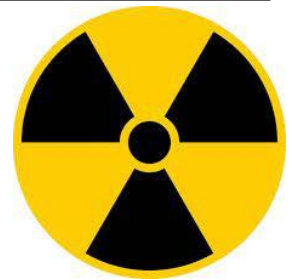


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### MAGNETIC RESONANCE IMAGING-BASED ANALYSIS OF CARTILAGE DEGENERATION AMONG PATIENTS WITH KNEE OSTEOARTHRITIS IN ABUJA METROPOLIS, NIGERIA

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#### ABSTRACT

**Background:** Knee osteoarthritis (KOA) is a degenerative disorder characterised by cartilage loss and abnormalities involving the subchondral bone and other joint structures. Magnetic resonance imaging (MRI) provides direct assessment of cartilage and soft tissues. [1,2]

**Objective:** To analyse MRI-detected cartilage degeneration among adults with KOA in Abuja Metropolis, quantify cartilage thickness and selected structural abnormalities, and examine their relationship with symptom severity.

**Methods:** This retrospective cross-sectional study reviewed knee MRI examinations and clinical records from three diagnostic imaging centres in Abuja between January 2020 and December 2025. It included 400 adults aged  $\geq 40$  years, comprising 200 participants with KOA and 200 controls. MRI examinations used 1.5T and 0.2T systems with scanner-specific protocols. Cartilage thickness was measured manually on sagittal PD-FS images at five predefined anatomical locations, with their mean representing overall thickness. Bone marrow lesions (BMLs), osteophytes, cartilage thinning and signal heterogeneity were assessed. Symptom severity was evaluated using a Proxy Symptom Severity Index (PSSI). Mann-Whitney U, Kruskal-Wallis and Spearman tests were used. Ethical approval was obtained (FHREC/2025/01/329/20-10-25).

**Results:** Among participants with KOA, BMLs occurred in 137 (68.5%), osteophytes in 121 (60.5%), cartilage thinning in 107 (53.5%), and signal heterogeneity in 101 (50.5%). Median cartilage thickness was significantly lower in KOA than controls (1.67 mm [IQR 1.45–1.89] vs 3.11 mm [IQR 2.95–3.25];  $p < 0.001$ ). Thickness did not differ significantly by sex ( $p = 0.795$ ), age group ( $p = 0.656$ ), or PSSI category ( $p = 0.509$ ). It showed a weak, non-significant inverse correlation with PSSI ( $\rho = -0.100$ ;  $p = 0.158$ ). Cartilage thickness was lower in participants with each assessed MRI abnormality (all  $p < 0.001$ ).

**Conclusion:** MRI demonstrated substantial structural abnormalities and reduced cartilage thickness among participants with KOA. The weak association between cartilage thickness and symptom severity supports considering structural MRI findings and clinical symptoms as complementary measures. Advanced compositional MRI techniques may provide additional information on early cartilage changes, although they were not performed.

**Keywords:** Knee osteoarthritis; magnetic resonance imaging; cartilage degeneration; cartilage thickness.

## Introduction

Knee osteoarthritis (KOA) is a chronic degenerative disorder involving the articular cartilage, subchondral bone, synovium, menisci and other components of the joint. Structural progression may include cartilage thinning, osteophyte formation, bone marrow lesions and other changes that contribute to pain and functional limitation. MRI-based imaging research has demonstrated that osteoarthritis is a whole-joint process rather than an isolated cartilage disorder. [1,2] Conventional radiography remains an important imaging modality in the assessment of KOA because of its availability, relatively low cost and established role in demonstrating osteophytes, joint-space narrowing and other osseous manifestations. However, radiographs do not directly visualise articular cartilage and may therefore provide limited information about early cartilage abnormalities. MRI provides multiplanar visualisation of cartilage, bone marrow, menisci, ligaments and other soft tissues and allows structural assessment of several components of the osteoarthritic joint. [2,3]

Ultrasound provides a readily accessible, radiation-free method for assessing superficial cartilage, synovium, effusion and other periarticular structures, but its field of view and operator dependence limit comprehensive assessment of deep joint structures and whole-joint cartilage. CT provides excellent depiction of osseous morphology but involves ionising radiation and has limited direct assessment of articular cartilage compared with MRI. MRI provides more comprehensive assessment of cartilage, bone marrow, menisci, ligaments and other soft tissues. MRI-PET remains primarily a research-oriented hybrid imaging approach rather than a routine modality for clinical assessment of knee osteoarthritis. [2,14]

The diagnostic performance of MRI for knee osteoarthritis varies considerably across published studies because of differences in reference standards, disease definitions, populations and imaging protocols. A recent scoping review of systematic reviews identified substantial methodological heterogeneity in the diagnostic-accuracy literature. Reported pooled estimates have included sensitivity of 61% and specificity of 82% in one meta-analysis and sensitivity of 74% and specificity of 95% in another review judged to have higher methodological quality. These figures should however not be interpreted as universal diagnostic sensitivity or specificity estimates for MRI. The present study was designed to characterise structural MRI findings in participants with

documented KOA and controls rather than to establish diagnostic sensitivity, specificity or any area under the receiver-operating-characteristic curve. [3,4,12]

Beyond conventional morphological imaging, quantitative and compositional MRI techniques have been developed to assess cartilage matrix characteristics. T2 and T1 $\rho$  mapping have been investigated as biomarkers of cartilage composition and early degenerative change, while T2\* mapping, diffusion-based methods, ultrashort echo-time imaging, dGEMRIC and other techniques provide additional approaches to evaluating cartilage composition and microstructure. These techniques may identify changes that precede some visually apparent morphological abnormalities. However, their clinical implementation is limited by issues including acquisition time, segmentation and protocol standardisation. [5-7]

T2 and T2\* relaxation behaviour is influenced by the composition and organisation of cartilage tissue. Quantitative mapping may therefore provide information beyond conventional morphological sequences. Recent literature continues to support the research potential of compositional MRI while emphasising the need for standardisation and further clinical validation. Importantly, T1 mapping, T2 mapping and T2\* mapping were not performed in the present study and are discussed only as advanced approaches in the literature. Quantitative assessment of cartilage morphology using MRI has also developed substantially. MRI-based cartilage morphometry permits objective assessment of cartilage thickness and volume and has become an established research outcome in studies of osteoarthritis progression. Manual measurement remains feasible where appropriately defined anatomical landmarks and reproducible measurement procedures are applied. [5-9]

MRI abnormalities do not necessarily correspond directly to symptom severity. Pain and functional limitation may reflect several interacting processes, including subchondral bone abnormalities, synovial inflammation, meniscal pathology and periarticular structures. Recent longitudinal work has similarly demonstrated substantial discordance between imaging findings and patient-reported symptoms. Semi-quantitative MRI assessment systems such as the MRI Osteoarthritis Knee Score (MOAKS) provide structured assessment of cartilage, bone marrow lesions, osteophytes and other abnormalities and have demonstrated good reliability in research settings. [10-

12] The present study did not use MOAKS or Kellgren–Lawrence grading. Instead, the study focused on selected MRI structural features and manually measured cartilage thickness.

Evidence describing MRI-based cartilage degeneration among Nigerian populations remains limited. Local data describing the frequency of MRI-detected structural abnormalities and their relationship with symptom burden are therefore relevant to musculoskeletal imaging practice. [15] The aim of this study was to analyse MRI-detected cartilage degeneration among adults with KOA in Abuja Metropolis, Nigeria. Specifically, the study assessed selected MRI structural abnormalities, quantified cartilage thickness, compared cartilage thickness between participants with KOA and controls, and examined the relationship between cartilage thickness and symptom severity.

## Materials and Methods

A retrospective cross-sectional study design was used. Previously acquired knee MRI examinations and corresponding clinical records of participants who underwent knee MRI between January 2020 and December 2025 were reviewed. The retrospective design enabled the assessment of existing MRI examinations and clinical information without additional imaging or direct contact with participants. The study was conducted at three diagnostic imaging centres in Abuja, Nigeria: National Hospital Abuja, LifeBridge Diagnostic Centre, Garki Area 11, and E-Clinic Diagnostics, Wuse II. The participating centres provided knee MRI services using different MRI systems and scanner field strengths.

The study population comprised adults aged 40 years or older who underwent knee MRI during the study period. A total of 400 participants were included, comprising 200 participants with KOA and 200 controls. Participants in the KOA group were identified from archived clinical and imaging records in which KOA had been documented as the clinical indication or diagnosis for the knee MRI examination. The MRI indication and relevant clinical diagnosis were obtained from the archived radiology request forms and associated clinical records. Controls were selected from individuals who underwent knee MRI for non-degenerative indications, including minor trauma or ligament-related assessment, and who had no documented diagnosis of KOA in the available clinical records. During record review, examinations showing

established degenerative abnormalities considered inconsistent with the control definition were not classified as controls. Incidental findings were therefore reviewed against the clinical records and MRI appearance before final group assignment. Cases with previous knee surgery, major knee trauma, inflammatory arthritis, systemic joint disease, poor-quality MRI examinations or age below 40 years were excluded. The minimum sample size was estimated using Cochran's formula, assuming a 50% prevalence of the characteristic of interest. The calculated minimum sample was approximately 385 participants. The final sample consisted of 400 participants.

MRI examinations were performed using scanner-specific knee musculoskeletal protocols. The acquisition parameters varied according to the MRI system and institutional protocol and are summarised in Table 1. At National Hospital Abuja, examinations were performed using a Philips Achieva 1.5T MRI system and a Siemens Magnetom 0.2T open MRI system. LifeBridge Diagnostic Centre used a Toshiba Estalan Vantage 1.5T MRI system, while E-Clinic Diagnostics used a Siemens Magnetom Essenza 1.5T MRI system. The principal morphological sequences used for the present analysis included PD-FS and T2-FS. STIR sequences were also used where available according to the institutional protocol. An example of the sagittal fat-suppressed T2-weighted fast spin-echo sequence used for assessment of the knee joint is shown in Figure 1.

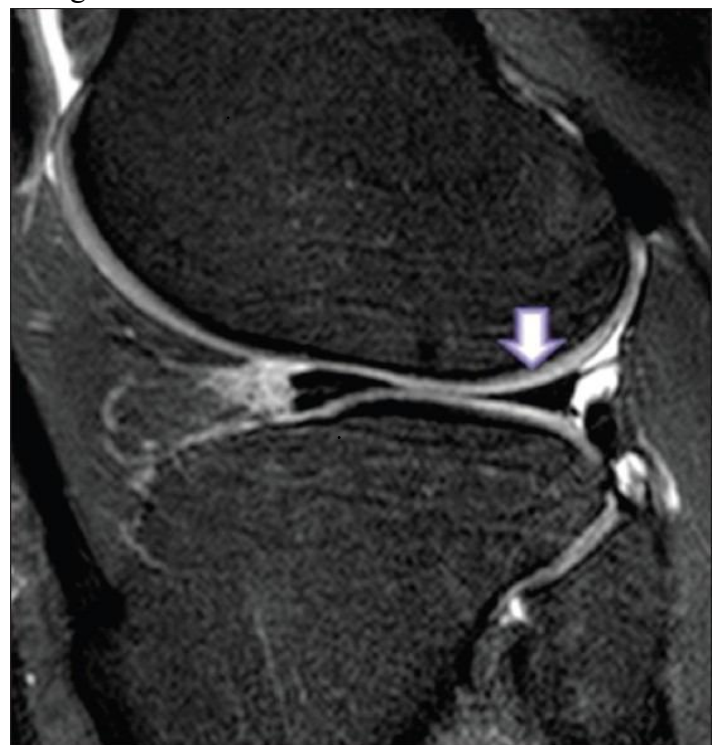


Figure 1. Sagittal FS T2W FSE image of the knee joint.

TR and TE influence tissue contrast, while slice thickness affects spatial resolution and partial-volume effects. Field of view (FOV) determines anatomical coverage and contributes to in-plane resolution, whereas matrix size influences pixel resolution. PD-FS images were used primarily for cartilage-thickness measurement and assessment of cartilage morphology. T2-FS images were used to evaluate fluid-sensitive structural abnormalities, including bone marrow lesions, cartilage abnormalities and other degenerative changes. Where available, STIR images provided additional fluid-sensitive assessment of bone marrow and soft-tissue abnormalities. Quantitative T1, T2 and T2\* mapping sequences were not acquired.

MRI examinations were assessed for selected degenerative features, including bone marrow lesions, osteophytes, cartilage thinning and cartilage signal heterogeneity. These predefined structural features were subsequently analysed and reported in the Results. The presence or absence of each feature was recorded for participants with KOA, and findings were not considered mutually exclusive because more than one structural abnormality could occur in the same knee. Articular cartilage thickness was measured manually on sagittal proton-density fat-suppressed (PD-FS) knee MRI images using the electronic calliper function of the DICOM image viewer. Measurements were performed at five predefined anatomical locations: the medial femoral condyle, lateral femoral condyle, medial tibial plateau, lateral tibial plateau and central patellar cartilage.

At each location, cartilage thickness was measured in millimetres as the perpendicular distance between the articular cartilage surface and the cartilage-subchondral bone interface. Measurements were obtained at the predefined anatomical landmarks using the same measurement orientation and procedure for all examinations. Where multiple consecutive sagittal slices demonstrated the relevant anatomical region, the slice showing the central portion of the cartilage was selected. The mean of the five regional measurements was calculated to represent the overall cartilage thickness for each assessed knee. To assess intra-rater reliability, 30 knee MRI examinations were randomly selected from the study sample. The same observer repeated the cartilage-thickness measurements after a 7-day interval while blinded to the results of the initial measurements. Reliability was assessed using a two-way mixed-effects, single-measure, absolute-agreement intraclass correlation coefficient (ICC).

Measurement of cartilage thickness demonstrated high intra-rater reliability, with an ICC of 0.93 (95% CI: 0.87–0.96).<sup>[13]</sup>

Symptom severity was assessed retrospectively using the Proxy Symptom Severity Index (PSSI), developed for the study because a standardised patient-reported outcome instrument such as WOMAC was not consistently available in the archived records. Pain, joint stiffness and functional limitation were retrospectively derived from symptoms documented in the archived clinical records. Each domain was assigned a score from 0 to 3 according to the documented severity, with 0 indicating absence of the symptom and higher scores indicating progressively greater symptom burden. Two reviewers independently assessed the available clinical records and assigned scores for the three symptom domains using the predefined scoring criteria. Where differences occurred between reviewers, the records were jointly reviewed and a consensus score was agreed. Records with insufficient information to assign a domain score were excluded from PSSI analysis. The three domain scores were summed to produce a total PSSI score ranging from 0 to 9. PSSI scores of 0-2, 3-5 and 6-9 were classified as mild, moderate and severe symptom burden, respectively. Because the PSSI was developed for this retrospective study and was not a validated patient-reported outcome instrument, its results were interpreted as symptom-severity measures rather than as a validated measure of structural KOA severity.

### Data collection

Data were abstracted retrospectively from MRI archives and available clinical records using a structured data-abstraction form. Variables included demographic characteristics, MRI findings, MRI acquisition information, cartilage-thickness measurements and PSSI scores. Data were analysed using IBM SPSS Statistics version 26. Continuous variables were summarised using means and standard deviations where appropriate and medians with interquartile ranges for non-normally distributed variables. Categorical variables were summarised using frequencies and percentages. Distributional assessment was performed before selection of inferential statistical tests. Because cartilage-thickness and PSSI data were non-normally distributed, non-parametric methods were used. The Mann-Whitney U test was used for comparisons between two independent groups, while the Kruskal-Wallis test was used for comparisons involving more than two groups.

Spearman's rank correlation coefficient was used to assess the relationship between cartilage thickness and PSSI score. For comparisons of cartilage thickness according to individual MRI features, effect sizes were reported using the rank-biserial correlation coefficient where applicable. Statistical significance was set at  $p < 0.05$ .

## Results

A total of 400 participants were included, comprising 200 participants with KOA and 200 controls. Females constituted 52.0% (208/400) of the study population and males 48.0% (192/400). The median age was 64 years (IQR 52 - 74), with an observed range of 40 - 85 years. Among the 200 participants with KOA, BMLs were the most frequently identified structural abnormality, occurring in 137 (68.5%). Osteophytes were identified in 121 (60.5%), cartilage thinning in 107 (53.5%), and cartilage signal heterogeneity in 101 (50.5%).

### Cartilage thickness

Median cartilage thickness was significantly lower among participants with KOA than controls, measuring 1.67 mm (IQR: 1.45 - 1.89) and 3.11 mm (IQR: 2.95 - 3.25), respectively ( $U = 40,000.0$ ;  $p < 0.001$ ). Cartilage thickness did not differ significantly between males and females, with median values of 2.38 mm (IQR: 1.75–3.00) and 2.39 mm (IQR: 1.80–3.02), respectively ( $U = 20,267.5$ ;  $p = 0.795$ ). Similarly, no statistically significant difference was observed across age groups, with median cartilage thicknesses of 2.45

mm (IQR: 1.85 - 3.10), 2.38 mm (IQR: 1.82 - 2.95), and 2.36 mm (IQR: 1.80 - 2.90) among participants aged 40 - 50, 51 - 70 and >70 years, respectively ( $H = 0.843$ ;  $p = 0.656$ ).

### Relationship between MRI abnormalities and cartilage thickness

Participants with each of the assessed MRI abnormalities had lower median cartilage-thickness measurements than those without the corresponding abnormality. The largest observed difference was between participants with and without BMLs, with median cartilage thickness of 1.60 mm and 2.92 mm, respectively.

### Relationship between cartilage thickness and symptom severity

Cartilage thickness demonstrated a weak inverse correlation with PSSI score (Spearman's  $\rho = -0.100$ ;  $p = 0.158$ ). This association was not statistically significant. Kruskal-Wallis testing showed no statistically significant difference in cartilage thickness across the three PSSI symptom-severity categories ( $H = 1.351$ ,  $p = 0.509$ ). These categories represent PSSI-defined symptom severity and not structural KOA severity.

### Knee involvement

Among the 200 participants with KOA, bilateral involvement was documented in 79 (39.5%), right-knee-only involvement in 66 (33.0%), and left-knee-only involvement in 55 (27.5%).

**Table 1: MRI acquisition parameters**

| Facility                     | MRI scanner              | Field strength | MRI sequences used                  | TR/TE (ms)              | Slice thickness (mm) | FOV (mm) | Matrix                 |
|------------------------------|--------------------------|----------------|-------------------------------------|-------------------------|----------------------|----------|------------------------|
| National Hospital Abuja      | Philips Achieva          | 1.5 T          | Sagittal/coronal/axial PD-FS; T2-FS | 2800–4000 / 30–40       | 3                    | 160      | 256 × 256              |
| National Hospital Abuja      | Siemens Magnetom Open    | 0.2 T          | PD-weighted; T2-weighted/T2-FS      | 2000–4000 / 30–90       | 3–5                  | 160–200  | 256 × 256              |
| LifeBridge Diagnostic Centre | Toshiba Estalan Vantage  | 1.5 T          | Sagittal/coronal FS; axial T2-FS    | PD-2300 / 18; 3500 / 60 | 3                    | 160–170  | 205 × 256 to 200 × 368 |
| E-Clinic Diagnostics         | Siemens Magnetom Essenza | 1.5 T          | PD-FS; T2-FS/STIR                   | 2500–4000 / 30–50       | 3–3.5                | 160–180  | 256 × 256 to 278 × 384 |

**Table 2. Sociodemographic characteristics of participants**

| Variable    | Category                     | n            | %    |
|-------------|------------------------------|--------------|------|
| Study group | KOA                          | 200          | 50.0 |
|             | Control                      | 200          | 50.0 |
| Facility    | LifeBridge Diagnostic Centre | 200          | 50.0 |
|             | National Hospital Abuja      | 100          | 25.0 |
|             | E-Clinic Diagnostics         | 100          | 25.0 |
| Sex         | Male                         | 192          | 48.0 |
|             | Female                       | 208          | 52.0 |
| Age (years) | 40 - 50                      | 94           | 23.5 |
|             | 51 - 70                      | 166          | 41.5 |
|             | > 70                         | 140          | 35.0 |
|             | Median (IQR)                 | 64 (52 - 74) | -    |
|             | Range                        | 40 - 85      | -    |

**Table 3. MRI-detected degenerative features among participants with KOA**

| MRI feature                    | n   | %    |
|--------------------------------|-----|------|
| Bone marrow lesions            | 137 | 68.5 |
| Osteophytes                    | 121 | 60.5 |
| Cartilage thinning             | 107 | 53.5 |
| Cartilage signal heterogeneity | 101 | 50.5 |

Note: Percentages are calculated within the KOA group (n = 200). Features were not mutually exclusive.

**Table 4. Cartilage thickness according to study group, sex and age**

| Variable          | Category | Median cartilage thickness, mm (IQR) | Test statistic | p      |
|-------------------|----------|--------------------------------------|----------------|--------|
| Group             | KOA      | 1.67 (1.45–1.89)                     | U = 40,000.0   | <0.001 |
|                   | Control  | 3.11 (2.95–3.25)                     |                |        |
| Sex               | Male     | 2.38 (1.75–3.00)                     | U = 20,267.5   | 0.795  |
|                   | Female   | 2.39 (1.80–3.02)                     |                |        |
| Age group (years) | 40–50    | 2.45 (1.85–3.10)                     | H = 0.843      | 0.656  |
|                   | 51–70    | 2.38 (1.82–2.95)                     |                |        |
|                   | >70      | 2.36 (1.80–2.90)                     |                |        |

**Table 5. Association between MRI-detected abnormalities and cartilage thickness**

| MRI feature                | Median thickness, mm (IQR) | n   | U      | Effect size (r) | p      |
|----------------------------|----------------------------|-----|--------|-----------------|--------|
| Signal heterogeneity - Yes | 1.72 (1.50 - 1.90)         | 101 | 15,230 | 0.56            | <0.001 |
| Signal heterogeneity - No  | 2.85 (2.60 - 3.20)         | 99  |        |                 |        |
| Cartilage thinning - Yes   | 1.65 (1.45 - 1.85)         | 107 | 14,875 | 0.58            | <0.001 |
| Cartilage thinning - No    | 2.90 (2.65 - 3.25)         | 93  |        |                 |        |
| Osteophytes - Yes          | 1.80 (1.55 - 2.05)         | 121 | 16,110 | 0.52            | <0.001 |
| Osteophytes - No           | 2.95 (2.70 - 3.28)         | 79  |        |                 |        |
| BMLs - Yes                 | 1.60 (1.40 - 1.85)         | 137 | 13,950 | 0.60            | <0.001 |
| BMLs - No                  | 2.92 (2.68 - 3.20)         | 63  |        |                 |        |

**Table 6. Cartilage thickness and distribution according to PSSI symptom-severity category**

| PSSI category | n (%)     | Median cartilage thickness, mm (IQR) |
|---------------|-----------|--------------------------------------|
| Mild          | 23 (11.5) | 1.70 (0.50)                          |
| Moderate      | 54 (27.0) | 1.70 (0.50)                          |

|               |             |             |
|---------------|-------------|-------------|
| <b>Severe</b> | 123 (61.5)  | 1.70 (0.50) |
| <b>Total</b>  | 200 (100.0) | 1.70 (0.50) |

Table 7. Distribution of knee involvement among participants with KOA

| <b>Knee involvement</b> | <b>n</b> | <b>% within KOA</b> |
|-------------------------|----------|---------------------|
| <b>Bilateral</b>        | 79       | 39.5                |
| <b>Right knee only</b>  | 66       | 33.0                |
| <b>Left knee only</b>   | 55       | 27.5                |
| <b>Total</b>            | 200      | 100.0               |

## Discussion

This study evaluated MRI-detected cartilage degeneration among 200 participants with KOA and 200 controls from three diagnostic imaging centres in Abuja. The principal findings were that BMLs, osteophytes, cartilage thinning and cartilage signal heterogeneity were frequent among participants with KOA, while median cartilage thickness was substantially lower than that observed among controls. BMLs were the most frequently observed MRI abnormality, occurring in 68.5% of participants with KOA. BMLs are recognised MRI manifestations of osteoarthritis and reflect abnormalities in the subchondral bone environment. Contemporary MRI research considers the subchondral bone, cartilage and other joint tissues as interacting components of the osteoarthritic process rather than isolated structures. [1,2,11,12]

The median cartilage thickness among participants with KOA was 1.67 mm compared with 3.11 mm among controls, representing a statistically significant difference. This finding is consistent with the established role of MRI-based cartilage morphometry in quantifying structural cartilage loss. Long-term MRI research has demonstrated the value of cartilage morphometry for evaluating osteoarthritis progression and treatment-related structural change. [8,9] The presence of BMLs, osteophytes, cartilage thinning and signal heterogeneity was associated with lower cartilage-thickness measurements. The strongest observed effect size was associated with BMLs. These findings support the concept that cartilage morphology and subchondral bone abnormalities are related components of structural osteoarthritis.

However, the cross-sectional nature of the study does not establish temporal or causal relationships. In particular, the finding that BMLs were associated with lower cartilage thickness should not be interpreted as evidence that BMLs caused cartilage loss. The relationship between structural MRI abnormalities and symptoms was weak. Cartilage thickness had a correlation coefficient of -0.100 with PSSI and the

association was not statistically significant ( $p = 0.158$ ). Similarly, cartilage thickness did not differ significantly across the PSSI-defined mild, moderate and severe symptom categories. This finding is consistent with the broader literature showing that imaging abnormalities and symptoms in knee osteoarthritis do not necessarily correspond closely. A recent large longitudinal analysis also found substantial discordance between imaging-based structural measures and pain or symptom measures. [10]

The absence of a significant relationship between cartilage thickness and PSSI should not be interpreted to mean that structural abnormalities are clinically irrelevant. Pain and functional limitation in KOA may reflect multiple processes, including synovial inflammation, subchondral bone abnormalities, meniscal pathology and periarticular tissue involvement. Therefore, a single morphological measure such as mean cartilage thickness cannot capture the full clinical burden of KOA. The study also demonstrates the practical feasibility of manual cartilage-thickness measurement using conventional PD-FS MRI images and predefined anatomical landmarks. Repeat measurement of 30 examinations after a 7-day interval was incorporated to evaluate intra-rater reproducibility. Reporting the ICC model is important because different ICC formulations make different assumptions and can produce different estimates. [13]

The present study did not use quantitative T2, T2\* or T1 $\rho$  mapping. These techniques have potential advantages because they can provide information about cartilage composition and matrix characteristics beyond conventional morphological imaging. T2 and T1 $\rho$  measurements have been investigated as markers of early cartilage degeneration, while T2\* mapping and other quantitative techniques may provide additional information concerning cartilage microstructure. Nevertheless, current literature identifies important barriers to routine clinical implementation, including acquisition and analysis standardisation, segmentation,

scan time and the lack of universally accepted reference values. [5-7]

The diagnostic performance of MRI also requires careful interpretation. Published studies and systematic reviews have reported variable sensitivity and specificity depending on the reference standard and study methodology. [3,4,12] The present study therefore does not claim that MRI has a particular sensitivity or specificity for KOA. Instead, it demonstrates differences in selected MRI structural findings between a group with documented KOA and controls. Because the study was not designed as a diagnostic-accuracy study and did not establish an independent reference standard against which MRI was tested, sensitivity, specificity, predictive values and ROC-AUC were not calculated.

Several methodological considerations are also recognised. First, the retrospective design depended on the completeness and accuracy of archived records. Second, the study involved three centres with different MRI systems and field strengths, which may introduce variation in image characteristics. The scanner-specific acquisition parameters were explicitly documented. Third, PSSI was developed as a retrospective symptom-severity measure because a standardised patient-reported outcome instrument was not consistently available. Although the PSSI provided a structured method of classifying recorded symptoms, it should however not be regarded as a validated replacement for instruments such as WOMAC. Its mild, moderate and severe categories represent symptom burden rather than structural KOA severity. Finally, the study used selected MRI features rather than a comprehensive validated whole-joint scoring system such as MOAKS. This limits direct comparison with studies using formal semi-quantitative scoring systems. [11,12] Future prospective studies could consider validated patient-reported outcome instruments, standardised MRI scoring systems and quantitative compositional MRI techniques.

## Conclusion

MRI demonstrated substantial structural abnormalities among participants with KOA, including bone marrow lesions, osteophytes, cartilage thinning and cartilage signal heterogeneity. Participants with KOA had significantly lower median cartilage thickness than controls. The observed relationship between cartilage thickness and symptom severity was weak and not statistically significant. These findings support the interpretation of MRI structural measures and clinical

symptoms as related but distinct dimensions of KOA. The study provides multi-centre MRI data on cartilage-related abnormalities among adults with KOA in Abuja, Nigeria. Further prospective research using validated symptom instruments, standardised MRI scoring systems and quantitative compositional MRI techniques may provide additional information on the relationship between cartilage structure, composition and clinical symptoms.

## Strengths and Limitations of the Study

The study included participants from three diagnostic imaging centres and incorporated MRI systems with different field strengths. Detailed acquisition parameters were documented, and cartilage thickness was measured using predefined anatomical landmarks and a standardised manual measurement procedure. Intra-rater reliability was assessed using repeat measurements after a 7-day interval. However, the retrospective design limited the availability and standardisation of clinical information. The PSSI was developed for retrospective symptom assessment and has not been presented as a validated substitute for established patient-reported outcome measures. The study did not perform T1, T2 or T2\* mapping and therefore cannot make direct claims concerning cartilage compositional biomarkers. Differences in scanner field strength and acquisition protocols may also influence image characteristics. Finally, the study was not designed as a diagnostic-accuracy study and therefore did not establish MRI sensitivity, specificity or ROC-AUC. The study involved three centres and multiple MRI systems with different field strengths and acquisition parameters, including two different MRI systems at National Hospital Abuja. Such variation may have influenced image quality, tissue contrast and measurement characteristics. Although scanner-specific acquisition parameters were documented, the retrospective design did not permit standardisation of imaging protocols across centres.

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## Competing Interests

The authors declare that they have no competing interests.

## Data Availability

The data supporting the findings of this study are available from the corresponding author upon reasonable request. The data are not publicly available because they comprise hospital records subject to institutional and ethical restrictions.

### Use of Generative Artificial Intelligence

Grammarly, Chatgpt, Quillbot and Phrasly were used to assist with language editing, including grammar, clarity and formatting. The authors reviewed the manuscript and accept responsibility for the accuracy and integrity of the scientific content.

### Ethical Approval

Ethical approval was obtained from the Health Research Ethics Committee of the Federal Capital Territory, Nigeria (FHREC/2025/01/329/20-10-25). A waiver of informed consent was granted because the study involved retrospective analysis of anonymised records without direct participant contact or additional imaging. Confidentiality and data security were maintained throughout the study.

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