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### Knee Osteoarthritis

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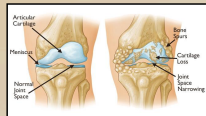
# Knee Osteoarthritis

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## Introduction

Osteoarthritis (OA) is a common age-related disorder of synovial joints that is classified as a noninflammatory joint disease (Ling & Bathon, 2018).

It is characterized by local areas of loss and damage of cartilage, new bone formation of joint margins, subchondral bone changes, variable degrees of mild synovitis, and thickening of the joint capsule (Ling & Bathon, 2018).



(OrthoInfo, 2017)

## Etiology

The exact cause of OA is unknown although it centers on load-bearing areas, specifically knee, hip, hand, finger, and spinal joints.

OA can be caused by any condition that damages cartilage, subjects the joint surfaces to chronic or excessive forces, or causes instability in the joint. It can also be caused by torn cartilage, dislocated joints, and ligaments injuries (Whelan, Heitz, & Higuera, 2018).

## Presentation of Case

Typically knee OA begins in adults over the age of 65. The distal and proximal interphalangeal joints of the hand have been identified as the joints most commonly affected by OA, but they are the least likely to be symptomatic.

In contrast, the knee, which is the second most common locations of radiographic OA, are nearly always symptomatic. The patient would likely present with common symptoms of OA, such as pain, swelling, inflammation, and also have one or more risk factors including: obesity, poor posture, joint malformation, and increased family history of knee OA (Ling & Bathon, 2018).

## Prevalence of OA

- OA affects over 30 million US adults (CDC, 2017)
- In the US population almost 40% people over 45 years old have some degree of OA
- These numbers are likely to increase over time based on:
  - Obesity epidemic, estimated 65 million more US adults to be diagnosed by 2030
  - Increased participation in sports- this leads to higher rates of injuries which will increase chances of developing OA (particularly in the knee)
  - Aging population, by 2060 92 million Americans will be 65 or older
  - (Mandl & Losina, 2015)

- According to the CDC (2017) many patients in the primary care setting present with comorbidities that will increase their chances of getting OA:
  - 27.7% of adults who have arthritis are also obese
  - 33.7% of adults who have arthritis also have diabetes
  - 36.4% of adults who have arthritis also have heart disease

- Knee osteoarthritis is a leading cause of disability that limits function and mobility, impairs quality of life. It is the primary indication for knee replacement, and is associated with substantial medical expenditure. A main factor in OA, particularly for the load-bearing knee joint, is excessive mechanical stress, which can lead to injury and irreversible damage to the joint (Qin et al., 2018).

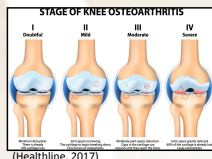
## Stages of OA

**Stage 0:** Normal healthy knee

**Stage 1:** Very minor bone spur growth, no pain or discomfort at this stage as a result of the very minor wear on the joint

**Stage 2:** "Mild" stage of the condition, X-rays of knee joints in this stage will reveal greater bone spur growth, this is the stage where people may first begin experiencing symptoms; pain after a long day of walking, greater stiffness in the joint when it's not used for several hours, or tenderness when bending

**Stage 3:** "Moderate" in this stage, the cartilage between bones shows obvious damage, and the space between the bones begins to narrow; this stage will present frequent pain when walking, running, bending, or kneeling and may experience joint stiffness after sitting for long periods of time or in the morning



(Healthline, 2017)

## Signs

- Inflammation
- Enlargement/deformity/misalignment of the joint
- Swelling
- Gait abnormalities- specific to knee OA

(CDC, 2017)

## Symptoms

- Pain in load-bearing joints
- Stiffness in load-bearing joints
- Tenderness
- Referred pain (higher incidence in spinal OA versus knee OA)

(Holland, 2017)

## Pathophysiological Process

- Osteoarthritis is a disease of cartilage. Cartilage is a flexible connective tissue with viscoelastic and compressive properties, which are imparted by its extracellular matrix, composed predominantly of type II collagen and proteoglycans (Ling & Bathon, 2018).

- In OA cartilage there is excess, in the extracellular matrix, of degrading enzymes shifting this balance in favor of net degradation, resulting in the loss of collagen and proteoglycans from the matrix (Ling & Bathon, 2018).

- In response to this loss of collagens and proteoglycans, chondrocytes initially proliferate and synthesize enhanced amounts of proteoglycan and collagen molecules. As the disease progresses, reparative attempts are outmatched by progressive cartilage degradation (Ling & Bathon, 2018).

- Fibrillation (flaking), erosion and cracking initially appear in the superficial layer of cartilage and progress over time to deeper layers, resulting eventually in large clinically observable erosions (Ling & Bathon, 2018).

- The primary enzymes responsible for the degradation of cartilage are the matrix metalloproteinases (MMPs). These enzymes are secreted by both synovial cells and chondrocytes and are categorized into three general categories: collagenases, stromelysins, and gelatinases (Ling & Bathon, 2018).

- In OA, synthesis of MMPs is greatly enhanced and the available inhibitors are overwhelmed, resulting in net degradation. Stromelysin can serve as an activator for its own proenzyme, as well as for procollagenase and prostromelysin, thus creating a positive feedback loop of proMMP activation in cartilage (Ling & Bathon, 2018).

- One candidate is interleukin-1 (IL-1). IL-1 is a potent pro-inflammatory cytokine that, in vitro, is capable of inducing chondrocytes and synovial cells to synthesize MMPs. IL-1 suppresses the synthesis of type II collagen and proteoglycans, and inhibits transforming growth factor- $\beta$  stimulated chondrocyte proliferation (Ling & Bathon, 2018).

- The presence of IL-1 RNA and protein have been confirmed in OA joints. IL-1 effects on OA include: actively promote cartilage degradation, suppress attempts at repair, and induces nitric oxide production, chondrocyte apoptosis, and prostaglandin synthesis, to further contribute to cartilage deterioration (Ling & Bathon, 2018).

## Significant Pathophysiology

- For primary care providers to adequately treat knee osteoarthritis they must be able to understand the underlying pathophysiology.

- Treatment of OA is mainly based on the pathophysiological events that alter the initiation and progression of OA.

- Understanding the mechanism and modulation of cytokines and MMPs would be a main target for treatment and prevention of osteoarthritis (Ling & Bathon, 2018).

- As stated MMPs and pro-inflammatory cytokines appear to be the most important mediators of cartilage destruction leading to OA. There are inhibitors of MMPs and cytokines but are inadequate in counteracting the degradative forces of OA (Ling & Bathon, 2018).

- New therapies and treatment for OA are focusing on reducing MMP activity and stimulating matrix synthesis (Ling & Bathon, 2018).

## Diagnosing

- Radiological studies (x-rays), CT scans, arthroscopy, MRI
- Blood tests are used to rule out other conditions that cause joint pain, such as Rheumatoid Arthritis or a joint fluid analysis can also be used to rule out gout or infection as the underlying cause (Holland, 2017)

- See figure below of right knee x-ray, left side (Figure 1) is a normal knee and right side (Figure 2) has OA



Figure 1 Figure 2  
(Healthline, 2017)

## Treatment

Current treatment for OA is limited to control of symptoms. At this time, there are no pharmacological agents capable of retarding the progression of OA or preventing OA (Manno & Bathon, 2018).

Treatment typically is focused on relief of pain, maintenance of quality of life, and preservation of functional independence (Manno & Bathon, 2018).

Pharmacology management includes medications such as:

- NSAID's or Acetaminophen
- COX-2 Inhibitors- pain and inflammation
- Opioids
- Corticosteroids
- Topical agents
- Intraarticular injections

Surgery can be an option for knee OA pain untreated by medication:

- Tibial Osteotomy
- Total Knee Arthroplasty

## Implications for Nursing Practice

To aid in the symptom management of OA patients need to educated on non-pharmacological treatment methods including:

- Exercise:** Encouraging leg/hip muscle strengthening exercises to help strength and protect the knee, low impact activity like walking, swimming, or biking a few times a week
- Weight loss:** Educating patients on the benefit of losing weight to help relieve the pressure off the joints that can cause pain
- Stretching:** Gentle stretching of joints may improve flexibility, lessen stiffness and reduce pain, yoga and tai chi also aid in preventing joint stiffness
- Adequate sleep:** Daily muscle rest will help reduce pain, swelling, and inflammation in joints
- Cold and Heat Therapy:** 20 minutes a day apply cold or hot compress to inflamed joint a few applications per day

(Manno & Bathon, 2018)

## Conclusion

Knee OA is a commonly diagnosed disorder seen over 45 percent of the US population. It has an increasing occurrence in the aging population and coincides with many comorbidities such as obesity, heart disease, and diabetes (Ling & Bathon, 2018).

This disease will be seen in the primary care setting and it is imperative that primary care providers understand this disease and the pathophysiology behind it in order to properly treat their patients. Pain control and maintaining quality of life are key treatment measures to ensure patients can properly control their disease.

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