Original Article

A Descriptive Study on Histo-Morphometric Analysis of Age-Related Changes in Human Hip Articular Cartilage in Tertiary Care Setting

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Abstract

Background: The primary objective of this study was to systematically investigate the age-related alterations in the morphological, cellular, and histomorphometric characteristics of human hip articular cartilage. Specifically, the study aimed to establish the correlation between advancing age and cartilage thickness, explore the progression of degenerative changes, and identify potential histological biomarkers that could aid in the early diagnosis of joint diseases such as osteoarthritis. **Material and Methods:** A cross-sectional study was conducted using femoral head cartilage specimens collected from patients undergoing hemiarthroplasty for neck or femur fractures. A total of 60 participants, aged between 50 and 80 years, were included. The cartilage specimens underwent meticulous processing through various histological procedures, including fixation, decalcification, dehydration, and paraffin embedding. Hematoxylin and eosin (H&E) staining was utilised for detailed histopathological evaluation of cartilage morphology, cellularity, and matrix integrity. Statistical analysis was carried out using SPSS to examine the relationship between age and cartilage thickness. **Results:** A significant negative correlation ($r = -0.95$, $p = 0.003$) between age and cartilage thickness was observed, revealing a substantial reduction in cartilage thickness as age advanced. Cartilage thickness in individuals aged 50–55 years averaged 2.2 µm, which progressively decreased to 0.7 µm in the 76–80-year age group. Early degenerative changes, such as surface fibrillation, discontinuities, and a decrease in chondrocyte density, were detected as early as age 50. These histological alterations indicated the onset of cartilage degradation, often occurring before the clinical manifestation of symptoms like joint pain and stiffness. **Conclusion:** The study conclusively demonstrated that the structural integrity of hip articular cartilage deteriorates significantly with age, as evidenced by a marked reduction in cartilage thickness and the presence of degenerative changes. These alterations are characteristic of the early stages of osteoarthritis, highlighting the importance of early detection and intervention to prevent further joint degeneration. Histological analysis provided invaluable insights into the mechanisms of cartilage degradation and suggested promising therapeutic approaches, such as stem cell or gene therapy, aimed at reversing or alleviating the effects of ageing on cartilage health.

Keywords: Ageing, Cartilage Degeneration, Osteoarthritis, Histomorphometry, Chondrocytes, Cartilage Thickness, Joint Pathologies, Regenerative Medicine, Early Detection, Hip Articular Cartilage.

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Introduction

A chondrocyte is a specialised cell in cartilage. It is key in maintaining the structure and function of cartilage tissue. These cells create and retain the extracellular matrix, which is a complex network of proteins, collagen, and other molecules that impart strength, flexibility, and recovery properties to cartilage. Cartilage integrity is maintained by the chondrocyte cells which remodel the extracellular matrix to support weight-bearing loads against joints. In normal cartilage, chondrocytes retain an equilibrium between the production of new ECM components and the degradation of the old ones, which assists in maintaining the shape of the cartilage. They, too, are mechanosensitive and modify the production of ECM in the face of loading changes within the cartilage.[1]

However, during ageing, chondrocyte function alters, promoting cartilage degeneration. Ageing is also associated with a decreased capacity of chondrocytes to produce ECM components like type II collagen and proteoglycan. This results in mildew on the cartilage in an early stage, and it loses the ability to resist the load. In addition, old chondrocytes are more active in the production of degradative enzymes (i.e., MMPs) for ECM degradation. The disruption of the balance between the formation and depletion of the ECM is responsible for the degradation of cartilage. Additionally, ageing chondrocytes can undergo

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morphological changes and become larger and more metabolically active (hypertrophic), although this response does not usually result in repair but in further breakdown. The number of chondrocytes in cartilage decreases dramatically with ageing, restricting the tissue's capacity for repair after damage.[1]

The relevance of these age-related modifications in chondrocyte function is of high importance regarding joint diseases, particularly osteoarthritis. OA is a chronic joint disease caused by the progressive disintegration of cartilage, causing joint pain, stiffness, and loss of joint function. As is known, chondrocytes in OA experience dramatic changes in their phenotype and functions. A significant feature of OA is the chondrocyte apoptosis, or programmed cell death, that depletes the number of chondrocytes responsible for maintaining the cartilage. OA chondrocytes also stimulate the production of matrix-degrading enzymes, including MMPs and aggrecanases, which result in the degradation of cartilage. As degeneration of the cartilage progresses, the ECM is worn away, resulting in rough, fibrillated cartilage surfaces that are no longer able to cushion the joint. In reaction to this deterioration, the affected bone beneath the cartilage may develop bony overgrowths, called osteophytes, that can also add to joint pain and loss of motion.[1]

Histologically, OA exhibits various alterations, which represent the developing course of cartilage degeneration. This involves the reduction in proteoglycans, which lessens the cartilage's capacity to hold water and influences its resistance to compression. The surface of the cartilage breaks down and fibrillates when the tissue is not smooth, along with a loss of structural integrity of the tissue. Together, these changes result in a gradual destruction of cartilage that underlies the pain, swelling, and physical dysfunction seen in OA patients. Further, the breakdown of cartilage, which is the hallmark of OA, is often associated with inflammation of the synovium, the lining of the joint, which worsens the condition.[1]

The destruction of cartilage and the involvement of chondrocytes in the latter play the most critical roles in the pathogenesis of joint diseases. Knowledge of the mechanisms behind chondrocyte dysfunction and cartilage breakdown can help develop novel diagnostic and treatment approaches. Newer imaging modalities and biomarkers would allow for earlier detection of cartilage harm, possibly allowing for intervention to prevent the death of cartilage. Moreover, the latest therapeutic strategies for rebalancing cartilage synthesis and degradation provide hope for controlling OA and other joint" disorders. These could include direct action against the responsible enzymes of the cartilage destruction, with stimulation of the chondrocytes, to the promotion of ECM formation, or exploring in the future new regeneration methods, such as stem cells or gene therapy, may be utilised to repair injured existing cartilage.[1]

Research in the chondrocyte milieu and its responsibility for the homeostasis of cartilage is essential in the scope of joint diseases such as OA. The alterations of chondrocyte morphology and function with ageing are responsible for the degradation of cartilage that causes joint stiffness, pain, and disability. By elucidating these processes, we can open up new avenues for therapeutic interventions that aim to stop, prevent, and even cure the underlying mechanisms of cartilage degeneration, thereby ultimately improving the lives of patients with joint diseases.

Objectives

1. To evaluate the age-related changes in chondrocyte morphology, density, and distribution in human hip articular cartilage.
2. To assess the correlation between age and cartilage thickness and its degeneration.
3. To identify potential histological biomarkers for early detection of joint diseases like osteoarthritis.
4. Many studies were based on hip joint radiology.

Scientific Relevance

1. This study can contribute to understanding the role of chondrocytes in the pathogenesis of joint diseases.
2. The study's results can have implications for understanding joint diseases and developing regenerative medicine approaches.
3. Collagen supplements, such as collagen peptides, may support joint health by promoting cartilage regeneration and reducing inflammation.

Review of Literature

Ageing and Cartilage Degeneration

The structure and function of cartilage in the joints change significantly with ageing. Cartilage is a specialised tissue composed of an extracellular matrix and cells called chondrocytes, which both degrade and synthesise the molecules comprising the ECM. The primary role of cartilage is to cushion and provide slip in the joints that minimise friction and aid fluid body movement. However, as we age, cartilage gradually loses its ability to perform its function and begins to break down.

This degradation is an essential component in the progression of joint diseases, including osteoarthritis, a debilitating loss of cartilage.

Multiple investigations have studied age-altered cartilage composition and morphology and have shown unequivocally that there exists a relationship between ageing and cartilage degeneration.The first evidence that such differences were significant for both matrix thickness and volume was reported by Mankin et al. (1971), with comparisons made between younger and older adults. They pointed out that the cartilage of weight-bearing joints, such as the knee and hip, is considerably thinner with age and that the volume of cartilage is also reduced. This observation was essential to demonstrate the physical changes that take place in cartilage in the process of ageing and degeneration, such as the appearance of OA.[2] In addition to these findings, Meacham et al. (1977) showed that cartilage thickness is reduced with ageing, confirming that cartilage wear decreases over time as human beings get older. As the cartilage thins, the joint becomes unstable and is more prone to damage. These age-associated changes are also especially problematic in the context of OA, given that cartilage destruction is one of the key features of the disease.[3]

Osteoarthritis and Cartilage Volume

Osteoarthritis is a prevalent degenerative joint disease associated with ageing. With age, cartilage remodels and changes occur that contribute to OA pathology, that is, loss of cartilage volume. A

study by Muir et al. (2002) was centred on cartilage volume loss in OA patients. They showed that beyond general depletion of cartilage with ageing, OA also includes a disproportionately substantial decrease in the volume of cartilage, especially at later-stage disease.[4] This cartilage loss is combined with the loss of normal cartilage function, which results in pain, stiffness, and limitation of joint movement. This study stressed the importance of a gradual decrease in cartilage volume in the OA bone, which is an essential factor in the patient's symptoms.

Cellular and Molecular Changes in Ageing Cartilage

The ageing changes of cartilage are the result of cellular and molecular changes. Kim et al. (2003) found that the density and viability of chondrocytes decreased with age.[5] The chondrocytes are indispensable for normal cartilage function because they can manufacture and organise the ECM. When people age, the number of functional chondrocytes of cartilage decreases, resulting in a decrease in the synthesis of significant components of ECM, such as collagen and proteoglycans. This loss of chondrocyte cell density and function contributes to the reduced capacity of cartilage for self-repair, causing it to become more susceptible to degeneration. Decreased chondrocyte viability leads to a lack of efficient cartilage repair such that cartilage in the joints is gradually destroyed.

Ageing cartilage also experiences dramatic alterations in its ECM content besides that observed from a reduced chondrocyte's activity. Clarke et al. (2011) reported that the cartilage matrix changes with age, especially the content of collagen and proteoglycans. The cartilage ECM is an essential component for the strength and elasticity of cartilage; alterations in the composition of the ECM can result in weakened cartilage. As people age, their cartilage matrix becomes less compact and less able to bear mechanical pressure, and that is a reason behind the deterioration of cartilage. This modified matrix content is, in part, responsible for the initiation of OA, as the cartilage becomes less resistant to insults and more susceptible to degradation.[6]

Fibrillation and Cartilage Damage

The enhanced production of fibrillation and fissuring is among the most obvious alternate appearances in the cartilage of old age. Hollander et al. (2005) showed that in older age, cartilage undergoes increased fibrillation, in which the collagen fibres in the cartilage start to unravel.[7] This fray is a precursor to the creation of cracks or fissures on the cartilage surface, destroying the strength of the cartilage. These cartilage surface structural defects are a means to accelerate further cartilage loss and are a source of symptoms in OA, including joint pain and stiffness. Fibrillation and fissure formation render the cartilage more susceptible to erosion and inhibit its ability to function correctly as a cushion and lubricant within the joints.

Implications for Osteoarthritis Pathogenesis

The age-dependent changes in cartilage discussed above emphasise the key role of cartilage degradation in the development of OA. Progression of OA is particularly characterised by the decrease of cartilage thickness, volume depletion, chondrocyte viability, changes of contents within the matrix, and increase of fibrillation. When the cartilage is worn thin and breaks down, the bone underneath may become exposed to more force and more pressure and can be damaged, which causes more inflammation and more destruction. This triggers a vicious cycle in which damage to cartilage causes inflammation of the joint, and that inflammation then drives degeneration of the cartilage.

These results emphasise the importance of investigating the cell biology and molecular biology of cartilage degeneration in ageing. The study of these changes will help to increase understanding of the pathogenesis of OA and devise more effective prevention and treatment modalities. For example, interventions aimed at saving chondrocytes (and/or inducing matrix repair) may delay and/or reverse the degradation of cartilage. In addition, regenerative interventions (stem cell therapy or gene therapy) could have the potential to (augment) cartilage repair or regeneration in ageing patients.

Materials and Methods

Study Design and Setup

The present study was carried out in SVMCH & RI, Histology Lab, and Hospital, and a cross-sectional study design was used. The study aimed to analyse the femoral head specimens taken from patients undergoing hemiarthroplasty for neck or femur fractures. 60 subjects were enrolled, and data were collected within 6 months. The institution's ethical committee approved the study, and the patients signed an informed consent form after receiving an adequate explanation of the purpose of the study.

Sample Collection and Preparation

Femoral head samples, including those with intact hyaline cartilage (macroscopically normal), were obtained from patients who had undergone partial hip replacement for neck or femur fractures. These samples were collected from the operating theatre of the Department of Orthopaedics, SVMCH & RI. After collection of the specimens, they were fixed in 40% formalin to preserve the tissue structure for histological study. The fixation procedure had an excellent ability to protect the specimens in a sound condition suitable for exact measurements. [Figure 1a,b]

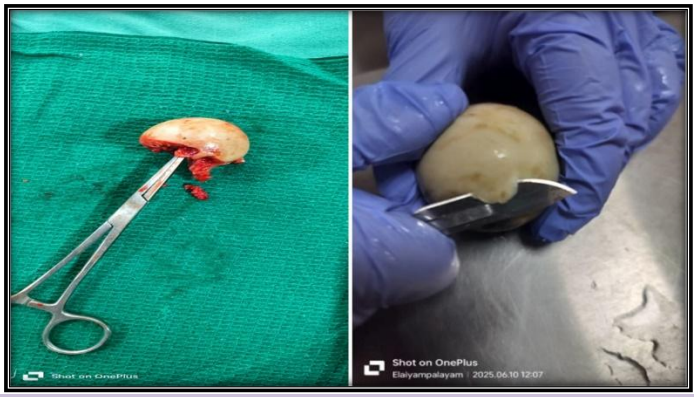

Figure 1: Well-preserved specimens after fixation for measurements

Histopathological Processing

Several procedures will be followed during the histopathological processing of the cartilage tissue obtained to make the samples suitable for extensive microscopic evaluation. Consequently,

cartilage samples were initially fixed for 24–48h in 10% NBF to achieve optimal preservation. Following fixation, the specimens were decalcified in 10% formic acid overnight since decalcification by formic acid was necessary for eliminating the calcium deposits in the bone and for rendering the cartilage better for sectioning.[8]

The samples will then be dehydrated in graded ethanol solutions. This step will allow the tissue to absorb water, and then the tissue was cleared in xylene, a solvent that made it ready for paraffin embedding. When the tissue is fully embedded, sections (5-7 µm-thick) will be cut with a rotary microtome. This fine sectioning permitted the microscopic study of the tissue.[9,10]

Histological Staining and Evaluation

The tissue sections were stained with Hematoxylin and Eosin for Histological examination. Indeed, H&E staining appears to be a widely utilised method to display the morphology and structure of the tissue and elucidate the contrast among various cartilage components (Wang et al., 2015). The stained sections were thereafter evaluated at a light microscope for general appearance and overview of cartilage integrity.

Comparison between histology and histopathological analysis was performed to investigate the cartilage thickness and cellularity. Both were significant predictors of cartilage health. The cartilage thickness and cellularity were assessed to estimate the damage or degeneration of the cartilage and the number and state of chondrocytes within the cartilage. The measurement of these factors was analysed with a tri-ocular microscope and Magnum software, which were used to analyse and measure the specimens even under the microscope.

Inclusion Criteria

1. Patients Scheduled for Joint Replacement Surgery.
2. Availability of femoral head cartilage
3. Age: >50 - <80 Years

Exclusion Criteria

1. Any Joint Inflammatory Disease
2. Metabolic bone disease / Malignancy / Genetic disease
3. Previous Joint Replacement surgeries
4. Inadequate tissue quality/quantity

Parameters of Study

Primary Parameter

1. Cartilage thickness

[Figure 2], Cartilage Morphology

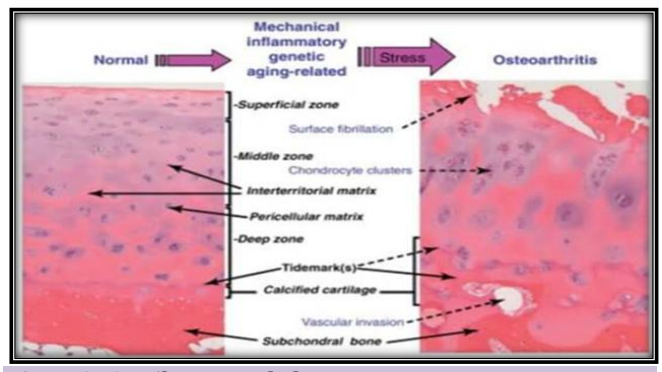

Figure 2: Cartilage Morphology

Source: Ageing and Osteoarthritis.[11]

Histological Observations with Increasing Age

As age increased, a variety of critical histological changes in cartilage were identified. These alterations contributed useful information on the degradation of cartilage and applied to joint diseases, such as osteoarthritis. The main observations are summarised as follows:

Decreased cartilage thickness: With ageing, the cartilage showed a reduced thickness, indicating a reduction in the integrity of the cartilage and the ability to resist mechanical loads. Such cartilage thinning was a sign of degeneration and made the cartilage less effective at providing motion and wear protection for the joint.

Reduced Cell Density: A decrease in the number of chondrocytes was also found with age. Chondrocytes are the sole cell type that synthesises the extracellular matrix (ECM) of cartilage. During ageing, the number of chondrocytes is lowered, and with it, the capacity to repair destroyed cartilage. Cartilage integrity became more damaged because of the loss of chondrocytes.

Increased matrix space: The matrix typically supports the structure of cartilage. Still, when the equilibrium is lost, then more space will be visible in the matrix. This change can be attributed to the changed composition of the cartilage along with the loss of mechanical strength, which accompanied the degeneration of the cartilage.

Surface Fibrillation: Fibrillation and irregularities on the cartilage surface were observed as the cartilage aged. Fibrillation is the wearing away of the collagen surface of the cartilage. These surface defects were the result of cartilage wear and were frequently associated with clinical clefts. Fibrillations suggested that the capacity of the cartilage to function as a smooth, lubricated interface of joint motion was compromised.

Tidemark Advancement: The advancement of the tidemark between the calcified and non-calcified zones of cartilage was an age-related phenomenon. The tidemark is an essential line that preserves the architecture of the cartilage. This boundary changed with age, demonstrating an increase in the amount of calcification in the cartilage. Tidemark progression denoted changes in the cartilage structural composition that are typical for cartilage degeneration and early osteoarthritis.

Histological Observations With increasing age, [Table 1]

Table 1: Histological Observations with increasing age

S. No	Age Group (years)	Mean Cartilage Thickness (µm)
1	50–55	2.2
2	56–60	1.9
3	61–65	1.6
4	66–70	1.3
5	71–75	1.0
6	76–80	0.7

Results

Results were analysed statistically by SPSS, and a significantly negative correlation was observed between age and cartilage thickness. The correlation coefficient was $r=-0.95$, $p=0.003$. This demonstrates a strong negative correlation between age and cartilage thickness. With age, the thickness of the cartilage decreases rapidly. The p-value (0.003) indicates that this is a

statistically significant result and not merely the result of chance. [Table 1]

Specifically, cartilage thickness was further analysed by age group, and the main findings of the descriptive analysis were as follows:

Decreased cartilage thickness: Cartilage thickness decreased in individuals of older age. The average thickness of cartilage in the group aged 50–55 years was 2.2 μm, which decreased to 0.7 μm in the group aged 76–80 years. This suggests that the ageing process degeneratively affects cartilage and results in less capability to serve the joint efficiently.

Trend Indicates Commonality between Both Genders: The general trend associated with age in the decrease of cartilage thickness was common in both male and female subjects. This concordance is further evidence that age-associated cartilage degeneration is a widely occurring process and that it is not substantially sex-dependent.

Histological Studies on Early Degenerative Changes: Early degenerative changes in the cartilage were noted at the age of 50 years. These changes included:

Surface fibrillation: The randomness of the surface, the character of fraying, and the damage of the collagen fibres; fibrillation over parts of the cartilage surface is evident. This is an early signal of cartilage degradation.

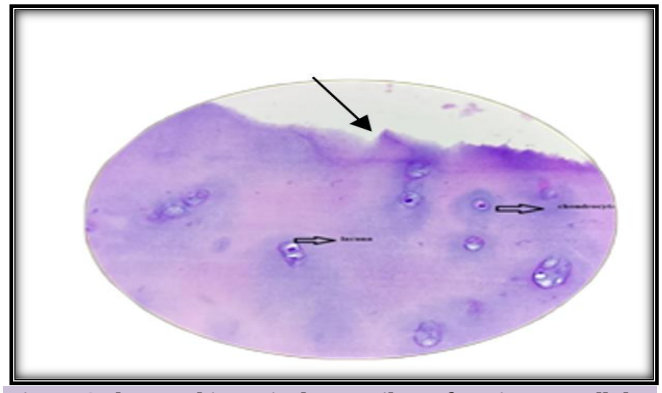

Figure 3: human hip articular cartilage, focusing on cellular changes (surface fibrillation) observed in 53-year-old samples stained with Hematoxylin & Eosin. 40x magnification

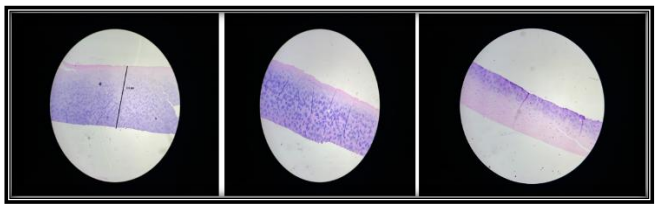

Figure 4: human hip articular cartilage, focusing on cellular changes observed in 53,56 & 74 -74-year-old samples stained with Hematoxylin & Eosin. 5x magnification.

Discontinuity: The cracks, fissures, or clefts identified on the cartilage surface, the presence of which was indicative of the loss of the integrity of the cartilage.

Sparse Chondrocyte Cells: The chondrocyte, which is the cell that keeps and regenerates the cartilage, is sparse. This lower cellularity makes the cartilage less able to heal itself. [Figure 4]

More expansive Matrix Space: The interchondrocyte matrix was widened, which suggested the compromise of architectural structure and the undermining of the cartilage. [Figure 5]

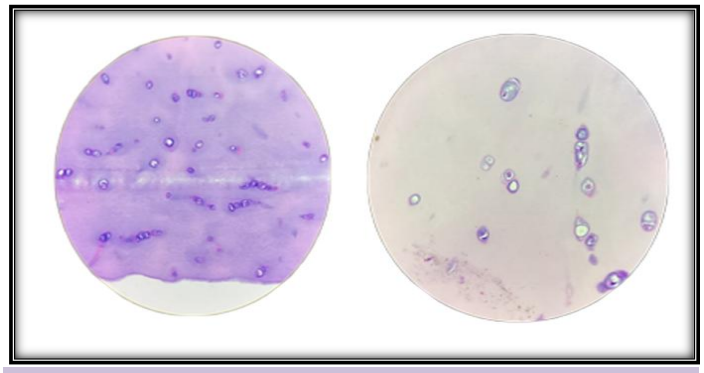

Figure 5: human hip articular cartilage, focusing on cellular changes (reduced density, increased matrix) observed in 69 and 80-year-old samples stained with Hematoxylin & Eosin. 40x magnification.

Diminished basophilic activity: The strength of staining of basophilic structures is decreased, denoting a reduction in metabolic activity of chondrocytes, and is thus related to cartilage degeneration. [Figure 6]

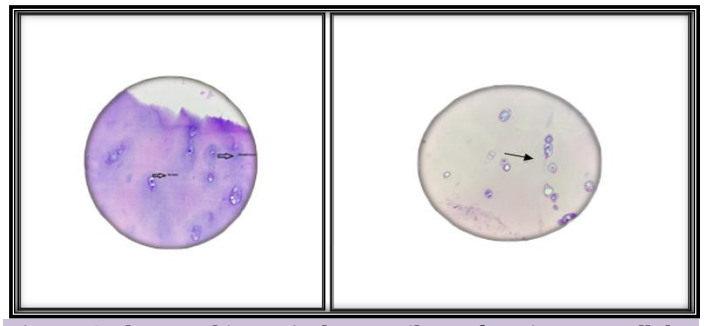

Figure 6: human hip articular cartilage, focusing on cellular changes (diminished basophilic) observed in 69 and 80-year-old samples stained with Hematoxylin & Eosin. 40x magnification.

Tidemark advancement: The tidemark, demarcating calcified and non-calcified cartilage, (near subchondral bone) showed signs of advancement. [Figure 7]

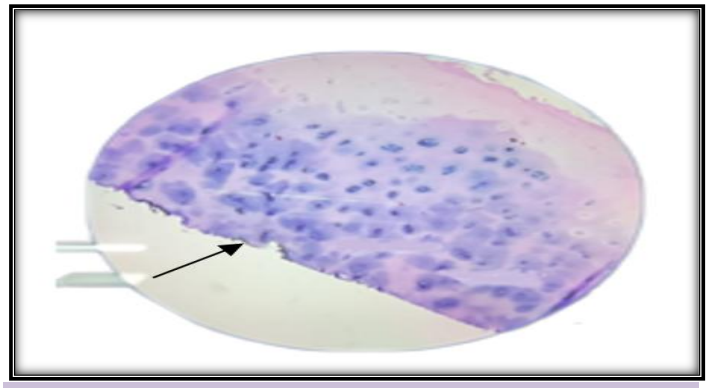

Figure 7: human hip articular cartilage, focusing on cellular changes observed in 56-year-old samples stained with Hematoxylin & Eosin. 40x magnification

These histological changes were signs of the onset of cartilage degeneration at an early stage of life, which would be present before symptoms such as pain or stiffness manifest.

Discussion

In the current investigation, there was a significantly strong negative relationship ($r = -0.95$, $p = 0.003$) between age and cartilage thickness, revealing a linear decrease in cartilage thickness with advancing age. The mean thickness of the cartilage was 2.2 μm in 50–55-year-olds and 0.7 μm in the 76–80-year-olds. This pattern was observed for both genders. Pathological examination revealed degenerative changes in the cartilage at an age as young as 50 years that include fibrillation of the surface, disruption of the cartilage structure, fewer chondrocyte-like cells, a decrease in the eosinophilic activity, and matrix space. This indicates that the breakdown of cartilage can begin at a young age – well before the joints start feeling painful or stiff, which is a characteristic of osteoarthritis.

Compared to previous reports, there are several similarities and differences between these findings. Mechlenburg et al. noticed that mean humeral head cartilage thickness declines with ageing, and in the current study, a decrease in chondrogenic tissue thickness with age was found.[11] Another research article by Wyler et al. reported that femoral cartilage thickness varied from 1.18 mm to 1.78 mm. That cartilage thickness decreased with age, which was consistent with our results, which showed that cartilage thickness decreased over time.[12] However, the study of Mecklenburg was performed with MRI and stereology as methods of measurement, in contrast to histological measurements in the present study, which can explain the variation in methodology but not the detection of cartilage degradation.

Wyler also found that there is a decrease in the thickness of cartilage with age, which is in line with our results. Wyler et al. utilized spiral CT and arthrography measurements in their analysis, which are innovative imaging methods capable of analyzing cartilage in detail. The current study, in contrast, was histological, offering a microscopic perspective on cartilage alteration.[12] Although the techniques used were different, both studies detected ageing-related depletion of cartilage. However, an opposing view was given by Armstrong et al., who pointed out that between 20 and 45 years of age, the thickening of articular cartilage occurs.[13] In the current study, old age groups were examined at which cartilage thinning and degeneration are more marked than in younger subjects, so such an observation does not contradict the present results. Armstrong's study may have represented the early phases of cartilage growth, while the current study focuses on the degenerative changes during ageing.

Lotz et al. also reported reduced cell density and cartilage thickness with increasing age, like those observed in the current study.[14] The reduction of chondrocyte counts, combined with the observation of cell clusters and matrix space increase, implies a comparable form of cartilage degradation in both studies. Altogether, these studies underscore the importance of ageing in the pathogenesis of cartilage destruction and have given us a better understanding of how age-associated tissue changes contribute to the onset of joint diseases such as OA.

Conclusion

The thickness of cartilage decreases with age, suggesting one of the most important adverse effects of ageing on joint health. This reduction in cartilage is a regular part of ageing and is associated with several degenerative changes within the cartilage. A loss of cartilage thickness can cause joint pain, stiffness, and a restricted range of motion, particularly in weight-bearing joints such as the knee and hip and is a primary cause of diseases such as osteoarthritis. Histological analysis of cartilage offers a non-comparable precision of information about cartilage morphology and is considered the gold standard of cartilage health evaluation. Histology can provide a close look at the structural integrity of the cartilage and the main changes, such as surface fibrillation, matrix composition changes, and chondrocyte density. This detailed perspective of cartilage degeneration can shed light on both the ageing-associated effects of cartilage at the cellular and molecular levels and provide a more realistic modelling of the degenerative process. Knowledge about the age-related changes within the cartilage is essential for the development of regenerative medicine protocols. These approaches could involve therapies that aim at inhibiting collagen synthesis since collagen is necessary for maintaining the integrity and function of cartilage. Regenerative approaches, such as stem cell therapy or gene therapy, might be helpful in stimulating cartilage repair and perhaps in reversing some of the damage of ageing. Promoting the body's regenerative capability to heal the cartilage using these two treatments completely represents a treatment whose time has come, and new hope for sufferers of cartilage diseases, and increasing joint functionality of the aged population. These results add to the increasing evidence on the ageing of cartilage and its importance for joint health. Because of the growing world population, there is a rising demand for suitable alternatives for the treatment of cartilage degeneration and joint diseases. The information from this study will provide knowledge for future investigations and therapeutic applications that either support cartilage in situ or strive to regrow cartilage, which would improve the quality of life for people suffering from age-related diseases of the joint.

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Nil.

Conflicts of interest

There are no conflicts of interest.

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