

Association Between Spinopelvic Sagittal Alignment and Femoral Cartilage Thickness in Knee Osteoarthritis: An Ultrasound-Based Observational Study

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ABSTRACT

Objective: Altered sagittal spinopelvic alignment may influence lower extremity joint loading, yet its relationship with femoral cartilage morphology in knee osteoarthritis (KOA) remains unclear. This study aimed to investigate the association between sagittal spinopelvic parameters and femoral cartilage thickness in individuals with symptomatic KOA using high-resolution ultrasonography (USG).

Methods: A total of 87 patients with symptomatic KOA, diagnosed per American College of Rheumatology criteria, were prospectively evaluated. Spinopelvic parameters—including pelvic incidence (PI), lumbar lordosis (LL), and PI–LL mismatch—were measured via lateral standing radiographs. Femoral cartilage thickness at the medial, intercondylar, and lateral compartments was assessed bilaterally using standardized musculoskeletal USG. Correlations were analyzed using Pearson's test and multivariate linear regression, with subgroup analysis based on PI–LL mismatch thresholds.

Results: PI–LL mismatch showed a weak negative correlation only with right intercondylar femoral cartilage thickness ($r = -0.203$, $p = 0.044$), while no significant correlations were found with medial or lateral compartments (all $p > 0.050$). Multivariate regression analysis confirmed LL as an independent predictor of left medial femoral cartilage thickness ($p = 0.042$), accounting for 7.8% of the variance. Each one-degree increase in LL was associated with a 0.012 millimeter increase in medial femoral cartilage thickness of the left knee. No significant associations were found between PI or LL alone and cartilage thickness in other compartments.

Conclusion: Increased PI–LL mismatch is associated with focal femoral cartilage thinning in KOA. These findings suggest that sagittal imbalance may affect joint morphology distal to the spine. Combined assessment of spinopelvic alignment and ultrasonographic cartilage thickness may offer added value in early-stage KOA evaluation and biomechanical risk stratification.

Keywords: femoral cartilage, knee osteoarthritis, lumbar lordosis, pelvic incidence, spinopelvic alignment, ultrasonography

INTRODUCTION

Knee osteoarthritis (KOA) is a major global health concern and a leading cause of disability, pain, and reduced quality of life, affecting hundreds of millions of adults worldwide [1]. Traditionally viewed as a localized joint disorder, KOA is now increasingly recognized as a multifactorial condition influenced by both systemic and biomechanical factors that extends beyond the knee joint. This aligns with emerging evidence suggesting that global sagittal alignment affects both spinal and peripheral joint mechanics [2].

Among biomechanical contributors to KOA, global sagittal balance, particularly in the spinopelvic axis, has drawn attention for its role in lower extremity loading and cartilage stress [3, 4]. Abnormal spinopelvic alignment, typically defined by the relationship between pelvic incidence (PI) and lumbar lordosis (LL), can disrupt postural equilibrium and gait mechanics [5]. In this context, sagittal imbalance represents a disruption of this harmony, and PI–LL mismatch is its quantifiable radiographic indicator. A PI–LL mismatch greater than 10° is widely accepted as a radiographic threshold for sagittal imbalance [3]. This imbalance often leads to pelvic retroversion and anterior trunk tilt, increasing mechanical load on the lower extremities during standing and walking [6, 7]. Anterior displacement of the

center of gravity alters ground reaction force vectors, increasing anterior shear forces along the tibia [8, 9]. These altered vectors disproportionately increase stress on the medial femorotibial compartment, which is highly susceptible to load-related degeneration [10]. This is further supported by recent evidence indicating that spinopelvic malalignment can modify lower extremity biomechanics and increase medial compartment loading [11, 12].

The medial knee compartment is biomechanically predisposed to varus stress, and sagittal imbalance may further exacerbate focal cartilage thinning due to uneven compartmental loading [13, 14]. Moreover, sagittal malalignment may lead to bilateral compensatory adaptations, not only in the symptomatic limb but also in the contralateral side, altering global lower limb mechanics [15, 16]. Oshima et al. reported that although total knee arthroplasty (TKA) improves sagittal alignment and symptoms, untreated contralateral knee pathology can diminish long-term postural gains [17]. Although most studies focus on postoperative cohorts or prosthesis-related outcomes, the structural consequences of native sagittal imbalance on natural knee joint morphology remain poorly characterized [16–18].

High-resolution musculoskeletal ultrasonography (USG) offers a validated, accessible, non-invasive, and cost-effective method for compartment-specific evaluation of femoral cartilage thickness [19]. USG can detect early-stage cartilage changes undetectable on radiographs and correlates well with magnetic resonance imaging (MRI) based cartilage quantification [20]. Although MRI remains the gold standard, USG provides a dynamic, real-time, and functional assessment of cartilage morphology, especially useful in load-bearing positions [21].

To our knowledge, no prior study has systematically evaluated the relationship between spinopelvic sagittal alignment and femoral cartilage morphology, using high-resolution USG in native, symptomatic knees. Clarifying this association is important for understanding whether sagittal imbalance contributes to compartmental chondral loss, particularly in the medial femorotibial joint. Such insights may inform alignment-targeted interventions and biomechanically tailored rehabilitation strategies. Therefore, this study aimed to investigate whether radiographic PI–LL mismatch is associated with changes in femoral cartilage thickness in the medial, intercondylar, and lateral compartments of symptomatic knees, using USG evaluation.

Main Points

- Pelvic incidence–lumbar lordosis (PI–LL) mismatch was significantly associated with reduced femoral cartilage thickness, particularly in the medial and intercondylar compartments, as measured by ultrasonography.
- Multivariate regression identified lumbar lordosis as an independent predictor of left medial femoral cartilage thickness, explaining 7.8% of the variance.
- Patients with higher PI–LL mismatch ($\text{PI–LL} \geq 10^\circ$) presented with more severe pain and functional impairment, highlighting the clinical relevance of sagittal imbalance.
- Combined radiographic and ultrasonographic evaluation of sagittal alignment and cartilage morphology may provide added clinical value for early diagnosis, risk stratification, and rehabilitation planning in knee osteoarthritis.

MATERIALS AND METHODS

Study Design and Patient Selection

This prospective observational study was conducted between July 2023 and April 2025 at a Başakşehir Çam and Sakura City Hospital, İstanbul. The study protocol was approved by the Clinical Research Ethics Committee of Istanbul Medipol University (Approval date: June 8, 2023; Approval No: 510/2023) and conducted in accordance with the Declaration of Helsinki. Written informed consent was obtained from all participants prior to enrollment.

A total of 238 patients aged 40 years and older who presented with chronic knee pain persisting for more than 12 weeks were initially screened following referral from musculoskeletal outpatient clinics with suspected KOA. Following a standardised clinical assessment, radiographic evaluation, and the application of predefined eligibility criteria, 87 participants were considered eligible and included in the final analysis (Figure 1).

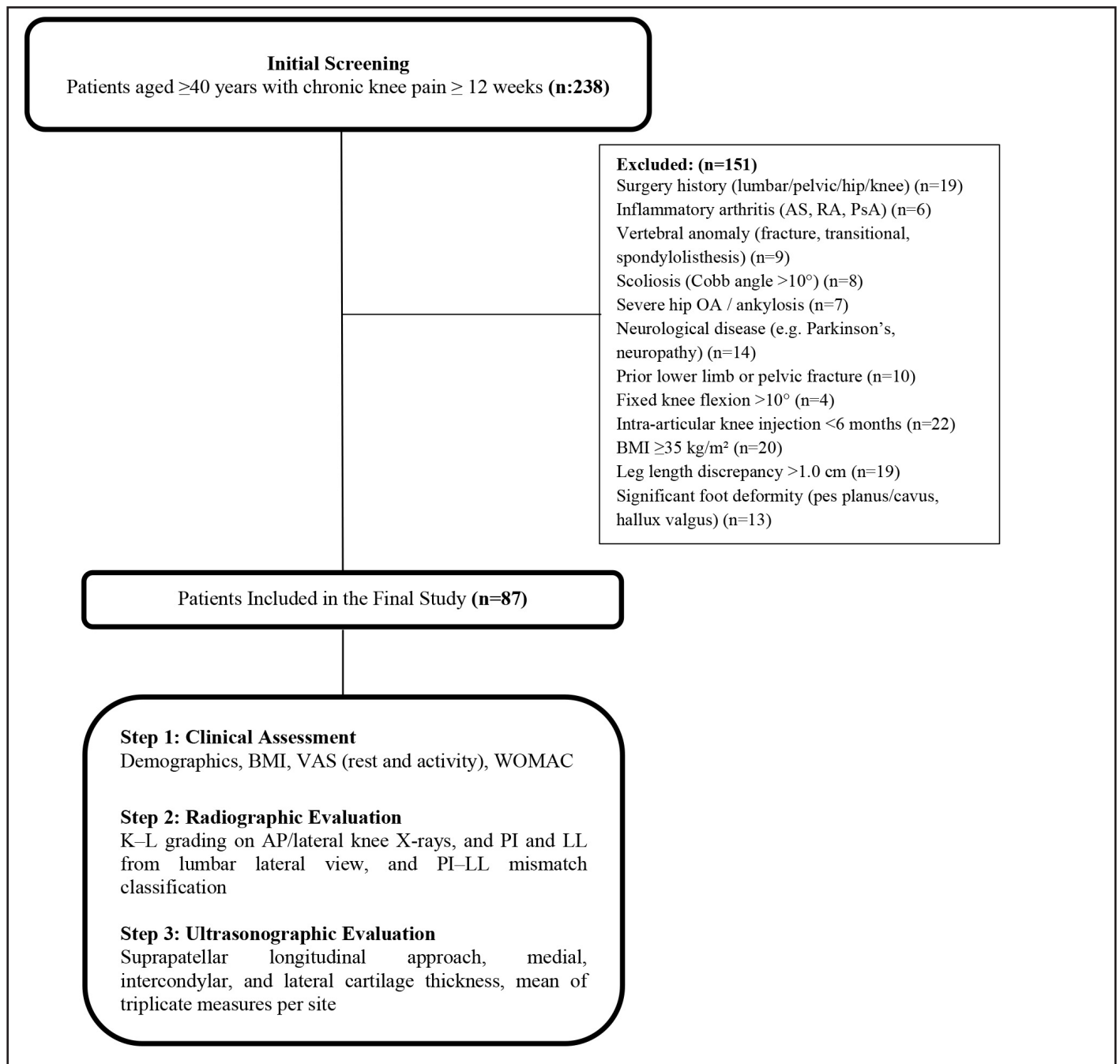


Figure 1. Flowchart of the study

Inclusion criteria comprised: (1) age ≥ 40 years; (2) diagnosis of KOA with Kellgren Lawrence (K–L) grade ≥ 1 on standing anteroposterior (AP) and lateral knee radiographs; (3) availability of standing lateral lumbar spine radiographs suitable for spinopelvic parameter measurement; and (4) availability of long-standing full-limb radiographs (hip–knee–ankle) for objective leg length assessment.

To ensure biomechanical homogeneity and minimize postural confounding, patients were excluded if they had: (1) history of surgery involving the lumbar spine, pelvis, hip, or knee; (2) inflammatory arthritis (e.g., rheumatoid arthritis, ankylosing spondylitis, psoriatic arthritis); (3) vertebral abnormalities including compression fractures, transitional lumbosacral anatomy, or spondylolisthesis; (4) scoliosis with a Cobb angle $>10^\circ$; (5) advanced hip osteoarthritis (OA), ankylosis, or sacroiliac joint fusion; (6) neuromuscular disorders such as Parkinson's disease, cerebrovascular disease, or peripheral neuropathy; (7) any history of lower extremity or pelvic fractures; (8) fixed knee flexion deformity exceeding 10° ; (9) history of intra-articular knee injection within the past 6 months; (10) body mass index (BMI) ≥ 35 kg/m², given its impact on sagittal alignment and USG accuracy; (11) radiographically confirmed leg length discrepancy >1.0 cm on long-standing alignment views; and (12) clinically significant foot deformities (e.g., rigid pes planus or cavus, hallux valgus requiring orthotic correction, or forefoot

structural anomalies) due to their potential influence on kinetic chain loading and postural dynamics.

Radiographic Evaluation

KOA severity was assessed using the K–L grading system on standardized weight-bearing AP and lateral knee radiographs. Spinopelvic alignment parameters were measured on lateral standing lumbar spine radiographs. PI was defined as the angle between a perpendicular to the sacral endplate and a line connecting its midpoint to the femoral head axis. LL was measured via the Cobb method between the superior endplates of L1 and S1. The PI–LL mismatch was calculated as the absolute difference between these two angles. Based on established thresholds, participants were categorized as having normal alignment (PI–LL $<10^\circ$) or sagittal imbalance (PI–LL $\geq 10^\circ$). Radiographic measurements were independently performed by two physiatrists with more than five years of clinical experience, both blinded to the clinical and ultrasonographic data.

Ultrasonographic Evaluation

Femoral cartilage thickness was assessed using a high-resolution ultrasound (US) system (Hitachi Aloka Arietta 65, Hitachi Medical Systems, Tokyo, Japan) equipped with a 6–18 MHz linear transducer. All scans were performed by a single physiatrist with over five years of musculoskeletal US experience, using a standardized suprapatellar longitudinal



Figure 2 (A) Standard probe positioning during suprapatellar longitudinal ultrasonographic (USG) assessment of femoral cartilage thickness in maximal knee flexion. (B). Representative high-resolution USG image showing articular cartilage layers over the medial femoral condyle, intercondylar notch, and lateral femoral condyle. Measurements were obtained perpendicularly to the subchondral bone interface at each region.

approach (Figure 2A and B). Participants were examined in the supine position with the knee flexed to approximately 120° to ensure a consistent acoustic window and reproducible cartilage visualization. Longitudinal images were acquired from three predefined anatomical landmarks: the medial femoral condyle, intercondylar notch, and lateral femoral condyle. At each site, measurements were obtained perpendicularly to the subchondral bone interface. Three consecutive measurements were performed at each site, and the mean value was recorded for analysis. The average of the three anatomical locations was used to calculate overall femoral cartilage thickness. To minimize the potential impact of diurnal variation in cartilage hydration and joint loading, all USG assessments were conducted between 09:00 and 11:00 am under standardized room temperature and patient resting conditions. Although a single operator performed all assessments, intraobserver reliability was evaluated in a random subsample of 20 participants who were reassessed 10 days later under blinded conditions, and measurements were repeated on the same knee side as in the initial assessment for each participant. ICC, computed using a two-way mixed-effects model with absolute agreement, exceeded 0.90 for all anatomical sites, indicating excellent measurement reproducibility [22].

Clinical Assessments

All participants underwent standardized clinical evaluation on the same day as imaging. The following parameters were recorded: age, sex, body mass index (BMI), symptom duration, pain severity using the Visual Analog Scale (VAS; at rest and during activity), and functional status using the Western Ontario and McMaster Universities Osteoarthritis Index (WOMAC), for which a validated Turkish version was used [23].

Statistical Analysis

All statistical analyses were performed using IBM SPSS Statistics, version 26.0 (IBM Corp., Armonk, NY, USA). Descriptive statistics were reported as mean \pm standard deviation (SD), median (min–max), and frequencies (n, %) where appropriate. The normality of continuous variables was assessed using the Shapiro–Wilk test. Since most variables violated the assumptions of normal distribution, non-parametric statistical methods were applied. Paired comparisons of femoral cartilage thickness between the right and left knees were conducted using the paired-samples t-test. Correlations between continuous variables were examined using the Pearson correlation coefficient. To explore the independent associations

of lumbar lordosis (LL) and pelvic incidence (PI) with femoral cartilage thickness, multiple linear regression analyses were performed. The assumptions of multicollinearity were evaluated using tolerance values and variance inflation factors (VIFs), while Durbin–Watson statistics were calculated to assess autocorrelation in residuals. The normality of residuals was visually examined using Q–Q plots. A two-tailed p-value <0.05 was considered indicative of statistical significance throughout all analyses.

RESULTS

Among the 87 patients included in the study, 66 (75.9%) were female and 21 (24.1%) were male. The mean age was 56.93 ± 8.38 years. The mean BMI was 30.75 ± 4.76 kg/m². The median pain duration was 60 months (range: 1–460). The median VAS scores were 4 at rest and 8 during activity. The mean WOMAC index score was 45.85 ± 18.96 . According to the K–L system, grade 2 was the most prevalent (n = 44, 50.6%). The mean PI was $60.28 \pm 10.21^\circ$, LL was $43.39 \pm 12.01^\circ$, and PI–LL mismatch was $16.89 \pm 12.88^\circ$ (Table 1).

As presented in Table 2, no statistically significant differences were observed between the right and left knees in femoral cartilage thickness measurements across the medial, intercondylar, and lateral regions (all $p > 0.05$).

As shown in Table 3, a statistically significant negative correlation was observed between the K-L grade and the medial femoral cartilage thickness of the left knee ($p = 0.002$). Additionally, cartilage thickness at the intercondylar notch of both knees showed a significant inverse correlation with VAS activity scores ($p = 0.030$ for right, $p = 0.038$ for left). A positive correlation was found between lumbar lordosis and right intercondylar cartilage thickness ($p = 0.015$), and between PI–LL mismatch and the same region ($p = 0.044$). Other parameters did not show statistically significant differences ($p > 0.05$) (Table 3).

Table 4 presents the results of the multiple linear regression analysis assessing the effect of lumbar lordosis and pelvic incidence measurements on femoral cartilage thickness. Separate models were constructed for each of the six regional cartilage thickness measurements. Among these, five models did not reach statistical significance ($p > 0.05$). However, the model for the medial compartment of the left femur was statistically significant ($F = 3.353$, $p = 0.042$).

Table 1. Baseline demographic, clinical and radiographic characteristics of the patients

Variables	n (%)
Male	21 (%24.1)
Female	66 (%75.9)
Age, (years)	
Mean \pm SD	56.93 \pm 8.38
Median (min-max)	56 (45-77)
BMI, (kg/m²)	
Mean \pm SD	30.75 \pm 4.76
Median (min-max)	30.8 (19.2-45.6)
Pain duration, (months)	
Mean \pm SD	90.89 \pm 100.64
Median (min-max)	60 (1-460)
VAS (Rest)	
Mean \pm SD	4.37 \pm 2.48
Median (min-max)	4 (0-10)
VAS (Activity)	
Mean \pm SD	7.67 \pm 2.39
Median (min-max)	8 (0-10)
WOMAC	
Mean \pm SD	45.85 \pm 18.96
Median (min-max)	46.87 (3.12-87.5)
Kellgren Lawrence Grade	
Grade 1	23 (%26.4)
Grade 2	44 (%50.6)
Grade 3	18 (%20.7)
Grade 4	2 (%2.3)
Pelvic Incidence (°)	
Mean \pm SD	60.28 \pm 10.21
Median (min-max)	59.3 (40.72-91)
Lumbar Lordosis (°)	
Mean \pm SD	43.39 \pm 12.01
Median (min-max)	43.49 (6.49-68.5)
PI-LL Mismatch (°)	
Mean \pm SD	16.89 \pm 12.88
Median (min-max)	15.08 (-6.29-54.5)

Data are presented as mean \pm standard deviation or median (minimum–maximum) for continuous variables, and as number (percentage) for categorical variables. BMI: Body Mass Index, VAS: Visual Analog Scale, WOMAC: Western Ontario and

McMaster Universities Osteoarthritis Index.

Collinearity diagnostics showed a tolerance value of 0.886 and a variance inflation factor (VIF) of 1.128, indicating no multicollinearity concern. The presence of autocorrelation in residuals was evaluated using the Durbin–Watson statistic, which was calculated as 1.865, suggesting acceptable independence of errors. Normality of residuals was verified through Q–Q plots, which demonstrated a normal distribution.

According to the model, each one-degree increase in lumbar lordosis was associated with a 0.012 millimeter (mm) increase in the medial femoral cartilage thickness of the left knee.

Collectively, lumbar lordosis and pelvic incidence explained 7.8% of the variance in the left medial femoral cartilage thickness (Table 4 and Figure 3).

DISCUSSION

The present prospective study investigated the association between spinopelvic sagittal alignment, specifically the PI–LL mismatch, and femoral cartilage thickness in patients with symptomatic KOA using high-resolution USG. The principal findings indicate that sagittal imbalance is associated with focal cartilage thinning, particularly in the medial and intercondylar compartments. It is noteworthy that LL emerged as a region and laterality-specific predictor of cartilage morphology.

A statistically significant positive correlation was observed between LL and right intercondylar cartilage thickness; however, this association did not persist in the multivariate regression model that included both LL and PI. This divergence highlights the limitations of bivariate analyses in reflecting intricate inter-variable relationships and underscores the value of multivariate modeling in adjusting for shared variance. Even though multicollinearity was excluded based on acceptable diagnostic thresholds (tolerance = 0.886; VIF = 1.128), the low explanatory power of the model ($R^2 = 0.021$) suggests that other biomechanical or postural determinants, such as anterior pelvic tilt, pelvic obliquity, or coronal malalignment, may account for the unexplained variance in cartilage morphology [2, 5, 6].

More importantly, LL emerged as a statistically significant independent predictor of cartilage thickness in the left medial femoral condyle ($F = 3.353$, $p = 0.042$), despite the absence of a significant bivariate correlation. This suggests that multivariate regression can reveal associations that are not apparent in simple

Table 2. Comparison of Femoral Cartilage Thickness (Right vs. Left Knee)

Femoral Subregion	Region		t	p
	Right	Left		
Medial				
Mean ± SD	1.74 ± 0.90	1.54 ± 0.48	1.906	0.060
M (min-max)	1.7 (0.5-8)	1.6 (0.5-2.8)		
Intercondylar				
Mean ± SD	2.23 ± 0.52	2.18 ± 0.52	0.747	0.457
M (min-max)	2.2 (1.3-4.2)	2.2 (1.1-3.6)		
Lateral				
Mean ± SD	1.75 ± 0.39	1.80 ± 0.46	-0.837	0.405
M (min-max)	1.7 (0.5-3.1)	1.8 (0.5-3.1)		

Paired samples t-test was applied. Statistics are presented as mean ± standard deviation and median (minimum–maximum) values.

Table 3. Correlation Between Regional Femoral Cartilage Thickness, Clinical Outcomes, and Spinopelvic Parameters

Parameters	Medial		Intercondylar		Lateral	
	Right	Left	Right	Left	Right	Left
BMI, (kg/m ²)	<i>r</i> = -0.105 <i>p</i> = 0.310	<i>r</i> = -0.081 <i>p</i> = 0.429	<i>r</i> = -0.069 <i>p</i> = 0.498	<i>r</i> = -0.042 <i>p</i> = 0.669	<i>r</i> = -0.123 <i>p</i> = 0.226	<i>r</i> = 0.098 <i>p</i> = 0.345
VAS (Rest)	<i>r</i> = 0.168 <i>p</i> = 0.121	<i>r</i> = -0.038 <i>p</i> = 0.726	<i>r</i> = -0.179 <i>p</i> = 0.098	<i>r</i> = -0.047 <i>p</i> = 0.665	<i>r</i> = -0.023 <i>p</i> = 0.830	<i>r</i> = 0.129 <i>p</i> = 0.233
VAS (Activity)	<i>r</i> = 0.075 <i>p</i> = 0.488	<i>r</i> = -0.116 <i>p</i> = 0.284	<i>r</i> = -0.233 <i>p</i> = 0.030	<i>r</i> = -0.223 <i>p</i> = 0.038	<i>r</i> = -0.052 <i>p</i> = 0.633	<i>r</i> = -0.010 <i>p</i> = 0.930
WOMAC	<i>r</i> = -0.015 <i>p</i> = 0.889	<i>r</i> = -0.133 <i>p</i> = 0.222	<i>r</i> = -0.023 <i>p</i> = 0.830	<i>r</i> = 0.056 <i>p</i> = 0.610	<i>r</i> = -0.200 <i>p</i> = 0.066	<i>r</i> = 0.022 <i>p</i> = 0.843
K-L Grade	<i>r</i> = -0.025 <i>p</i> = 0.818	<i>r</i> = -0.321 <i>p</i> = 0.002	<i>r</i> = -0.087 <i>p</i> = 0.425	<i>r</i> = -0.128 <i>p</i> = 0.239	<i>r</i> = 0.042 <i>p</i> = 0.700	<i>r</i> = -0.142 <i>p</i> = 0.191
Pelvic Incidence	<i>r</i> = 0.114 <i>p</i> = 0.293	<i>r</i> = 0.047 <i>p</i> = 0.666	<i>r</i> = 0.062 <i>p</i> = 0.571	<i>r</i> = -0.089 <i>p</i> = 0.410	<i>r</i> = 0.191 <i>p</i> = 0.077	<i>r</i> = 0.056 <i>p</i> = 0.609
Lumbar Lordosis	<i>r</i> = 0.116 <i>p</i> = 0.285	<i>r</i> = 0.028 <i>p</i> = 0.795	<i>r</i> = 0.259 <i>p</i> = 0.015	<i>r</i> = 0.076 <i>p</i> = 0.486	<i>r</i> = 0.116 <i>p</i> = 0.285	<i>r</i> = 0.016 <i>p</i> = 0.886
PI-LL Mismatch	<i>r</i> = -0.018 <i>p</i> = 0.871	<i>r</i> = 0.011 <i>p</i> = 0.920	<i>r</i> = -0.203 <i>p</i> = 0.044	<i>r</i> = -0.142 <i>p</i> = 0.191	<i>r</i> = 0.043 <i>p</i> = 0.692	<i>r</i> = 0.030 <i>p</i> = 0.785

r: Pearson correlation coefficient. Bolded values indicate statistically significant results (*p* < 0.05). BMI: Body Mass Index, VAS: Visual Analog Scale, WOMAC: Western Ontario and McMaster Universities Osteoarthritis Index, K-L: Kellgren Lawrence

Table 4. Impact of Lumbar Lordosis and Pelvic Incidence on Regional Femoral Cartilage Thickness: Multivariate Regression Analysis

Dependent Variable	Independent Variables	Right		Left	
		β (%95 CI)	Model Significance	β (%95 CI)	Model Significance
Medial	Intercept	1.009 (-0.194 - 2.212)	$F = 0.847$	1.809 (1.124 - 2.494)	$F = 3.353$
	Pelvic incidence	0.007 (-0.013 - 0.028)	$p = 0.432$	-0.001 (-0.013-0.01)	$p = 0.042$
	Lumbar lordosis	0.007 (-0.011 - 0.024)	$R^2 = 0.020$	0.012 (0.002 - 0.021) **	$R^2 = 0.078$
Lateral	Intercept	1.281 (0.767 - 1.795)	$F = 1.721$	1.397 (0.746 - 2.048)	$F = 0.100$
	Pelvic incidence	0.006 (-0.002 - 0.015)	$p = 0.185$	0.002 (-0.009 - 0.013)	$p = 0.905$
	Lumbar lordosis	0.002 (-0.005 - 0.009)	$R^2 = 0.039$	0.001 (-0.009 - 0.01)	$R^2 = 0.002$
Intercondylar	Intercept	2.357 (1.66 - 3.054)	$F = 0.885$	1.649 (1.032 - 2.266)	$F = 0.131$
	Pelvic incidence	-0.007 (-0.018-0.005)	$p = 0.416$	0.003 (-0.008 - 0.013)	$p = 0.877$
	Lumbar lordosis	0.005 (-0.005 - 0.015)	$R^2 = 0.021$	0.001 (-0.009 - 0.009)	$R^2 = 0.003$

Regression analysis (F -test); β : Regression coefficient, CI: Confidence Interval, R^2 : Coefficient of determination. Bold values indicate statistically significant results ($p < 0.05$).

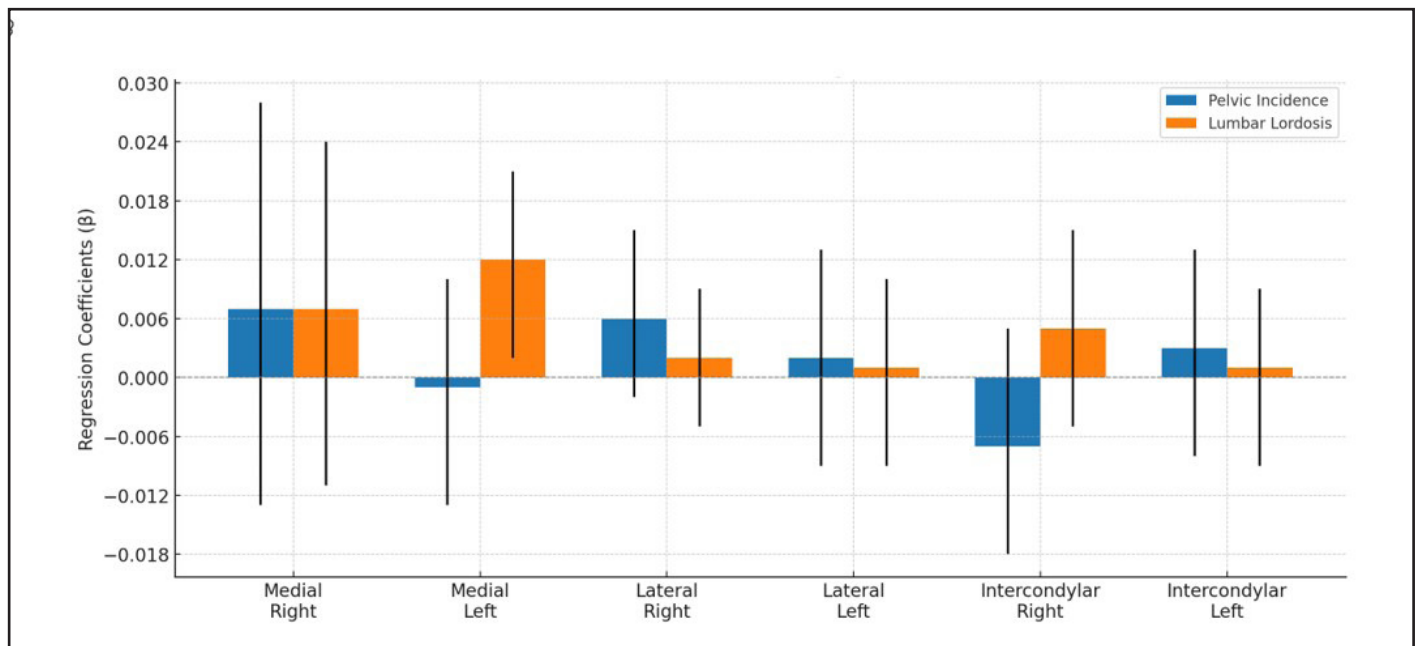


Figure 3. The Effect of Pelvic Incidence and Lumbar Lordosis on Femoral Cartilage Thickness

correlation analyses, by adjusting for confounding variables. According to the model, each one-degree increase in LL was associated with a 0.012 mm increase in medial femoral cartilage thickness, with LL and PI collectively explaining 7.8% of the variance. Model assumptions were verified through Q–Q plots and the Durbin–Watson statistic (1.865), confirming normality and residual independence. According to the model, each one-

degree increase in LL was associated with a 0.012 mm increase in medial femoral cartilage thickness, with LL and PI collectively explaining 7.8% of the variance. Model assumptions were verified through Q–Q plots and the Durbin–Watson statistic (1.865), confirming normality and residual independence. Nevertheless, the low explanatory power ($R^2 = 0.078$) indicates that spinopelvic parameters account for only a small portion

of cartilage variability, implying that other biomechanical or systemic factors may also contribute to this relationship.

Biomechanically, the medial femoral condyle bears the highest compressive loads during gait and is typically the first region to undergo degeneration in KOA [1, 13]. An increased lumbar lordotic angle may anteriorly shift the body's center of mass, influencing lumbopelvic tilt and femorotibial kinematics, particularly on the non-dominant limb, which serves a stabilizing function during gait [2, 6, 18]. This asymmetrical loading pattern may explain the preferential association between LL and left-sided medial cartilage morphology. Our findings support the hypothesis that subclinical sagittal deviations may propagate biomechanical stress through the kinematic chain and impact joint-level cartilage health, even in the absence of overt spinal pathology [3, 15, 16].

Furthermore, the laterality-specific findings align with existing literature suggesting biomechanical asymmetries between lower limbs during gait, particularly in load distribution and muscular activation patterns [8, 24]. The dominant limb is primarily involved in propulsion, whereas the non-dominant limb plays a greater role in postural control and stabilization [24]. In the presence of sagittal imbalance, such asymmetries may exaggerate limb-specific compensations, potentially altering joint-level cartilage homeostasis [15-17]. Importantly, the use of high-resolution USG enabled the detection of subtle cartilage variations, highlighting its value as a dynamic, cost-effective modality for early-stage OA evaluation [20, 21].

To the best of our knowledge, no previous study has combined high-resolution USG with sagittal spinopelvic alignment parameters to evaluate their association with femoral cartilage morphology in native knees. By integrating radiographic sagittal metrics with region-specific cartilage quantification, our findings advance the understanding of KOA as a manifestation of broader kinematic dysfunction. The identification of lumbar lordosis as an independent, laterality-specific determinant of medial cartilage thickness supports the hypothesis that even subclinical deviations in proximal alignment can propagate mechanical stress through the spine–pelvis–knee axis. Prior studies have predominantly examined the relationship between sagittal alignment and joint biomechanics in the context of spine or hip pathologies, with limited attention to how native spinopelvic parameters may influence femoral cartilage morphology in the knee joint [11, 12, 25]. Moreover, USG's

ability to detect subtle focal cartilage changes, particularly in compartments vulnerable to asymmetrical loading, reinforces its value as a dynamic, non-invasive modality for the early monitoring of disease.

Early identification of sagittal imbalance may allow clinicians to implement targeted physiotherapy strategies, such as core stabilization, pelvic tilt re-education, and gait retraining to mitigate abnormal load transmission through the spine–pelvis–knee axis. Such interventions could help preserve cartilage integrity and delay disease progression in patients with KOA.

Limitations

This study has several limitations. Firstly, its cross-sectional design and absence of longitudinal follow-up preclude any causal inference regarding the relationship between spinopelvic alignment and femoral cartilage morphology. Secondly, the study was conducted at a single center using operator-dependent ultrasonographic measurements. Although intra-observer reliability was excellent, inter-observer variability was not assessed, which may introduce potential measurement bias. Also, two physiatrists independently evaluated radiographs and resolved disagreements by consensus, formal inter-rater reliability (e.g., ICC) was not calculated. Thirdly, the cohort was restricted to individuals with symptomatic KOA and a BMI <35 kg/m², which may limit generalizability to broader populations. Additionally, since limb dominance could not be recorded, the asymmetric load distribution between dominant and non-dominant extremities, particularly regarding findings on the left side, could not be evaluated. Finally, dynamic gait analysis and additional biomechanical parameters such as knee adduction moment or coronal malalignment were not evaluated, which could have provided a more comprehensive understanding of load distribution across the kinetic chain.

CONCLUSIONS

This study is the first to examine the association between sagittal spinopelvic alignment and femoral cartilage thickness using USG in native knees. Lumbar lordosis emerged as an independent, laterality-specific determinant of medial cartilage morphology, suggesting that even subclinical proximal malalignment may influence distal joint health. These findings underscore the need to incorporate sagittal alignment assessment into routine KOA evaluations. From a surgical perspective, residual imbalance may jeopardize long-term outcomes by perpetuating distal overload. Integrating cartilage-focused screening into

sagittal realignment protocols could enhance patient selection, rehabilitation strategies, and overall surgical success.

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Data Availability Statement: The datasets generated and analyzed during the current study are not publicly available due to institutional regulations and the protection of participant confidentiality. However, anonymized data supporting the main findings of this study may be made available from the corresponding author upon reasonable request. Detailed descriptions of the methodology and statistical procedures are provided in the manuscript to allow reproducibility.

Ethics Committee Statement: The study protocol was reviewed and approved by the Clinical Research Ethics Committee of Istanbul Medipol University (Approval date: June 8, 2023; Approval No: 510/2023). All procedures were conducted in accordance with the ethical standards of the institutional and national research committee and with the Helsinki Declaration and its later amendments.

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