

**Study Of Wide Nailfold Capillary Changes  
(Capillaroscopy) In Patients With  
Osteoarthritis Of Hands**

**Thesis Submitted For The Partial Fulfillment  
Of M.Sc. degree In  
Internal Medicine**

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بِسْمِ اللَّهِ الرَّحْمَنِ الرَّحِيمِ

قَالُوا سُبْحَانَكَ لَا عِلْمَ لَنَا إِلَّا مَا عَلَّمْتَنَا  
إِنَّكَ أَنْتَ الْعَلِيمُ الْحَكِيمُ

صدق الله العظيم

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# Introduction & Aim of The Work

## INTRODUCTION

Osteoarthritis is a slowly progressive monoarticular (or less commonly polyarticular) disorder of unknown cause and obscure pathogenesis. The condition occurs late in life, affecting principally the hands and large weight-bearing joints, and is characterized clinically by pain, deformity, enlargement of the joints, and limitation of motion. Pathologically, the disease is characterized by focal erosive lesions, cartilage destruction, subchondral sclerosis, cyst formation and large osteophytes at the margins of the joint. The disease appears to originate in the cartilage and the changes in that tissue, virtually pathognomonic, are progressively more severe with advancing disease; structural aberrations in the underlying bone and inflammatory alterations in the synovium are usually milder and thought to be secondary. Systemic abnormalities have not been detected. Therapeutically, the disorder is characterized by lack of a specific healing agent (*Mankin 1993*).

Description of nail changes in osteoarthritis (O.A.) of the distal interphalangeal (DIP) joints have been reported (*Alarcon-Segovia and Vega-Ortiz, 1981-Goldman et al., 1977 Cutolo et al., 1990*). These changes have been attributed to pressure exerted on the nail bed by mucinous cyst or osteophytes. In addition, inflammation of Heberden's nodes could interfere with nail growth. The scarcity of reports of nail changes could be assumed to imply that it is rare despite the large prevalence of such osteoarthritis.

To the best of our knowledge, however, the real prevalence of these possible complications of (O.A.) has not been previously investigated (*Cimino et al., 1994*).

With respect to the mechanisms underlying joint damage in (O.A.), some observations suggest that this condition may be related or predisposed to by disturbance in the microcirculation of the extremities, although there is controversy as to the nature of these abnormalities of the circulation (*Harrison et al., 1953-Schubert, Hamerman, 1968 and Lee et al., 1974*).

Nailfold capillaroscopy has been demonstrated to be useful in distinguishing between various connective tissue diseases, notably scleroderma, systemic lupus erythematosus and rheumatoid arthritis (*McGill and Gow, 1986*) but there have few reports of the abnormalities associated with (O.A.) of the fingers.

## AIM OF THE WORK

The aim of the present work is to study nail and/or finger alterations in a selected series of patients diagnosed as having osteoarthritis (O.A.) of the hand and the relation of these alterations to nail-fold capillary abnormalities as detected by capillary microscopy.

# Review of Literature

## OSTEOARTHRITIS

### DEFINITION:

A heterogeneous group of conditions sharing common pathologic and radiologic features.

Focal loss of articular cartilage in part of a synovial joint is accompanied by a hypertrophic reaction in the subchondral bone and margin of the joint.

Radiographic changes include joint space narrowing, subchondral sclerosis and cyst formation, and marginal osteophytosis.

Extremely common and age-related, with a particular predilection for the knees, hips, hands and the apophyseal joints of the spine.

Often accompanied by clinical manifestations which may include use-related joint pain, gelling of joints after inactivity and loss of range of joint movement (*Dieppe.,1994*).

### BASIC ASPECTS OF JOINTS:

#### Normal Articular Cartilage Structure:

The cells of the cartilage, chondrocytes, only comprise 2% of the tissue volume. The remainder of the tissue comprises the extracellular matrix. Eighty percent of the weight of cartilage is water. Of the dry weight, 70% is collagen (mostly type II), 20% are proteoglycans and 10% comprise non collagenous proteins (*Campion and Watt., 1994*).

### **Cartilage Homeostasis:**

Under physiologic conditions, chondrocytes regulate a dynamic metabolic steady state in which anabolism (synthesis of matrix macromolecules) is balanced by catabolism (degradation and loss from the matrix) (*Hascall et al., 1990*).

#### **Synthesis:**

Chondrocytes secrete proteoglycan components together with hyaluronic acid for proteoglycan manufacture. They also synthesize collagen.

#### **Degradation:**

Chondrocytes also secrete metalloproteases (collagenase, proteoglycanase), prostaglandins and cytokines. Certain of them e.g; IL1 and IL6 can increase the degradative process by stimulating prostaglandin synthesis and metalloprotease synthesis. In the adult, homeostasis process helps to maintain constant concentrations of the extra cellular matrix (ECM) components.

#### **Matrix Metalloproteases (M.M.Ps):**

Are endogenous degradative enzymes mostly proteases. They are secreted by the chondrocytes as a proenzymes that must be activated extracellularly either stromelysin or plasmin, which is derived from