups, with less volume and concentrated cement dispersion demonstrating strong association with residual pain (5.67 ± 0.67 vs 5.03 ± 1.01 , $P=0.002$; 11.5% vs 73.6%, $P=0.001$). The difference between the two groups of age, sex, BMI, incidence of diabetes and hypertension, smoking, alcohol consumption, trauma severity, fractured level, FI% of PS and total area of ES and MF did not reach statistical significance.

Multivariate analysis identified four independent predictors of residual LBP ([Table 3](#)) including elevated VBQ scores (OR=85.212, 95% CI 7.006–1036.458; $P=0.001$), incremental kyphotic deformity progression (OR=1.139, 95% CI 1.017–1.276; $P=0.025$), diminished total PS area (OR=0.509, 95% CI 0.285–0.910; $P=0.023$), and increased FI% of ES and MF (OR=1.082, 95% CI 1.008–1.160; $P=0.028$). Notably, fascial injury (OR=4.222, $P=0.092$), muscle area of PS (OR=1.534, $P=0.119$) and cement dispersion abnormalities (OR=0.118, $P=0.052$) demonstrated marginal associations that approached but did not reach statistical significance.

Table 1 Baseline Demographic and Fracture Characteristics of Patients Undergoing PKP (N = 213)

Characteristic	N or Mean \pm SD
Age	70.88 \pm 8.58
Sex (male/female)	38/175
BMI (kg/m ²)	23.64 \pm 3.02
Fracture level	
Thoracic (T5-T9)	12
Thoracolumbar (T10-L2)	178
Lumbar (L3-5)	23

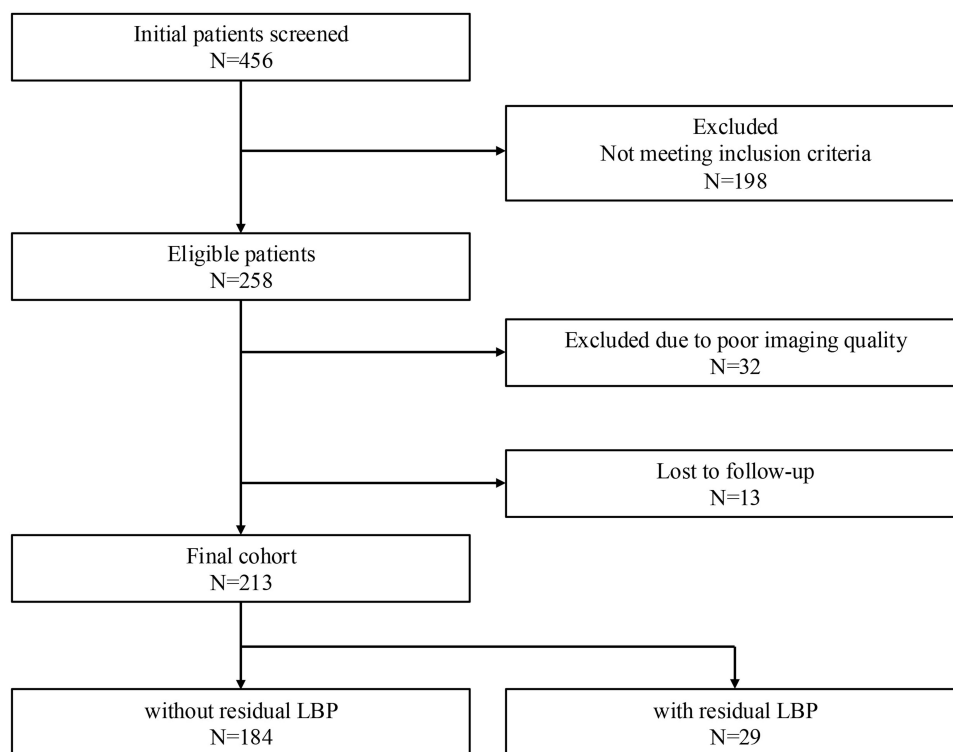


Figure 1 Flow chart of participants in the study.

Receiver operating characteristic (ROC) curve analysis was performed to assess the predictive value of the total PS area and the FI% of the ES and MF for residual LBP following PKP. The optimal cutoff values for each parameter were determined based on the maximum Youden Index (sensitivity + specificity - 1). The optimal cutoff value for the total PS

Table 2 Comparative Analysis of Clinical, Radiographic, and PVMs Parameters Between Patients with and without Residual LBP After PKP

Characteristic	Without Residual LBP (n=184)	With Residual LBP (n=29)	t or χ^2	P
Age (years)	70.56±8.42	72.86±9.41	-1.340	0.182
Sex (male/female)	32/152	6/23	0.348	0.840
BMI (kg/m ²)	23.66±2.49	23.52±3.45	0.224	0.823
Diabetes (Yes/No)	51/133	10/19	0.561	0.454
Hypertension (Yes/No)	59/125	10/19	0.067	0.796
Smoking (Yes/No)	40/144	5/24	0.304	0.581
Alcohol consumption (Yes/No)	27/157	4/25	0.016	0.901
VBQ	2.57±0.25	3.14±0.38	-10.373	0.001
Trauma severity			0.348	0.841
None/Slight	73	12		
Moderate	97	14		
Severe	14	3		
Fascia injury (Yes/No)	33/151	12/17	8.263	0.004
Fractured level			3.565	0.168
Thoracic (T5-T9)	11	1		
Thoracolumbar (T10-L2)	156	22		
Lumbar (L3-5)	17	6		

(Continued)

Table 2 (Continued).

Characteristic	Without Residual LBP (n=184)	With Residual LBP (n=29)	t or χ^2	P
Pre-operation kyphosis (°)	16.55±5.47	27.69±6.37	-9.964	0.001
Post-operation kyphosis (°)	6.70±4.80	16.03±6.69	-9.175	0.001
Last follow-up kyphosis (°)	8.17±4.67	20.48±6.21	-10.227	0.001
Cement volume (mL)	5.67±0.67	5.03±1.01	4.462	0.002
Cement diffusion (Diffused/Concentrated)	78/106	3/26	10.917	0.001
Total area of PS (cm ²)	16.15±3.71	10.74±4.23	7.158	0.001
Muscle area of PS (cm ²)	13.07±3.86	8.49±4.08	5.890	<0.001
FI% of PS (%)	19.65±10.37	21.83±11.19	-1.042	0.298
Total area of ES and MF (cm ²)	36.53±9.18	37.33±9.17	-0.441	0.660
Muscle area of ES and MF (cm ²)	20.58±7.43	15.86±3.98	3.348	0.001
FI% of ES and MF (%)	43.40±14.93	57.28±5.63	-4.943	0.001
Pre-operation VAS	7.40±0.97	7.69±0.96	-1.491	0.137
Post-operation VAS	2.51±0.65	2.90±0.72	-0.899	0.003
Last follow-up VAS	2.20±0.61	4.41±0.95	-16.814	0.001

Abbreviations: BMI, body mass index; VBQ, vertebral bone quality; PS, Psoas major; ES, Erector spinae; MF, Multifidus; FI%, fat infiltration percentage; VAS, visual analog scale.

Table 3 Binary Logistic Regression Model Identifying Independent Risk Factors for Residual LBP Following PKP

Characteristic	β	Wald	Sig.	Exp(β)	95% CI for EXP(β)
Fascia injury	1.440	2.843	0.092	4.222	0.791–22.521
VBQ	4.445	12.160	0.001	85.212	7.006–1036.458
Post-operation kyphosis	0.130	5.038	0.025	1.139	1.017–1.276
Cement diffusion model	-2.134	3.787	0.052	0.118	0.014–1.015
Muscle area of PS	0.491	2.430	0.119	1.634	0.881–3.031
Total area of PS	-0.675	5.195	0.023	0.509	0.285–0.910
FI% of ES and MF	0.078	4.813	0.028	1.082	1.008–1.160

area was 11.937 cm², with an area under the curve (AUC) of 0.877, a sensitivity of 86.1%, and a specificity of 76.9%. For the FI% of the ES and MF, the optimal cutoff was 49.782%, yielding an AUC of 0.792, a sensitivity of 89.7%, and a specificity of 62.5% (Figure 2). These findings suggest a moderate predictive accuracy for residual LBP after PKP based on these muscle parameters. Patients with an FI% of ES and MF \geq 49.782% or a total PS area \leq 11.937 are at an increased risk of developing residual LBP following single-level PKP. Figure 3 shows transverse lumbar MRI images from representative patients in each group. Patient A, without residual LBP, demonstrated a total PS cross-sectional area of 14.41 cm² and fat infiltration percentage (FI%) of 33.24% in the ES and MF muscles. In contrast, Patient B, with persistent residual LBP, exhibited marked paravertebral muscle degeneration, characterized by a significantly reduced total PS area (8.71 cm²) and elevated FI% (74.75%) in the ES and MF muscles. These comparative findings underscore the association between diminished muscle mass, advanced fat infiltration, and the clinical manifestation of residual pain following kyphoplasty.

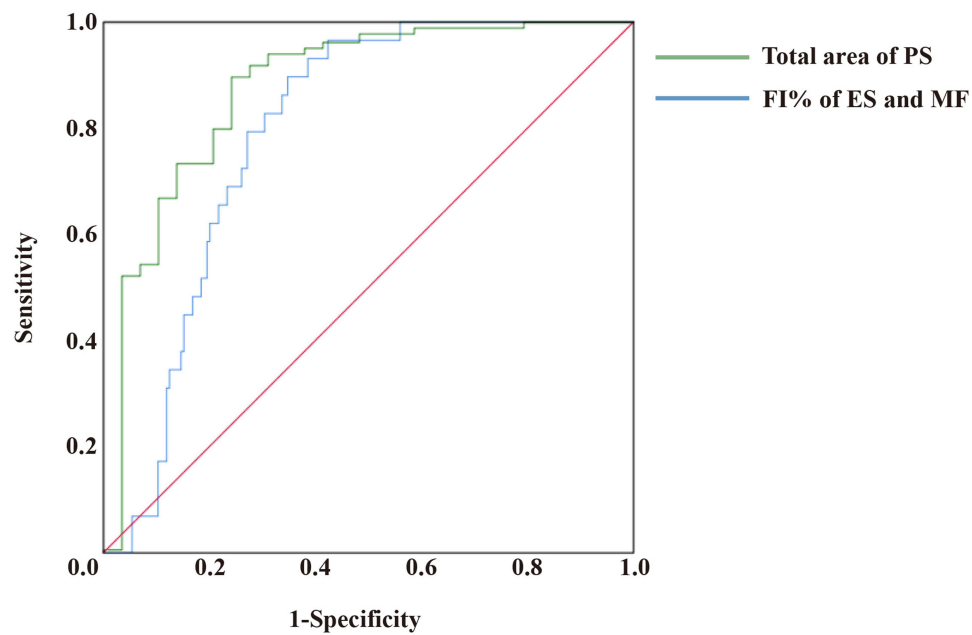


Figure 2 The ROC curve of total area of psoas major (PS) and fat infiltration percentage (FI%) of erector spinae (ES) and multifidus (MF) muscles for predicting residual low back pain (LBP).

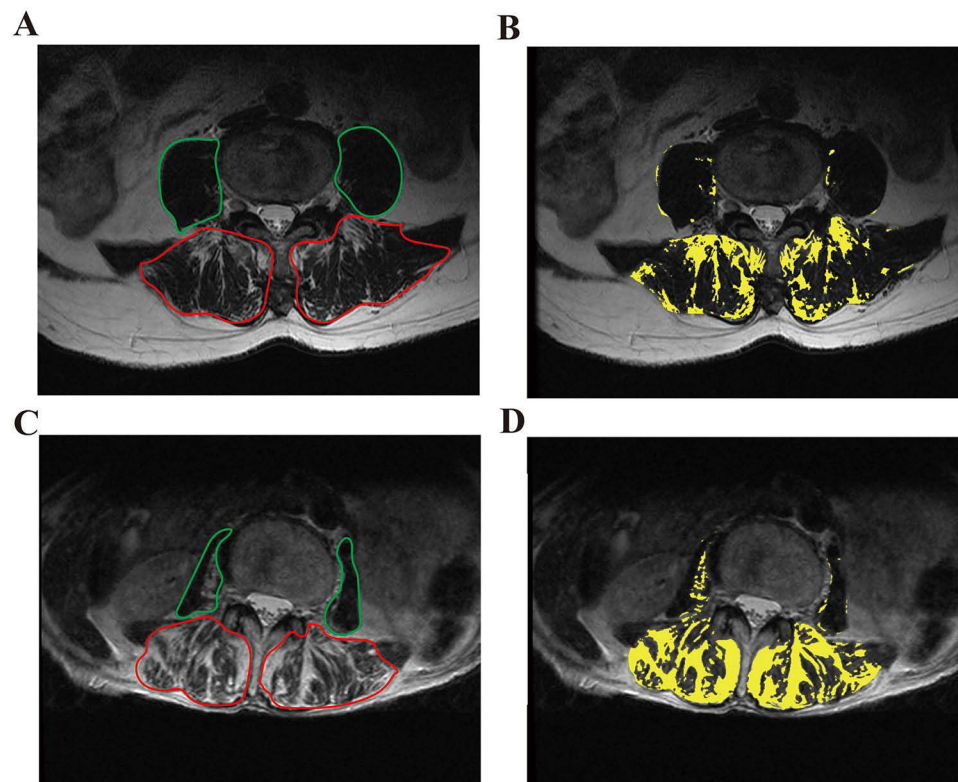


Figure 3 Transverse section of lumbar MRI at the L4 level of a 65-year-old female patient (**A** and **B**) demonstrating a total psoas major (PS) area (**A**, green) of 14.41 cm² and fat infiltration (**B**, yellow) percentage (FI%) of 33.24% in the erector spinae (ES) and multifidus (MF) muscles (**A**, red). Transverse section of lumbar MRI at the L4 level of a 69-year-old female patient (**C** and **D**) with reduced total PS (**C**, green) area (8.71 cm²) and elevated FI% (**D**, yellow) (74.75%) in the ES and MF muscles (**C**, red).

Discussion

In this study, a VAS score of 3.5 was established as the threshold for defining residual LBP. Our results demonstrated that despite the immediate and significant pain relief achieved postoperatively, 13.7% of patients experienced residual LBP. Previous studies have reported that the incidence of residual LBP following PKP ranges from 1.8% to 15.6%,^{7,19} which is consistent with our findings.

Residual LBP after PKP is multifactorial and may reflect structural, muscular, and procedural contributors. Consistent with previous research, our study also suggests that residual LBP after PKP arises from a triad of structural incompetence including poor bone quality (higher VBQ scores), persistent kyphosis, insufficient cement volume, suboptimal cement distribution and fascia injury. However, among these factors, only VBQ scores and post-operation kyphosis were identified as independent predictors of residual LBP after PKP. In this study patients with residual LBP experienced severe pain than those without on the first day after surgery as well. Our previous study also found that thoracolumbar fascia injury was associated with acute residual LBP but did not impact the long-term efficacy of PKP.²⁰ This may be attributed to the fact that, although PKP effectively alleviates fracture-related pain, damaged fascia and soft tissue edema serve as an additional source of pain in the acute phase, whereas fascial healing over time mitigates its long-term effects.

Aging is associated not only with a decline in bone mass but also with a significant reduction in muscle mass. Skeletal muscle mass and function typically begin to deteriorate after the age of 40, with postural and core-stabilizing muscles being affected earlier than other muscles.²¹ And its strength is influenced not only by muscle volume but also by muscle quality, with fat infiltration being a hallmark of muscle degeneration. Several studies have identified fat infiltration as a key contributor to muscle atrophy, diminished strength, and impaired spinal stability.^{22–25}

Our study revealed that patients experiencing residual LBP exhibited significantly higher FI% of the MF and ES compared to those without residual LBP ($P=0.001$). Binary logistic analysis further identified FI% of the MF and ES as an independent risk factor for residual LBP. These findings confirm the role of thoracolumbar extensor degeneration in compromising spinal support. The pathophysiological mechanisms linking PVMs degeneration to residual LBP are likely multifactorial. Fat infiltration reduces muscle strength, and consequently diminishes spinal support, and impairs postural control, thereby increasing the mechanical burden on passive stabilizing structures such as intervertebral discs, ligaments, and facet joints.²⁶ Muscle degeneration is often accompanied by intramuscular inflammation, fibrosis, and altered neuromuscular control, sensitizing peripheral nociceptors and promoting central sensitization-amplifying pain perception even in the absence of structural injury.²⁷ Over time, disuse-related atrophy and aberrant muscle recruitment may sustain a pain-deconditioning cycle, further contributing to long-term functional decline.^{24,26}

Interestingly, although the difference of FI% of PS between the two groups did not reach statistical significance, significant differences were observed in both the total cross-sectional area and muscle area of the PS between the two groups ($P=0.001$ and $P<0.001$, respectively). Further binary logistic analyze confirmed that diminished total area of PS (OR=0.509, $P=0.023$) played a role in residual LBP other than FI% and muscle area of PS. As a key stabilizer of the lumbar spine-bridging the diaphragm and pelvis-the PS contributes to resisting anterior pelvic tilt and maintaining lumbar stiffness.^{28,29} Its atrophy may compromise core stability, leading to biomechanical dysfunction. Previous study has demonstrated that patients with unilateral lumbar disc herniation exhibit significant reductions in the cross-sectional area of the PS, with a positive correlation to symptom duration.³⁰ Denaro et al³¹ further reported that a decrease in PS cross-sectional area was generally associated with higher VAS scores in patients with chronic LBP, with each square centimeter reduction in total PS area increasing the probability of reporting higher pain levels. These findings suggest that PS atrophy or dysfunction may lead to excessive or insufficient lumbar lordosis, resulting in compensatory pain.³²

In this study, the ROC analysis determined optimal cutoff values of PS area ≤ 11.937 cm² (AUC=0.877, sensitivity=86.1%, specificity=76.9%) and ES/MF FI% $\geq 49.78\%$ (AUC=0.792, sensitivity=89.7%, specificity=62.5%). Therefore, in the assessment and management of OVFs, it is essential to comprehensively evaluate the impact of FI% of ES and MF, as well as the reduction in the total cross-sectional area of the PS, on muscle structure and function. Postoperative rehabilitation aimed at enhancing PVMs strength to mitigate disuse atrophy may play a crucial role in

reducing residual pain and improving long-term outcomes. While our findings suggest that paravertebral muscle integrity may influence postoperative outcomes, the effectiveness of specific strengthening programs remains to be established. Future randomized controlled trials are needed to determine the most effective rehabilitation strategies for minimizing residual pain and promoting functional recovery after PKP.

This study has certain limitations. Its retrospective design precludes causal inferences, and the relatively modest sample size ($N = 213$) restricts the ability to perform detailed subgroup analyses. Additionally, the absence of long-term follow-up data limits insights into the trajectory of muscle degeneration and pain progression and certain lifestyle factors which were not consistently documented in the medical records, particularly nutritional status, pain sensitivity phenotypes, physical activity levels, medication adherence and dietary habits, may influence PVMs degeneration and, consequently, residual LBP. Despite these limitations, the strong association observed between PVMs degeneration-quantified via muscle area and fat infiltration-and residual LBP after PKP supports the validity of our conclusions. Future prospective studies should incorporate multimodal imaging, standardized rehabilitation protocols, and patient-reported outcomes to further validate these associations. Investigating targeted interventions, such as preoperative muscle strengthening programs, intraoperative navigation for precise cement injection, and anti-inflammatory therapies aimed at reducing fat infiltration, may help optimize PKP outcomes.

Conclusion

Residual low back pain (LBP) after percutaneous kyphoplasty (PKP) is a multifactorial condition associated with deteriorations in vertebral bone quality, insufficient kyphosis correction, and degeneration of the paravertebral muscles (PVMs). Our findings highlight the potential utility of MRI-based assessment of vertebral bone quality and paravertebral muscle composition as part of preoperative risk stratification. Intraoperative optimization of sagittal alignment, along with postoperative rehabilitation focusing on muscle preservation, functional recovery, and osteoporosis management, may contribute to improved outcomes. Referral to physical therapy after surgery may be considered, particularly in patients with marked PVMs atrophy or fat infiltration. While these recommendations are grounded in our current findings, further prospective studies are warranted to validate their clinical efficacy. Additionally, this study may serve as a reference framework for future research on imaging-based predictors of post-PKP pain outcomes.

Abbreviations

PVMs, paravertebral muscles; OVFs, osteoporotic vertebral fractures; PVP, percutaneous vertebroplasty; PKP, percutaneous kyphoplasty; LBP, low back pain; VAS, visual analog scale; BMI body mass index; MRI, magnetic resonance imaging; VBQ, vertebral bone quality; PS, psoas major; ES, erector spinae; MF, multifidus; FI%, fat infiltration percentage; ROC, Receiver Operating Characteristic.

Data Sharing Statement

The research data supporting this study are available from the corresponding author upon reasonable request.

Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

Funding

This work was supported by the National Natural Science Foundation of China (82472426); the Suzhou Basic Research Pilot Program (SSD2024048) and the Suzhou Medical Application Basic Research (SKY2023147).

Disclosure

The authors report no conflicts of interest in this work.

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