

## Case Report

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# Heberden's nodes and joint capsule fibrosis

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### Abstract

The joint cartilage, subchondral bone and synovium are currently thought to be responsible for the different structural changes seen during the evolution of disease process in osteoarthritis. There is not enough published literature about joint capsule changes in Heberden's nodes, one of the most common manifestations of osteoarthritis. To investigate is possible role of the joint capsule in the pathologic process in the development of Heberden's nodes. We report the histological examination of joint capsule distal interphalangeal joints in two patients with Heberden's nodes. We excised V shaped joint capsule parts longitudinally and laterally of distal interphalangeal joints of patients with Heberden's arthritis and aimed to see histopathologic examination of capsule. Histological examination showed the capsule to be composed of hyalinized collagenous stroma containing thickened, closely packed, hypo cellular collagen bundles and small blood vessels and some mature lipocytes groups. The study revealed that fibroblast proliferation and fibrosis occurs in the joint capsule of Heberden's nodes, we postulated those changes are predictive of osseous proliferation and further research to be established if the joint capsule or soft tissue around the distal interphalangeal joints contribute in the pathological process in osteoarthritis.

**Keywords:** Heberden's nodes, joint capsule, fibrosis, osteoarthritis

### Introduction

Osteoarthritis (OA) represents a common age-related disease characterized by progressive joint destruction, loss of function, and joint failure. Generalized nodal OA represents a striking example of this bone-forming phenotype. Nodal OA may present acutely with painful joint swelling or gelatinous synovial cysts, known as Heberden's nodes (HNs), which subsequently are associated with ossification and radiographic features of OA. Ossification transformation of tendinous insertion into the joint capsule creates the osteophytes [1].

The evolution of osteoarthritis disease process divided into three stages. Stage 1; proteolytic breakdown of cartilage matrix, Stage 2; fibrillation and erosion of cartilage matrix, Stage 3; chronic inflammatory reaction in synovium and additional cartilage breakdown. Subchondral bone remodeling and osteophyte formation follows the third stage [2].

In some instances myxoid transformation of the periarticular fibro adipose tissue is associated with proliferation of myxoid fibroblasts and cyst formation [3]. Radiological DIP joint osteophytes are a better marker of knee and multiple joint OA than Heberden's nodes [4]. Alexander et al. suggested a set of hypotheses

about Heberden's and Bouchard nodes; lateral nodes are palpable osteophytes and marker for OA [5]. The central nodes are traction spurs and marker of contracture. They commonly coexist and fuse.

While the disease process is fairly complicated one, involving three important tissues (cartilage, synovial membrane, and subchondral bone), a number of pathways have been identified as being responsible for the different structural changes seen during the evolution of the disease process [6].

In inflammatory osteoarthritis low-grade synovitis leads to capsular thickening and fibrotic shortenings, which may cause pain and muscle spasm [7]. Lloyd-Robertset al. explained that the low-grade synovitis led to capsular thickening and fibroid shortening, which might cause pain and muscle spasm in osteoarthritis of hip joint [8]. Those changes of capsular tissue were not well documented in Heberden's nodes in the literature. This is the first study on the evaluation of joint capsular structure in Heberden's nodes.

Based on those literature data we thought that in Heberden's nodes, some additional different pathological abnormalities might be seen in joint capsule, such as fibrosis, cartilaginous or bony metaplasia. We excised V shaped joint capsule parts longitudinally and laterally of distal interphalangeal joints of patients with Heberden's arthritis and aimed to see histopathologic examination of capsule. After was procedure was told to patients and a written consent was obtained.

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## Case 1

Fifty-five years old, housewife complained of pain in her hands and knee during long distance walk and climbing stairs. She had been in menopause for seven years. Routine laboratory tests and acute phase reactants were normal. On physical examination she had Heberden's nodes, especially very hypertrophied in the forth and fifth fingers. Radiological findings were consistent with Heberden arthritis of the finger joints.

## Case 2

A 63 years old, retired female, had a complaint of pain on walk and pain in her hands. She had Heberden's nodes in both hands. Physical examination yielded quite large Heberden's nodes especially in the third and forth fingers. There were no other clinical or serological signs of other rheumatological diseases, like psoriatic or rheumatoid arthritis. Routine laboratory tests were normal. X-rays of the joints showed osteophyte formation in dip joints of both hands.

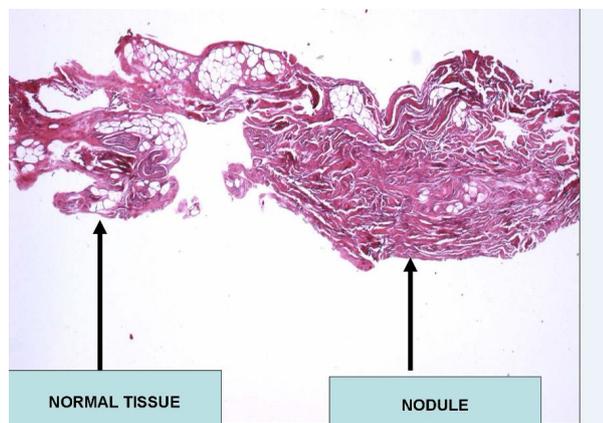
## Surgical Technique

In case 1 longitudinal skin incision of one cm. was made on dorsa-medial of DIP joint of left hand little finger under sterile conditions with digital nerve block. The hypertrophied joint capsule was exposed after subcutaneous incision. V-shaped joint capsule together with synovial tissue parallel to skin incision was excised. After then the joint capsule and skin was closed routinely. In case 2, the same biopsy procedure was made in DIP joint of the left long finger.

## Histopathology

Histological examination showed that both materials were composed of hyalinized collagenous stroma containing thickened, closely packed, hypos cellular collagen bundles and small blood vessels and some mature lipocytes groups (Fig. 1).

There were no inflammation, hemorrhage and necrosis. Dense collagenous matrix was verified by Masson trichrome staining



**Figure 1.** Normal tissue on the left and Heberden's nodes in the right are seen (Hematoxylin-Eosin stain, original magnification, X 2.5

## Discussion

Based on the current data starting tissue of osteoarthritis are subchondral bone and cartilage [2,3,7,8]. Some researchers may still ask; does OA start in elsewhere?

Tan et al concluded Heberden's and Bouchard's nodes formation occurred at regions where soft tissue bulged through the capsule between the dorsal tendons and collateral ligaments [9]. The high-resolution images of every joint structure showed comparable abnormalities in both early and chronic OA, including cartilage loss, bone edema, synovial enhancement, osteophytosis, and erosions.

In 1996 Grieve et al. published the first histopathological report on typical lateral nodes, confirming the constant presence of an osteophyte, and a histological study of postmortem subjects with nodes confirmed their finding [10]. In both studies it was noted that the sub-nodal osteophytes could arise from either or both phalanges.

Irlenbush et al. investigated morphologically 218 fingers from 56 cadavers and concluded that OA starts with a sub-chondral ossification [11-12]. At this time the surface of cartilage was not yet destroyed. Reactive tidemark flaking is the beginning of the general degradation. When compared to a control group a significant differentiation by histological score were possible.

Another group of researchers postulated that although HNs are forerunners of new bone formation, no significant osteogenic bias was seen in any HN-MSC (mesenchymal cell) cultures. This may be explained by the fact that MSCs are unlikely to have intrinsic osteogenic bias, perhaps with the aberrant new bone formation seen in HNs a result of subsequent alterations in joint biomechanics [13].

Although digital nodes remain largely unexplained, there is a consensus that Heberden's nodes are a strong marker of interphalangeal joint OA. Most investigators have concluded that they are caused by osteophytes [11].

We assumed that a progressive pathologic process could occur such as fibroblast proliferation, fibrosis, and cartilaginous and bony metaplasia. We saw the first and second stages of pathological abnormalities in joint capsule histopathological examination.

In our study obtaining tissue samples from distal interphalangeal joint capsule represented a novel way for exploring the micro anatomic basis of Heberden's nodes and may have considerable potential in the clinical setting and a new approach in the treatment of osteoarthritis.

In the present evaluation in nodal OA, previously unappreciated capsular abnormalities could especially be appreciated. Further studies are therefore required to assess whether the same pathogenesis mechanism can be seen in OA of the large joints such as hips and knees.

## Conclusion

In conclusion, the changes of joint capsule could not to be responsible for the development of Heberden's nodes, therefore we are not postulating a new theory of the pathogenesis of Heberden's nodes. The changes of the joint capsule are not more be seen as a part of the degenerative process of the whole joint but could be contributing findings in degenerative process.

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