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Psoas–lumbar vertebral index and short-term pain relief after pulsed radiofrequency treatment of the dorsal root ganglion in postherpetic neuralgia in Korea: a retrospective pilot study

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Purpose: The psoas–lumbar vertebral index (PLVI), a marker of central sarcopenia measured using computed tomography (CT) or magnetic resonance imaging (MRI), has been linked to the development of postherpetic neuralgia (PHN). Whether PLVI predicts short-term pain relief after pulsed radiofrequency (pRF) treatment of the dorsal root ganglion (DRG) in established PHN remains unknown. This pilot study examined the association between PLVI and the reduction in numeric rating scale (NRS) pain scores 1 month after pRF.

Methods: Patients with PHN who underwent DRG pRF and had CT or MRI scans available for PLVI measurement were retrospectively reviewed. Correlations of the NRS reduction rate with PLVI and other variables were assessed, with 95% confidence intervals (CIs). Patients were divided into low- and high-PLVI groups using sex-specific medians and compared; multivariable regression was adjusted for sex. A supplementary responder analysis used thresholds of ≥30% and ≥50% reduction.

Results: In 19 patients, NRS scores decreased from 4.8±1.7 to 3.2±1.7, with a reduction rate of 29.3%±34.0%. PLVI was not significantly correlated with the reduction rate (r=−0.17; 95% CI, −0.58 to 0.31; P=0.49); neither the reduction rate (P=0.71) nor responder rates differed between the low- and high-PLVI groups. Male sex was associated with a smaller reduction (adjusted B=−47.2 percentage points; 95% CI, −90.9 to −3.5; P=0.036), although the unadjusted group comparison was not significant (Welch P=0.051) and 15 variables were tested without correction for multiple comparisons.

Conclusion: This pilot cohort showed no detectable association between PLVI and short-term pain relief after pRF; its small size precludes establishing the absence of an association. The exploratory sex difference warrants investigation in larger prospective studies with comprehensive sarcopenia assessment.

Keywords: Neuralgia, postherpetic; Herpes zoster; Pulsed radiofrequency treatment; Sarcopenia; Psoas muscles; Republic of Korea

Introduction

Background/rationale

Postherpetic neuralgia (PHN), the most common complication of herpes zoster (HZ), disproportionately affects older adults and reportedly occurs in 8%–24% of HZ cases [1]. Its pathogenesis is thought to involve peripheral and central sensitization after viral damage to the dorsal root ganglion (DRG) [2]. Pulsed radiofrequency (pRF) treatment of the DRG is an established intervention for PHN refractory to pharmacological therapy; when combined with standard medical treatment, it improves pain scores and reduces analgesic requirements [2]. Sarcopenia is an age-related loss of skeletal muscle mass and a core component of the frailty phenotype. It can be quantified using the psoas–lumbar vertebral index (PLVI), calculated as the mean cross-sectional area (CSA) of the bilateral psoas muscles divided by the L4 vertebral body area. This simple, reproducible marker requires no dedicated body-composition software [1,3]. Low PLVI has been linked to adverse outcomes in surgical and interventional settings, including degenerative spine surgery [3], morbidity and mortality in older patients with trauma or hip fracture [4], and postoperative acute kidney injury after hip fracture surgery [5].

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An et al. [1] reported that low PLVI independently predicted PHN development in patients aged ≥60 years with HZ, prompting the question of whether PLVI also predicts treatment response after PHN is established. Evidence from related interventional pain settings is mixed. Sarcopenia defined by the psoas muscle index was associated with slower pain improvement after radiofrequency ablation for lumbar facetogenic pain, but PLVI itself did not predict response in that cohort [6]. High-grade fatty infiltration of the cervical multifidus independently predicted a poor response to cervical epidural steroid injection [7], whereas PLVI correlated with a validated frailty scale but not with postoperative complications in a surgical spine cohort [3]. In addition to central sarcopenia, PHN risk and severity have been linked to delayed initiation of supplemental analgesia after HZ onset [8]. Once PHN is established, they have also been linked to central sensitization (brush-evoked allodynia), anxiety, depression, and younger age rather than the extent of peripheral nerve fiber damage [9].

Interventional treatments for established PHN vary in efficacy across targets and protocols. Studies of angulus costae pRF [10], computed tomography (CT)-guided thoracic nerve root coblation [11], DRG pRF combined with gabapentin [2], pRF with device-based pain assessment [12], and ultrasound-guided intercostal nerve pRF [13] all reported benefits over pharmacotherapy alone. However, response definitions and follow-up durations differed considerably, and none examined whether sarcopenia or frailty modifies treatment response. Recent evidence-based Korean Pain Society guidelines recommend pRF combined with medication for refractory PHN (recommendation grade: general use, strong; level of evidence: low), reserving spinal cord stimulation (SCS) as an elective next step after an inadequate response to pRF or nerve blocks [14]. Consistent with this hierarchy, a randomized trial in older patients with PHN found a substantially higher 12-month effective rate with short-term SCS than with pRF (79.3% vs. 42.1%) [15], although an earlier trial in a similar population found no significant difference between the techniques [16]. Because our cohort also consisted of older patients, these findings raise the possibility that age-related factors, potentially including central sarcopenia, influence the durability of the pRF response.

Objectives

Given the heterogeneity of procedures and sarcopenia markers, and the lack of prior studies specifically examining PLVI in relation to pRF outcomes in PHN, we conducted a retrospective pilot analysis. We tested the hypothesis that lower PLVI is associated with a smaller reduction in numeric rating scale (NRS) pain scores from baseline to 1 month after DRG pRF in patients with PHN.

Methods

Ethics statement

The study complied with the Declaration of Helsinki and was approved by the Institutional Review Board of Ewha Womans University Mokdong Hospital (approval no. 2026-05-038-002). The board waived informed consent because the study was retrospective.

Study design

This was a single-center retrospective analysis.

Setting

Patients were considered for inclusion if they attended the outpatient pain clinic of Ewha Womans University Mokdong Hospital between 2016 and 2026, received a PHN diagnosis based on clinical history and neurological examination, and subsequently underwent DRG pRF.

Participants

PHN was defined as dermatomal neuropathic pain that persisted after resolution of the HZ rash, consistent with contemporary clinical definitions requiring pain beyond approximately 1 month after rash onset [14]. Before pRF, all patients had pain refractory to first-line pharmacological therapy with gabapentinoids, tricyclic antidepressants, and/or lidocaine patches.

Patients without cross-sectional imaging for measurement of the lumbar psoas muscles and L4 vertebral body were excluded. For patients who underwent multiple pRF procedures during the study period, only the first treatment episode was included, yielding a final analytic sample of 19 patients.

Pulsed radiofrequency procedure

An experienced pain physician (W.J.K.) performed DRG pRF under fluoroscopic guidance at the spinal segment(s) corresponding to each patient's dermatomal pain distribution, using a technique consistent with previously described PHN protocols [2]. After satisfactory local anesthesia, a radiofrequency cannula (22G, 10-mm active tip) was advanced under fluoroscopic guidance toward the intervertebral foramen at the affected DRG level. Appropriate sensory stimulation thresholds were confirmed before treatment to ensure accurate needle placement. Radiofrequency current was then delivered in 3 treatment cycles at standard settings: output voltage, 45 V; pulse frequency, 2 Hz; pulse width, 20 ms; upper temperature limit, 42°C; and duration, 120 seconds per cycle. This uniformly applied routine technique was broadly consistent with previous studies of DRG pRF for PHN [2]. Treat-

ment was delivered at a thoracic level in 16 of the 19 patients (84.2%), at a sacral level in 2 (10.5%; both S1), and at a lumbar level in 1 (5.3%; L3); T3 and T4 were the most frequently treated individual levels (4 patients each). Concomitant pharmacological therapy with gabapentinoids and/or other adjunctive analgesics continued at the treating physician's discretion and was not standardized for this retrospective analysis.

Variables

The primary outcome was the NRS reduction rate, defined as the percentage decrease in the NRS pain score (0–10) from baseline to the 1-month follow-up after pRF: (NRS at baseline−NRS at 1 month)/NRS at baseline × 100. Baseline NRS scores were recorded immediately before pRF, and 1-month scores at each patient's first postprocedural follow-up visit. Absolute NRS reduction (baseline NRS−1-month NRS, in points) was also calculated as a secondary descriptive measure of pain relief. The NRS reduction rate was the dependent variable in all correlation, group-comparison, and regression analyses described below. Responders were additionally defined as patients with a ≥30% or ≥50% decrease in NRS score from baseline to 1 month.

Data sources and measurement

Central sarcopenia was quantified using PLVI, calculated as the mean CSA of the bilateral psoas muscles divided by the CSA of the L4 vertebral body at the inferior endplate level on axial CT or magnetic resonance imaging (MRI): PLVI = [(right psoas CSA+left psoas CSA)/2]/L4 vertebral body CSA. This followed previously published methodology [1,3,4]. PLVI cutoff strategies for sarcopenia vary across studies: some use the lowest sex-specific quartile (e.g., PLVI < 0.57 in men and < 0.43 in women in a hip fracture cohort) [5], whereas others, including this study, use a sex-specific median split because of smaller sample sizes, as detailed below.

PLVI was measured using CT in 15 patients (78.9%) and MRI in 4 (21.1%). The modalities were considered equivalent for CSA measurement, consistent with previous PLVI studies that did not distinguish between them. The median imaging-to-pRF interval was 9 months (range, 0–176 months). A single investigator performed all psoas and L4 vertebral body CSA measurements and was not otherwise involved in the patients' clinical care or outcome assessment.

Affected-side psoas CSA was examined separately as a univariate predictor instead of the bilateral mean. Because PHN is unilateral, localized disuse atrophy ipsilateral to the symptomatic dermatome was hypothesized to be more sensitive than the bilateral average. A sensitivity analysis using bilateral mean psoas CSA is also reported in Table 1 for consistency with the PLVI definition.

Statistical methods

Continuous variables are presented as means ± standard deviations (SDs). Associations between the NRS reduction rate and candidate variables (age, sex, height, weight, body mass index [BMI], rash-to-pRF interval, comorbidities, affected-side psoas CSA, bilateral mean psoas CSA, L4 vertebral body CSA, PLVI, number of pre-pRF diagnostic blocks, and medication use) were

Table 1. Univariate correlations with NRS reduction rate

Variable	Pearson r (95% CI)	P-value	Spearman ρ	P-value
Age (yr)	−0.062 (−0.502 to 0.403)	0.800	−0.140	0.567
Sex (male = 1)	−0.459 (−0.756 to −0.006)	0.048*	−0.440	0.059
Height	−0.405 (−0.726 to 0.060)	0.085	−0.411	0.080
Weight	−0.057 (−0.498 to 0.408)	0.818	−0.147	0.547
Body mass index	0.233 (−0.247 to 0.621)	0.337	0.101	0.681
Rash-to-pRF interval	−0.241 (−0.626 to 0.240)	0.321	0.090	0.713
Hypertension	0.324 (−0.153 to 0.678)	0.177	0.252	0.297
Diabetes mellitus	0.111 (−0.362 to 0.538)	0.652	0.111	0.650
Cardiovascular disease	0.219 (−0.262 to 0.612)	0.368	0.241	0.321
Affected-side psoas CSA	−0.295 (−0.661 to 0.184)	0.220	−0.365	0.125
Bilateral mean psoas CSA	−0.305 (−0.667 to 0.174)	0.205	−0.318	0.185
L4 vertebral body CSA	−0.429 (−0.739 to 0.031)	0.067	−0.424	0.071
PLVI	−0.170 (−0.579 to 0.308)	0.488	−0.216	0.374
No. of pre-pRF blocks	−0.032 (−0.479 to 0.429)	0.897	0.104	0.671
Medication use	−0.143 (−0.561 to 0.333)	0.560	−0.161	0.510

NRS, numeric rating scale; CI, confidence interval; pRF, pulsed radiofrequency; CSA, cross-sectional area; PLVI, psoas–lumbar vertebral index.
*P<0.05.

assessed using Pearson and Spearman rank correlations. For Pearson correlation coefficients, 95% confidence intervals (CIs) were calculated using the Fisher z-transformation. Baseline medication consumption was quantified with the Medication Quantification Scale III [17]. Because PLVI differs by sex, patients were also divided into low- and high-PLVI groups using sex-specific median values (male: 0.796; female: 0.576), following previous PLVI studies in the absence of a universally accepted cutoff [1,3,5]. Between-group differences were assessed using the independent-samples t-test with Welch correction and the Mann-Whitney U test; mean differences are reported with 95% CIs. A multivariable linear regression model included the NRS reduction rate as the dependent variable and PLVI and sex as covariates to assess the PLVI–outcome association after adjustment for sex. The dependent variable was expressed in percentage points, consistent with its definition under Variables. In a sensitivity analysis, PLVI was standardized as a within-sex z-score and the regression was repeated to assess potential confounding of the raw PLVI–outcome association by sex-related differences in body size. A supplementary responder analysis compared the proportions achieving ≥30% and ≥50% NRS reductions between the low- and high-PLVI groups using Fisher's exact test. A 2-sided P-value <0.05 was considered statistically significant. All analyses were performed using R ver. 4.3.3 (R Foundation for Statistical Computing).

Results

Participants

Nineteen patients (10 women and 9 men) formed the analytic sample. Their mean age was 75.7±8.6 years, mean BMI was 23.4±3.1 kg/m^2, and mean PLVI was 0.7±0.2 (Table 2).

Main results

NRS scores decreased from 4.8±1.7 at baseline to 3.2±1.7 at 1 month. The mean absolute reduction was 1.6±2.0 points, and the mean reduction rate was 29.3%±34.0% (Table 2).

Sex was the only variable significantly associated with the NRS reduction rate in univariate analysis (Pearson r=−0.459; 95% CI, −0.756 to −0.006; P=0.048); the mean reduction rate was greater in women (43.7%) than in men (13.3%; Welch t-test, P=0.051). L4 vertebral body CSA showed a nonsignificant trend toward an association (r=−0.429; 95% CI, −0.739–0.031; P=0.067). PLVI was not significantly correlated with the NRS reduction rate (Pearson r=−0.17; 95% CI, −0.58–0.31; P=0.49; Spearman ρ=−0.22, P=0.37). Substituting bilateral mean psoas CSA for affected-side CSA in a sensitivity analysis produced a materially similar nonsignificant result (r=−0.31; 95% CI, −0.67 to 0.17; P=0.21). No other variable reached statistical significance (Table 1, Fig. 1).

Table 2. Baseline characteristics of the study population (n=19)

Characteristic	Value
Age (yr)	75.7±8.6
Sex	
Male	9
Female	10
Height (cm)	160.2±8.8
Weight (kg)	60.2±10.4
Body mass index (kg/m^2)	23.4±3.1
Rash-to-pRF interval (mo)	15.2±22.6
Hypertension	6 (31.6)
Diabetes mellitus	2 (10.5)
Cardiovascular disease	1 (5.3)
Imaging modality for PLVI	
Computed tomography	15 (78.9)
Magnetic resonance imaging	4 (21.1)
Imaging-to-pRF interval (mo)	9 (0–176)
PLVI	0.7±0.2
Baseline NRS score	4.8±1.7
NRS score at 1 month	3.2±1.7
NRS reduction (points)	1.6±2.0
NRS reduction rate (%)	29.3±34.0

Values are presented as mean±standard deviation, number, number (%), or median (range).
pRF, pulsed radiofrequency; PLVI, psoas–lumbar vertebral index; NRS, numeric rating scale.

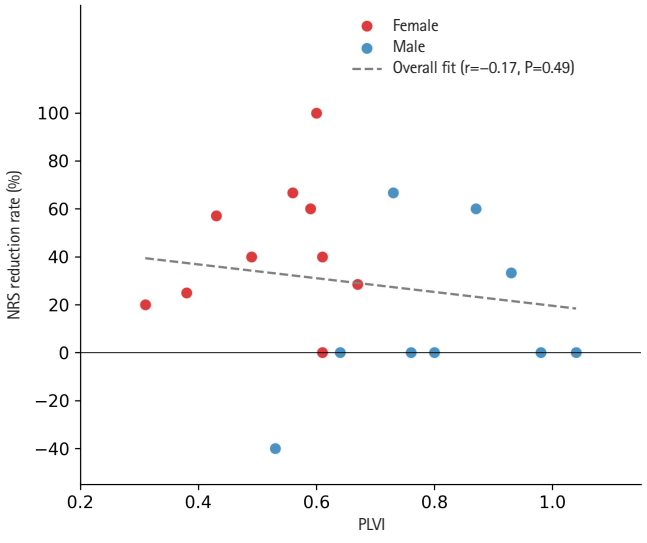


Fig. 1. Scatter plot of psoas–lumbar vertebral index (PLVI) versus numeric rating scale (NRS) reduction rate (%), stratified by sex. The dashed line shows the overall linear regression fit, which was not statistically significant.

Table 3. Comparison of NRS reduction between low- and high-PLVI groups defined using sex-specific median cutoffs

Group	NRS reduction (points)	NRS reduction rate (%)	Mean difference (95% CI)
Low PLVI (n = 9)	1.56 ± 1.51	26.2 ± 39.1	Reference
High PLVI (n = 10)	1.60 ± 2.55	32.2 ± 30.0	+6.0 (−27.9 to 39.9)
P-value (t-test)	0.963	0.712	
P-value (Mann-Whitney U test)	0.967	0.934	

The mean difference in NRS reduction rate is calculated as high minus low PLVI.
NRS, numeric rating scale; PLVI, psoas–lumbar vertebral index; CI, confidence interval.

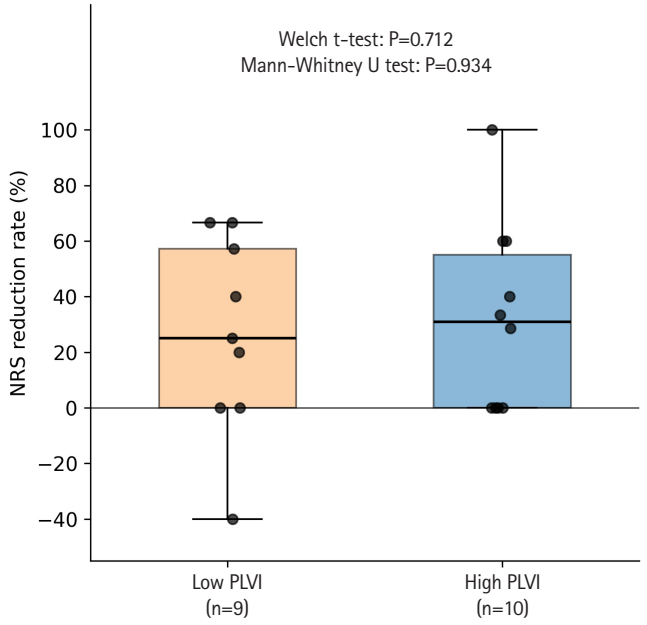


Fig. 2. Box plots of numeric rating scale (NRS) reduction rate (%) by psoas–lumbar vertebral index (PLVI) group, defined using sex-specific median cutoffs. Jittered points show individual patient values. The P-values for the Welch t-test and Mann-Whitney U test are shown.

After division into low- and high-PLVI groups using sex-specific median cutoffs, patients had similar absolute NRS reductions (low: 1.56 ± 1.51 points vs. high: 1.60 ± 2.55 points; P = 0.963) and reduction rates (low: 26.2% ± 39.1% vs. high: 32.2% ± 30.0%; mean difference, +6.0 percentage points; 95% CI, −27.9 to 39.9; P = 0.712 and P = 0.934 for the t-test and Mann-Whitney U test, respectively) (Table 3, Fig. 2). The supplementary responder analysis likewise showed no significant difference between the low- and high-PLVI groups at either the ≥ 30% threshold (4/9 [44.4%] vs. 5/10 [50.0%]; Fisher's exact P = 1.000) or the ≥ 50% threshold (3/9 [33.3%] vs. 3/10 [30.0%]; Fisher's exact P = 1.000).

In the multivariable model adjusted for sex, PLVI remained nonsignificantly associated with the NRS reduction rate (B = 59.2 percentage points; 95% CI, −52.8 to 171.1; P = 0.28), whereas male sex remained independently associated with a lower reduction rate (B = −47.2 percentage points; 95% CI, −90.9 to −3.5; P = 0.036). The model explained 26.8% of the variance (R^2 = 0.268; adjusted R^2 = 0.177), and the overall F-test approached but did not reach significance ($F(2,16) = 2.93$, P = 0.082) (Table 4). In the sensitivity analysis using within-sex standardized PLVI to address potential confounding by sex-related body size, the PLVI association remained nonsignificant (B = 8.1 percentage points per SD; 95% CI, −7.7 to 24.0; P = 0.29), while the male-sex association remained significant (B = −30.4; 95% CI, −60.5 to −0.3; P = 0.048).

Table 4. Multivariable linear regression of NRS reduction rate on PLVI, adjusted for sex

Predictor	B (% points)	95% CI	t	P-value
Intercept	12.68	−49.6 to 75.0	0.428	0.672
PLVI	59.16	−52.8 to 171.1	1.117	0.279
Sex (male)	−47.20	−90.9 to −3.5	−2.289	0.036*

The dependent variable is the NRS reduction rate in % points, as defined in the Methods. Coefficients (B) are unstandardized. R^2=0.268; adjusted R^2=0.177; overall F(2,16)=2.93, P=0.082.
NRS, numeric rating scale; PLVI, psoas–lumbar vertebral index; CI, confidence interval.
*P<0.05.

Discussion

Key results

In this retrospective pilot study of patients with established PHN, PLVI was not significantly associated with short-term pain relief after DRG pRF in either univariate or sex-adjusted analysis.

Interpretation

This result contrasts with An et al.'s finding that low PLVI strongly and independently predicted PHN development after HZ [1]. Together, the findings suggest that PLVI may be more informative for predicting PHN development than for predicting response to a specific intervention after PHN is established. This distinction between risk prediction and treatment-response prediction has

not previously been highlighted in this literature.

This interpretation requires an important qualification: PLVI is, at best, an imperfect proxy for sarcopenia. It measures only psoas CSA relative to the L4 vertebral body, a single structural measure, whereas consensus definitions of sarcopenia require assessment of muscle strength and physical performance as well as muscle mass [18]. The null finding may therefore reflect limitations of PLVI as a measurement tool rather than a true absence of a relationship between sarcopenia and pRF outcomes. The results strictly indicate only that PLVI does not predict short-term pain relief after pRF in this cohort. Whether sarcopenia, as a broader multidimensional construct, influences the pRF response remains unknown and was not addressed here.

Sex was notably associated with the NRS reduction rate in this cohort, with women experiencing greater pain relief than men in the univariate analysis and in the model adjusted for PLVI. Female sex has been identified as a risk factor for PHN development [1], but its relationship with the response to pRF is poorly characterized. The present finding warrants caution because of the small sample and the possibility of confounding or chance findings from multiple comparisons. Height and L4 vertebral body CSA both showed nonsignificant negative trends with outcome ($r \approx -0.4$) (Table 1) and were themselves strongly correlated with male sex (height vs. male sex, $r = 0.75$, $P < 0.001$; L4 vertebral body CSA vs. male sex, $r = 0.43$, $P = 0.064$). These patterns suggest that the trends likely reflect confounding by sex-related body size rather than independent associations. Consistent with this interpretation, standardizing PLVI as a within-sex z-score to remove sex-related differences in absolute body/muscle size left its association with outcome nonsignificant but reversed the point estimate (raw PLVI $r = -0.17$ vs. sex-standardized PLVI $r = +0.23$). This further supports sex, rather than PLVI itself, as the more consistent correlate of outcome in this cohort.

The stronger association of sex than of PLVI with pRF outcomes is consistent with the finding by Schlereth et al. [9] that PHN pain severity correlated with central sensitization (allodynia), anxiety, depression, and younger age, but not with peripheral nerve fiber loss on quantitative sensory testing. Together, these observations suggest that central and psychological factors may contribute more to pain trajectories and treatment response in established PHN than peripheral musculoskeletal markers such as PLVI. However, we did not directly measure allodynia, anxiety, or depression and cannot confirm this hypothesis. Similarly, Xing et al. [8] found that the timing of supplemental analgesic initiation after HZ onset strongly predicted subsequent PHN risk. This underscores the possibility that the acute-phase pain trajectory, largely determined before pRF, carries more prognostic weight for interventional outcomes than a static structural measurement at the time of the procedure. The rash-to-pRF interval was not significantly associated with outcome in our cohort, but this comparison was likely underpowered.

Comparison with previous studies

Our null finding is broadly consistent with work in other interventional pain settings. Kim et al. [3] found that PLVI correlated significantly with a validated frailty scale in older spine surgery patients but was not independently associated with postoperative complications or length of stay after multivariate adjustment. They concluded that PLVI may reflect frailty without strongly predicting procedural outcomes [3]. Yoo and Kim [6] likewise found that psoas muscle index-defined sarcopenia was associated with slower pain improvement after radiofrequency ablation for lumbar facetogenic pain, whereas PLVI did not predict response and responder proportions did not differ significantly by sarcopenia status. In contrast, Kwon et al. [7] identified high-grade fatty infiltration of the cervical multifidus, assessed qualitatively rather than by CSA, as an independent predictor of poor response to cervical epidural steroid injection. Together with our findings, these results suggest that muscle quality (fatty infiltration), muscle group, and procedure may be more relevant than psoas CSA alone when predicting response to a particular intervention. Psoas-based indices such as PLVI may instead be more consistently linked to broader constructs, including frailty and disease risk, than to short-term procedural response.

Our modest mean NRS reduction rate (29.3%) should also be considered alongside the wider literature on pRF and other interventions for PHN, although differences in response definitions and follow-up periods limit direct comparison. In a sham-controlled trial, Ke et al. [10] reported significant reductions in visual analog scale scores after angulus costae pRF that persisted to 6 months. Luo et al. [11] reported approximately 75% responders ($\geq 50\%$ pain relief) at 6 months after thoracic nerve root coblation. Huang et al. [2] reported a total effective rate of 86.2% with DRG pRF plus gabapentin versus 70.7% with gabapentin alone, and Wang et al. [12] found significantly greater reductions in device-measured pain degree with pRF plus pharmacological therapy than with pharmacological therapy alone. Our cohort's lower average response was measured only at 1 month using a continuous reduction rate rather than a binary responder threshold. It may reflect differences in patient selection, outcome definition, or follow-up duration and does not necessarily indicate inferior treatment efficacy.

This comparison is particularly relevant to our older cohort (mean age, 75.7 ± 8.6 years), in whom pRF benefits may be less

durable. In a randomized trial limited to older patients with PHN, Sheng et al. [15] found that the effective rate after pRF fell from 63.2% at 1 day to 42.1% at 12 months, whereas short-term SCS maintained a substantially higher rate (79.3%) and lower pregabalin requirements. However, an earlier trial in a similar population found no significant difference between SCS and pRF [16], leaving their relative durability in older patients with PHN unresolved. The Korean Pain Society guidelines reflect this pattern by recommending pRF for refractory PHN and reserving SCS as an elective option after an inadequate response [14]. Our 1-month follow-up was too short to assess durability. Longer follow-up is needed to investigate whether central sarcopenia beyond that captured by PLVI contributes to an attenuated pRF response in older patients and whether these patients might preferentially benefit from SCS.

Limitations

This study has several limitations. First, the small sample limited power to detect moderate associations and increased the risk of type II error; this study alone cannot exclude a true association between PLVI and treatment response. Second, PLVI is an imperfect proxy for sarcopenia because it measures only psoas CSA, without assessing muscle strength or physical performance, both integral to consensus diagnostic criteria [18]. The study can therefore conclude only that PLVI did not predict short-term pRF outcomes, not that sarcopenia is unrelated to treatment response. Third, no universally accepted PLVI cutoff exists [1,3]. We pragmatically used sex-specific medians, but other strategies, such as quartile-based or outcome-optimized thresholds, might produce different results. Fourth, the single-center retrospective design limits generalizability and precludes causal inference. Fifth, only short-term outcomes were assessed, so the influence of sarcopenia on longer-term PHN outcomes remains unknown; other studies have followed patients for up to 6 months [2,10,11]. Sixth, PLVI was measured by one investigator who was not otherwise involved in the patients' clinical care. Formal blinding to outcomes and intra- or inter-rater reliability of CSA measurements were not assessed; this methodological limitation should be addressed in prospective work. Seventh, imaging for PLVI measurement was obtained at widely varying intervals before pRF (median, 9 months; range, 0–176 months), often for unrelated clinical indications. Muscle mass can change substantially over these intervals, particularly the longer ones, so PLVI may not accurately represent sarcopenia status at the time of pRF, potentially misclassifying the exposure. Finally, psychological and central sensitization variables, including allodynia, anxiety, and depression, which correlate with PHN pain severity [9], were unavailable for adjustment and may confound the observed associations with sex and PLVI.

Suggestions

Larger prospective multicenter studies with standardized PLVI cutoffs, comprehensive sarcopenia assessment including muscle strength and physical performance, and longer follow-up are needed to clarify whether sarcopenia influences responses to interventional PHN treatment. Using the present estimates with 80% power and a 2-sided $\alpha = 0.05$, approximately 270 patients would be required to detect a PLVI association of the observed magnitude ($r \approx -0.17$), and approximately 35 to confirm the observed sex effect ($r \approx -0.46$).

Conclusion

This small retrospective cohort showed no significant association between PLVI and short-term pain relief after pRF for PHN, contrasting with the previously reported association between PLVI and PHN development. The small sample and wide CIs mean that this finding does not establish the absence of a true association and should not be extrapolated to clinical practice. Sex had a consistent, exploratory association with outcome across analyses, but confirmation in a larger, adequately powered cohort is required before it can be interpreted predictively. PLVI measures only psoas CSA and may incompletely represent the broader construct of sarcopenia. Thus, the findings indicate that PLVI specifically did not predict short-term pRF response in this pilot cohort, but they do not establish whether sarcopenia itself influences treatment outcomes. These findings are hypothesis-generating rather than definitive.

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Authors' contribution

Conceptualization: WJK. Data curation: EC. Formal analysis: SHY, WJK. Methodology: SHY, WJK. Validation: SHY, WJK. Project administration: WJK. Funding acquisition: none. Writing–original draft: SHY. Writing–review & editing: SHY, EC, WJK. Final approval of the manuscript: all authors.

Conflict of interest

No potential conflict of interest relevant to this article was reported.

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Data availability
The data analyzed in this study are available from the corresponding author upon reasonable request.

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Supplementary materials
None.

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