Section: Orthopaedics

Original Research Article

CORRELATION BETWEEN CLINICAL FEATURES, X-RAY FINDINGS AND WEAR PATTERN OF CARTILAGE USING T2 MAP MRI IN KNEE OSTEOARTHRITIS

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Abstract

Background: The purpose of this study was to analyze the pattern of cartilage degeneration in symptomatic Osteoarthritis (OA) knees and its correlation with X-Ray and T2 Map MRI. **Materials and Methods:** The present study included 40 knees over a period of 12 months. We used the Western Ontario and McMaster University (WOMAC) osteoarthrosis index for clinical assessment, then plain radiographs of AP view of involved knee joint were done and graded according to Kellgren-Lawrence Classification. T2 Mapping MRI of the same knee joint was done. The "ICRS-knee cartilage mapping system" was used to establish precise location of the lesion. **Result:** Significant correlation ($p<0.05$) was found between WOMAC Score and T2 Map MRI findings in parts of trochlea of femur, parts of condyle of femur and tibial cartilage. Significant correlation ($p<0.05$) was found between WOMAC Score and K-L Grade. Correlation between T2 Map MRI and K-L Grade was found to be highly significant ($p<0.001$) in parts of trochlea of femur, medial posterior condyle of femur and all the compartments of cartilage of tibia but not significant ($p>0.05$) in lateral condyle of femur. **Conclusion:** T2 Map MRI is a noninvasive tool for cartilage evaluation, can be used to assess treatment related changes in cartilage over time and early-OA disease state when no morphological changes have occurred but biochemical changes have started in the cartilage.

INTRODUCTION

Osteoarthritis (OA) refers to a clinical syndrome of joint pain accompanied by varying degrees of functional limitation and reduced quality of life. Osteoarthritis is characterized pathologically by localized loss of cartilage, remodeling of adjacent bone and associated inflammation.[1] It is more common in women than men and the prevalence increase dramatically with age (>60 years).[2]

Articular cartilage is hypocellular, with only 4% of its wet weight consisting of chondrocytes. The main components of articular cartilage are water (65-85% of weight) and the Extra Cellular Matrix (ECM) composed of collagen (15-20% of weight) and proteoglycans (PGs) (3-10% of weight).[3,4] The most abundant collagen in articular cartilage is type II collagen, forming microfibrils, fibrils and later collagen fibers intertwined with proteoglycan aggregates.[5] The main proteoglycan type in articular cartilage is aggrecan, while the key glycosaminoglycans (GAGs) are hyaluronic acid, chondroitin sulfate, keratan sulfate and dermatan sulfate. The GAGs with a high density of sulfate anions in the aggrecan can attract cations in water and offer the articular cartilage with osmotic properties.[6] The collagen fiber network and the attached proteoglycan aggregates collaboratively give rise to the compression resistance of the cartilage.[7]

Osteoarthritis is characterized by following changes in the cartilage biochemistry and microstructure: earliest changes include reduced PGs concentration, possible changes in the size of collagen fibril and aggregation of PGs, increased water content and increased synthesis and degradation of matrix macromolecules.[8] These lead to breakdown and decreased content of the PGs matrix, which in turn lead to ulceration with inflow of PGs into the synovial fluid with decreased water content of the cartilage, making it less resistive to stress. As osteoarthritis progresses, collagen, PGs, and water content are reduced further and the collagen network becomes severely disrupted.[9]

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Plain radiographs have been used primarily in the evaluation of OA, which depict only narrowing of the joint space or gross osseous changes that tend to occur late in the disease. Early changes in the articular cartilage may not be visible on plain radiographs. Cartilage loss can only be indirectly inferred by the development of joint space narrowing (JSN), which can be highly unreliable even with careful attention to proper technique.[10] In addition, plain radiographs are insensitive to reveal focal cartilage loss, and shows widening of the joint space despite significant cartilage loss in one compartment of the knee simply as a result of narrowing in the other compartment.[11]

Standard cartilage dedicated MR techniques, are also inconclusive in quantifying early degenerative changes of the cartilage matrix, especially biochemical changes in cartilage.[12]

MRI based T2 Mapping method allows for the indirect assessment of collagen content and orientation, which are important indicators for early OA.[13] The collagen matrix of healthy cartilage traps and immobilizes water protons, so signal intensity on T2-weighted images is low. In the earliest stages of OA, the matrix begins to break down and becomes more permeable to water, causing an elevation in T2 relaxation times.[14]

MRI has emerged as a useful tool for clinicians and scientists to assess the health of cartilage and other soft tissues. Conventional MRI provides sufficient tissue contrast to detect morphological changes in cartilage where radiography cannot.[15] However, changes in cartilage physiology prior to morphological changes cannot be visualized or measured with conventional MRI.[16]

The purpose of the study was therefore to analyze knees, clinically and radiographically in varying stages of OA and detect subtle and early changes in cartilage and treat the disease at its earliest and know the cartilage response to treatment over time.

MATERIALS AND METHODS

The present study was an observational type of study.As soon as the patients reported in the OPD, a complete survey was carried out to record other significant complaints.For subjects to be designated as “knee pain” positive, a positive response was required to both parts of the question:

(a) “Have you ever had pain in or around the knee on most days for at least a month?
(b) If so, have you experienced any pain during the last year?”

A negative response to both parts of the above question was designated as “knee pain” negative. The present study included 33 cases (40 knees) both males and females, after obtaining informed consent, clinical and radiological examination was done during a period of 12 months from April 2016 to March 2017.

Exclusion criteria were cases with history of surgery or intraarticular steroid injection and cases with history of trauma, neuropathic joints or infection.

Methodology: After the case selection all subjects completed the Western Ontario and McMaster (WOMAC) questionnaire for the affected knee on the day knee radiographs and MR images were acquired.

X-Ray Plain radiographs of AP view in standing position (weight bearing) of involved knee joint was done. After obtaining the X-Rays, it was graded according to Kellgren-Lawrence Classification from 0-4 Grades. [Figure 1 and 2][1]

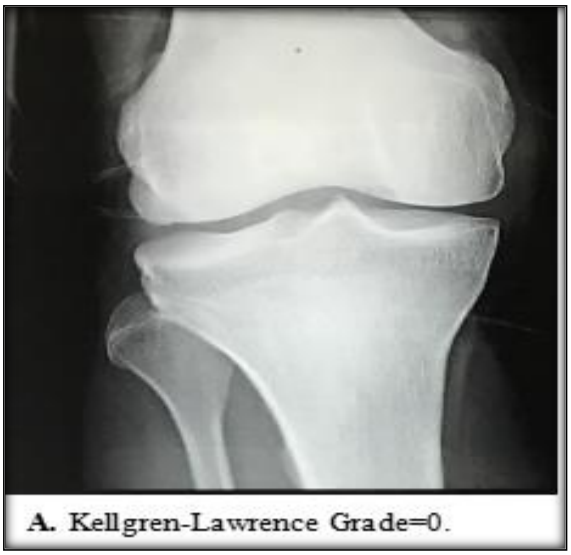

Figure 1

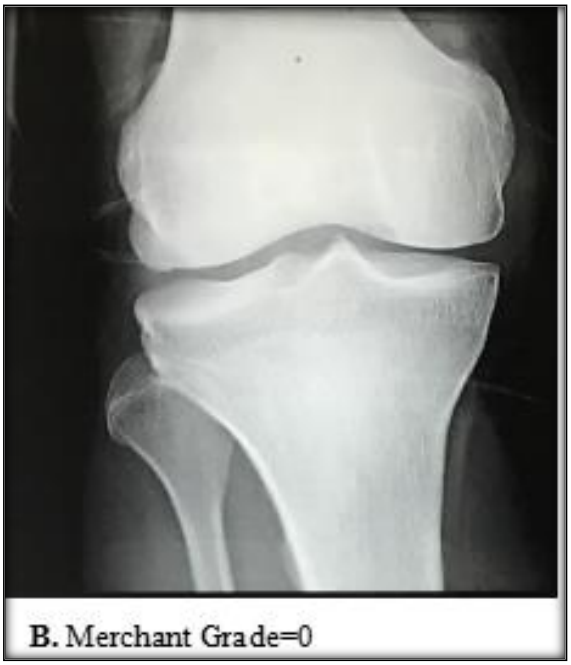

Figure 2

MRI was performed with a SIEMENS AMIRA SYSTEM with customized 16 element knee coil for knee joint. T2 Relaxation time of the articular cartilage of the knee joint, with a colour scale

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ranging between 0ms and 100ms, were created from the T2 Map source data. The colour change from blue (0ms) to red (100ms) with increasing T2 time with interval of 25ms each was generated. Then mean time of two values was calculated to produce the T2 relaxation time of the particular cartilage segment. [Figure 3-6]

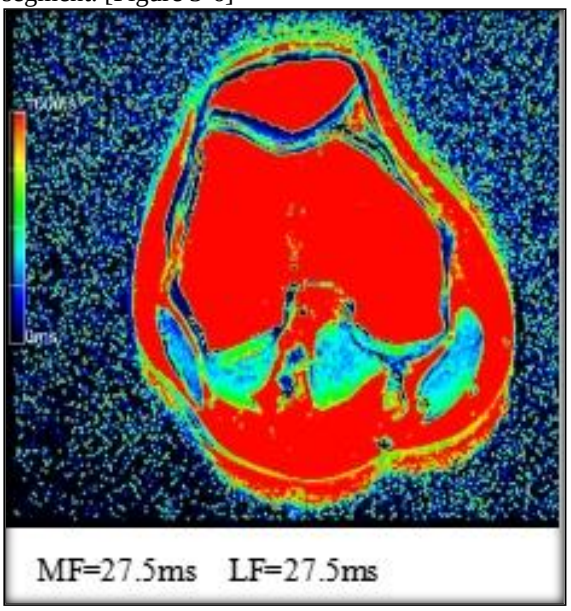

Figure 3

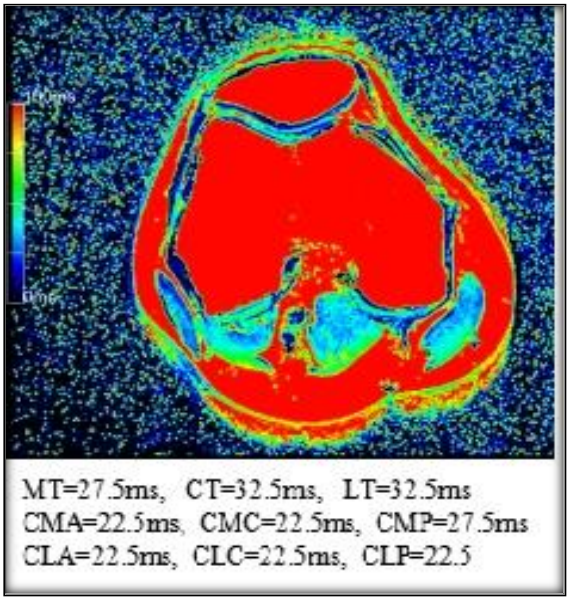

Figure 4

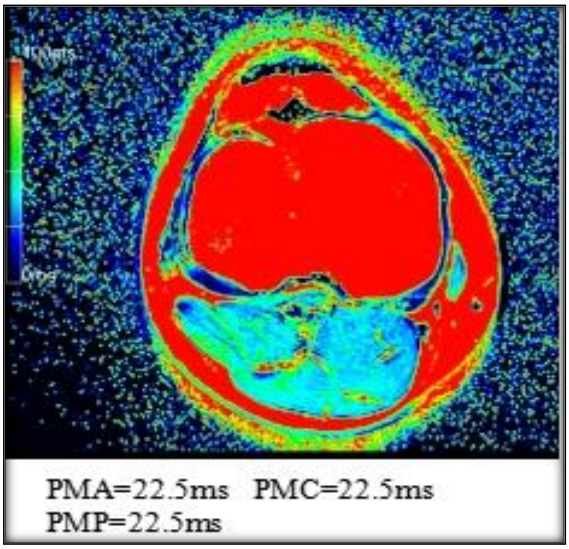

Figure 5

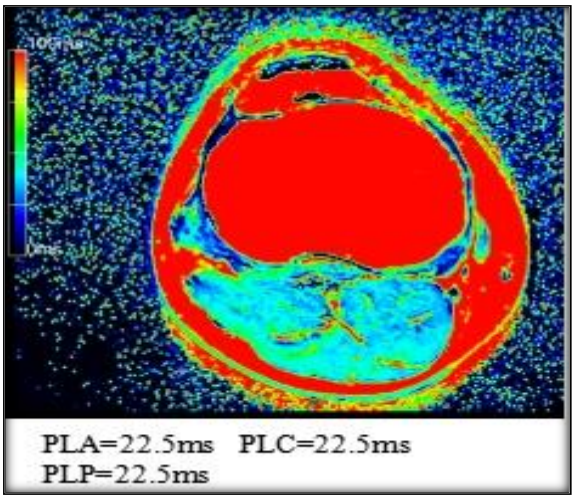

Figure 6

The articular surfaces of the cartilage were divided into six regions: patella, trochlea, medial, lateral femoral condyles, medial and lateral tibial surface. The "ICRS-knee cartilage mapping system" was used to establish precise location of the lesion. (Figure-1)

The cartilage of femur was divided into Medial Trochlea (MT), Central Trochlea (CT) and Lateral Trochlea (LT), and condyles as Condyle Medial Anterior (CMA), Condyle Medial Central (CMC), and Condyle Medial Posterior (CMP) and for lateral condyle as Condyle Lateral Anterior (CLA), Condyle Lateral Central (CLC), and Condyle Lateral Posterior (CLP).

Tibia was divided as Plateau Medial Anterior (PMA), Plateau Medial Central (PMC), Plateau Medial Posterior (PMP) and Plateau Lateral Anterior (PLA), Plateau Lateral Central (PLC), Plateau Lateral Posterior (PLP).

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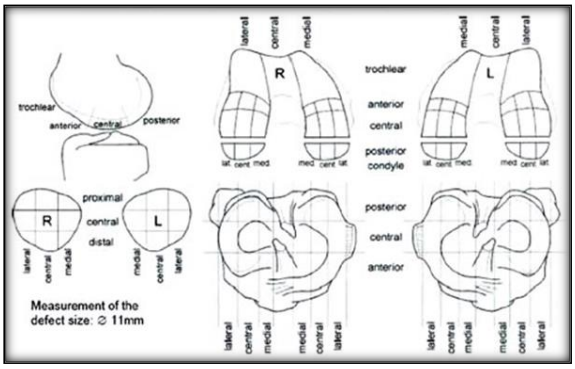

Figure 7: ICRS Knee Cartilage Lesion Mapping System. (Taken from ICRS Cartilage Injury Evaluation Package

Statistical Analysis

A one-way analysis of variance (ANOVA) was used to compare T2 values and their zonal variation with K-L Grade and Merchant Grade. The correlation between T2 values of MRI and X-Ray changes was investigated using a Spearman's rank correlation analysis. Correlation between WOMAC and K-L Grade and Merchant Grade was done using Spearman's rank correlation analysis. Correlation between WOMAC and T2 Map MRI was done according to Pearson correlation analysis. Statistical analysis was performed using SPSS version 17.0 for Windows and a level of significance of 0.05.

RESULTS

We got maximum cases in the age group 61-70 yrs in the present study of 40 knees, according to K-L Grade, 3 (7.5%) knees had Grade 0, 8 (20%) had Grade 1, 11 (27.5%) Grade 2, 8 Grade 3 (20%) and 10 (25%) knees Grade 4. [Table 1] In the study, it was found that as Grades of OA increases on X-Ray, mean T2 relaxation time of cartilage on T2 Map MRI also increases. The association was statistically highly significant in MT, CT, LT, CMP in femur ($p<0.001$), and PMA, PMC, PMP, PLA, PLC, PLP in tibia ($p<0.001$), significant in CMA, CMC of female ($p<0.05$) and no statistical significance was found with lateral condyle of femur CLA, CLC and CLP ($p>0.05$).

In present study, highly significant correlation was found in OA knee between X-Ray and associated cartilage degeneration in MT, CT, LT, CMP, PMA, PMC, PMP, PLA, PLC, PLP ($p<0.001$) on T2 Map MRI and statistical significant correlation of CMC, CLC, CLP ($p<0.05$) with T2 Map MRI. No statistical significance was found between X-Ray and T2 Map MRI in CMA, CLA ($p>0.05$). [Table 2]

In our study, there was only PMP ($p<0.001$) which had highly significant correlation with pain subscore and MT, CT, LT, CMP, CLC, PMA, PMC, PLA, PLC, PLP, MF was only significantly related with pain ($p<0.05$). No association was found between compartment CMA, CMC, CLA, CLP, LF of articular cartilage with pain subscore ($p>0.05$). With stiffness subscore, only significant association was found between LT, CMP, CLC, PMA, PMC, PMP, PLA, PLC ($p<0.05$) and no significant association with rest of the compartments.

Physical function subscore was significantly associated with MT, LT, CMP, CLC, PMA, PMP, MF, LF ($p<0.05$) and was not significantly related with any other compartment. In our study, correlation between pain ($r=0.657$) and stiffness ($r=0.576$) sub scores of WOMAC were statistically highly significant ($p<0.001$) with K-L Grade and only significantly correlated with physical function ($r=0.384$; $p<0.05$). [Table 3]

Table 1: Kellgren Lawrence Grade Distribution

K-L grade	Number	Percentage
Grade 0	3	7.5
Grade 1	8	20.0
Grade 2	11	27.5
Grade 3	8	20.0
Grade 4	10	25.0
TOTAL	40	100.0

Table 2: Association Between T2 Map MRI and Kellgren Lawrence Grade

MRI	K-L Grade 0 n=3	K-L Grade 1 n=8	K-L Grade 2 n=11	K-L Grade 3 n=8	K-L Grade 4 n=10	P value
	Mean±SD	Mean±SD	Mean±SD	Mean±SD	Mean±SD	
MT	27.50±0.00	35.00±7.07	39.31±8.44	41.87±6.78	51.00±10.55	<0.001**
CT	30.83±2.88	32.50±5.34	41.59±7.68	45.62±9.23	53.00±9.55	<0.001**
LT	32.50±0.00	31.87±6.78	38.40±7.00	41.87±9.42	52.00±11.16	<0.001**
CMA	27.50±5.00	43.12±9.03	45.22±10.57	46.87±10.50	47.00±7.97	0.040*
CMC	30.83±10.40	37.50±8.45	37.50±5.47	42.50±8.45	46.00±9.73	0.037*
CMP	27.50±0.00	33.12±5.62	39.31±4.04	42.50±7.07	51.50±6.14	<0.001**
CLA	35.83±12.58	43.75±11.87	42.04±6.50	41.25±9.16	46.50±3.94	0.361
CLC	30.83±7.63	39.37±6.51	41.59±6.64	39.37±10.32	45.00±4.85	0.062
CLP	34.16±12.58	41.87±6.23	45.22±6.06	44.37±9.97	48.00±8.31	0.128
PMA	22.50±0.00	40.00±6.54	47.50±5.00	41.87±8.21	61.50±10.48	<0.001**
PMC	22.50±0.00	35.62±5.93	43.86±5.51	46.25±7.90	58.00±9.22	<0.001**
PMP	22.500±0.00	33.75±2.31	41.59±4.90	43.12±6.78	55.00±9.50	<0.001**
PLA	25.83±5.77	35.62±5.93	42.50±6.32	47.50±3.77	50.50±7.14	<0.001**

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PLC	24.16±2.88	33.12±6.78	37.04±5.68	46.87±5.62	51.00±9.73	<0.001**
PLP	24.16±2.88	33.75±6.40	35.68±5.60	43.12±9.42	47.00±11.16	<0.001**

*p<0.05; Significant; **p<0.001; Highly significant

Table 3: Correlation between womac score and T2 MAP MRI

MRI	PAIN		STIFFNESS		PHYSICAL FUNCTION		TOTAL	
	r value	p value	r value	p value	r value	p value	r value	p value
MT	0.364	0.021*	0.251	0.119	0.347	0.028*	0.404	0.010*
CT	0.347	0.028*	0.292	0.067	0.259	0.107	0.342	0.031*
LT	0.372	0.018*	0.366	0.020*	0.321	0.043*	0.388	0.013*
CMA	0.029	0.859	0.062	0.703	0.049	0.765	0.058	0.724
CMC	0.141	0.387	0.189	0.243	0.273	0.088	0.262	0.103
CMP	0.493	0.001*	0.479	0.002*	0.367	0.020*	0.452	0.003*
CLA	0.265	0.098	0.311	0.051	0.217	0.179	0.288	0.072
CLC	0.344	0.030*	0.357	0.024*	0.443	0.004*	0.470	0.002*
CLP	0.237	0.141	0.210	0.194	0.237	0.140	0.234	0.145
PMA	0.452	0.003*	0.444	0.004*	0.491	0.001*	0.506	0.001*
PMC	0.444	0.004*	0.367	0.020*	0.288	0.072	0.387	0.014*
PMP	0.620	<0.001**	0.485	0.002*	0.403	0.010*	0.504	0.001*
PLA	0.413	0.008*	0.414	0.008*	0.195	0.229	0.325	0.041*
PLC	0.461	0.003*	0.433	0.005*	0.290	0.069	0.402	0.010*
PLP	0.364	0.021*	0.247	0.124	0.225	0.162	0.287	0.073
MF	0.367	0.020*	0.303	0.057	0.403	0.010*	0.454	0.003*
LF	0.172	0.288	0.267	0.095	0.363	0.021*	0.345	0.029*

*p<0.05; Significant; **p<0.001; Highly significant

DISCUSSION

Progressive loss of hyaline cartilage is one of the hallmark features of OA, initiated by a loss of proteoglycans (PGs) and an increase in water content, followed by loss of type II collagen and a change in collagen fiber orientation.[17] OA progression is usually graded based on plain radiographs, using joint space width, continuity of bony contours, and the presence and size of osteophytes as criteria.[18] However, these criteria do not help for the detection of early cartilage changes.[19] As articular cartilage has only limited capability for self-repair an early diagnosis of cartilage degeneration and a sensitive non-invasive diagnostic tool are highly desirable.

Recent MRI studies have included measurements of biomechanical and biochemical properties of cartilage such as the GAG and water content as well as the collagen organization and content.[20] A technique reported to quantify cartilage water content and collagen fiber orientation is quantitative T2 mapping. Focal increase in T2 relaxation time has been associated with cartilage matrix damage, in particular a loss of collagen integrity and an increase in water content.[21]

In present study of 33 patients with OA knee, OA was more common in females (63.6%) as compared to males (36.3%) which was comparable to the studies of Bhandarkar P et al. (2016) with more cases of females (63%) than males (37%).[22]

In our study, maximum patients of OA knee were found between age group of 61-70yrs (39.39%) which was comparable with the study of M.S. Radha (2014) with highest cases of OA knee in the age group of 60-65years.[23]

In the present study, out of 40 knees, K-L Grade was 0 in 3 cases, 1 in 8, 2 in 11, 3 in 8, and Grade 4 in 10 cases respectively. Out of these maximum cases were of K-L Grade 2.

In the present study, there was highly significant (p<0.001) increase in mean T2 values with increasing grade of osteoarthritis according to K-L Grade in all the compartments of cartilage of tibia [PLA: (K-L Grade 0=25.83±5.77 to Grade 4=50.50±7.14), PLC: (K-L Grade 0=24.16±2.88 to Grade 4=51.00±9.73), PLP: (Grade 0=24.16±2.88 to Grade 4=47.00±11.16), PMA: (Grade 0=22.50±0.00 to Grade 4=61.50 to 10.48), PMC: (Grade 0=22.50±0.00 to Grade 4= 58.00±9.22), PMP: (Grade 0=22.50± 0.00 to Grade 4=55.00±9.50) and trochlea of femur [MT: (Grade 0= 27.50±0.00 to Grade 4= 51.00±10.55), CT: (Grade 0=30.83±2.88 to Grade 4=53.00±9.55), LT: Grade (0=32.50±0.00 to Grade 4=52.00±11.16) and medial posterior condyle of femur [CMP: (Grade 0=27.50±0.00 to Grade 4=51.50±6.14) with only significant (p<0.05) increase in medial anterior and central condyle of femur (CMA, CMC) and no statistical (p>0.05) difference was found in rest of compartments of lateral condyle (CLA, CLC, CLP). This is comparable with the results of Buckwalter and Mankin (1998),[24] who have reviewed the mechanism of cartilage degradation in detail, and have ascribed the initial stage of OA changes to increased water mobility in the cartilage. Our longer T2 values found with increasing OA agree with this mechanism. Another study by Li X. et al,[25] (2007) also showed that mean T2 increased with KL scores (X-Ray) and overall cartilagelesion grade (analysis of clinical MR sequences). However, due to the small sample size (n=10 patients of OA), they could not test the statistical significance of this relation.

Our study showed highly significant correlation (p<0.001) between T2Map MRI for trochlea of femur (MT: r=0.637, CT: r=0.718, LT: r=0.694), medial posterior condyle of femur (CMP: r=0.822)

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and all the compartments of cartilage of Tibia (PLA: r=0.751, PLC: r=0.791, PLP: r=0.653, PMA: r=0.682, PMC: r=0.797, PMP: r=0.830) with K-L Grade. Our study found significant ($p<0.05$) correlation of CMC: r=0.454, CLC: r=0.371, CLP: r=0.328 and no correlation ($p>0.05$) with CMA: r=0.266, CLA: r=0.241 with K-L Grade. This is comparable with observations of Blumenkrantz G. et al,[26] (2004) in which T2 was observed to significantly ($p<0.05$) increase over time for all compartments except the lateral tibia and so they demonstrated a longitudinal relationship between the morphological changes in bone and cartilage structure in patients with varying degrees of OA. Another study by Klaus M. Friedrich et al,[27] (2009) observed small but positive correlation between the K-L Grade and the T2 values of cartilage. Our observation also fits with the results of morphologic studies of articular cartilage by Link et al,[28] (2003) who found a correlation between the K-L Grade and cartilage lesions on morphologic MRI.

Our result showed better correlation between T2 Map MRI values and WOMAC pain subscore than stiffness and physical function subscores. A highly significant correlation was found between WOMAC pain subscore of PMP ($p<0.001$) and significant ($p<0.05$) correlation between the segments of tibial cartilage (PMA, PMC, PMP, PLA, PLC, PLP), trochlea of femur (MT, CT, LT) and facets of patella (MF, LF) but it did not correlate well with femoral condyles (except CMP, CLC). Stiffness subscore significantly ($p<0.05$) correlated with LT, CMP, CLC, PMA, PMC, PMP, PLA, PLC compartments. Physical function significantly ($p<0.05$) correlated with only MT, LT, CMP, CLC, PMA, PM, PMF, LF segments on T2 Map MRI findings. Another study done by DUNN et al,[29] (2004) observed that there was a significant ($p<0.05$) positive correlation between the medial cartilage compartments of femur and tibia and WOMAC pain scores similar to our study. Also, a significant ($p<0.05$) positive correlation was found between mean cartilage T2 values and WOMAC function assessment for all compartments except the lateral tibia which did not match with our study. No significant correlation ($p>0.05$) was found between mean T2 values and WOMAC stiffness scores in their study.

There was a strong positive correlation between K-L Grade and WOMAC Score in study done by S. Singh et al (2014).[30] The correlation coefficient was found to be r= 0.904. Our results were comparable with significant correlation coefficient ($r=0.552$; $p<0.05$) with their study.

The limitation in our study was that analysis was performed in the less patients with early OA (K-L Grade 0=3, Grade 1=8) to investigate the normal distribution of cartilage T2 relaxation time and to correlate with severe OA of the knee joint. T2 values obtained with different acquisition methods and at different MRI scanners showed substantial variations.[31] Thus, the same T2 acquisition method and calibration procedures are mandatory to assure a reliable comparison of T2 measurements longitudinally and across different MRI scanners. Also a fully-automated segmentation algorithm for T2 maps seems to be the best way to implement T2 relaxation time measurements in clinical practice.

Accepting these limitations, we also believe that our results add credence to the argument of association of increasing osteoarthritis with damage to hyaline cartilage. At present plain radiographs are still considered standard to diagnose and monitor knee OA. However, quantitative MRI parameters, such as T2 relaxation time measurements, allow for the evaluation of structural disruption in the cartilage matrix depicting early biochemical changes at initial stages of cartilage degeneration that occur before OA changes are seen on radiographs.[32] Associations between T2 measurements and cartilage degeneration have been demonstrated in numerous in-vivo studies,[33,34] as well as in animal studies,[35,36] and with histology in specimen studies in vitro.[37]

CONCLUSION

T2 Map of the articular cartilage findings are associated with cartilage matrix damage, in particular a loss of collagen integrity and an increase in water content. It correlates to morphologic imaging findings on X-Ray with clinical symptoms of osteoarthritis. The results of this study may indicate the potential of T2 Map MRI to quantify pathologic cartilage changes in conditions that alter the biomechanical properties of the knee joint.

T2 Map MRI is a noninvasive comprehensive tool for cartilage evaluation and can be used to identify individuals in a “pre-OA” disease state when no morphological changes have occurred but biochemical changes have started in the cartilage. Clinically, WOMAC pain subscore has better correlation than stiffness and physical function subscore with X-Ray and T2 Map MRI. Development of non-invasive methods to assess early cartilage matrix changes is potentially important to initiate early treatment, monitor disease progression, plan for operative procedure and follow-up of operative cartilage repair.

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