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Osteoarthritis (OA)

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Osteoarthritis (OA)

Signs & Symptoms

- Pain: Affecting joints and acute pain during or after joint
- Stiffness: Joint and muscle stiffness after periods of inactivity or upon awakening
- **Swelling:** Soft tissue inflammation around joints.
- **Synovitis:** in advanced stages
- Loss of Flexibility or Motion: Unable to move joints through full range of motion
- **Tenderness:** Light pressure or tenderness near the inflamed
- **Bone Spurs**: Hard lumps can form around the joint and cause severe pain
- Grating Sensation: During joint movements may experience a popping or crackling sensation

Underlying Pathophysiology

(Primary-idiopathic vs. Secondary-trauma or mechanical misalignment)

- Multifactoral, not just a degenerative disease of cartilage
- **Involves:** trauma, mechanical forces, inflammation, biochemical reactions, metabolic derangements
- Cartilage is not vascular or innervated, alone will not cause
- Pain from the joint capsule, synovium, subchondral bone, ligaments, periarticular muscles
- As disease progresses the **non-cartilaginous components** are affected:
 - bone remodeling
 - osteophyte formation
 - weakening of periarticular muscles
 - laxity of ligaments
 - synovial effusion
- Chronic, low-grade inflammation, innate immune mechanisms
- Synovial fluid contains **inflammatory mediators** including:
 - Plasma proteins (c-reactive protein)
 - Prostaglandins (PGE2) Leukotrenes (LKB4)
 - Cytokines (TNF, IL1-beta, IL6, IL15, IL17, IL18, IL21)
 - Growth factors (TGF-beta, FGFs, VEGF, NGF)
 - Nitric oxide
 - Complement components
- These mediators induce matrix metalloproteinases and hydrolytic enzymes that breakdown cartilage d/t proteoglycan and collagen destruction. WBC molecules are released from the breakdown of extracellular matrix recognized by the innate immune cells (macrophages and mast cells). Overtime, tissue
- destruction occurs Animal studies suggest macrophages play a role in development of osteophytes (Mora, Przkora, & Cruz-Almeida,
- Protective growth factor mechanisms lacking with OA:
 - Insulin-like
 - Platelet-derived
 - Fibroblast 18
 - TGF-beta

Significance of Pathophysiology

Understanding the underlying cause is key for preventing, slowing the progression of and developing the best treatment plan. Exercise has been proven to decrease pain and morbidity in OA patients. "Muscle around the affected joints become stronger, bone loss and joint swelling decrease, and stiffness and pain improve thanks to a better lubrication of the cartilage" (Castrogiovanni, etal, 2019, P. 2)

OA Prevention-physical activity

- (Rat studies suggest moderate physical activity is the key) Synovium has multiple functions:
 - Lubrication
 - Phagocytosis
 - Immune function
- Articular cartilage is avascular
- Diffusion through synovial fluid to chondrocytes provide
- cartilage nourishment
- Superficial cellular lining of synovium is composed of two synoviocytes:
 - Type A- vacuoles related to macrophages (phagocytic
 - Type B- secretory fibroblast, producing hyaluronan Together, Type A and Type B diminish joint friction
- Extracellular matrix contains lubricating molecules:
 - Collagen
 - Prosteoglycans
 - These are the semi-permeable membrane of synovial
- Rat study examined the impact moderate physical activity had on synovium:
 - Pro-inflammatory molecules (IL-14, IL-10, TNF-alpha)
 - OA related enzymes (MMP-13)
 - Anti-inflammatory cytokines (IL-4, IL-10) Chondroprotective markers (lubricin)
- "Exercise therapy may decrease cytokines and related genes expression and inhibit inflammatory factors-mediated cartilage degradation, through the synthesis of IL-10 by synoviocytes type A, thus, effectively blocking cartilage damage" (Castrogiovanni,

Importance of Vitamin D

- (One randomized controlled pilot trial suggested Vitamin D decreased pain in patients with low levels)
- Vitamin D impact on prevention and treatment:
 - Increases bone mass
 - Prevents bone loss

etal, 2019, p.9)

- Obtained from
- Diet (fish, mushrooms, eggs, liver)
- Ultraviolet light
- Supplement 1000 International Units (IU)/d over 60 years age
- Increases calcium absorption through the endocrine pathway
- Metabolized in the liver
- Vitamin D-cellular level:
 - TGF-beta binds to its receptor and phosphorylates SMAD3 and suppresses MMP13 expression
 - Vitamin D may be involved with TGF-beta/SMAD pathways
 - Cartilage damage and early osteophyte development are associated with lack of TGF-beta in mice who develop OA

In vitro studies suggest vitamin D may inhibit the progression of OA in humans by interacting with TGF-beta, however more studies are indicated (Parks, 2019)

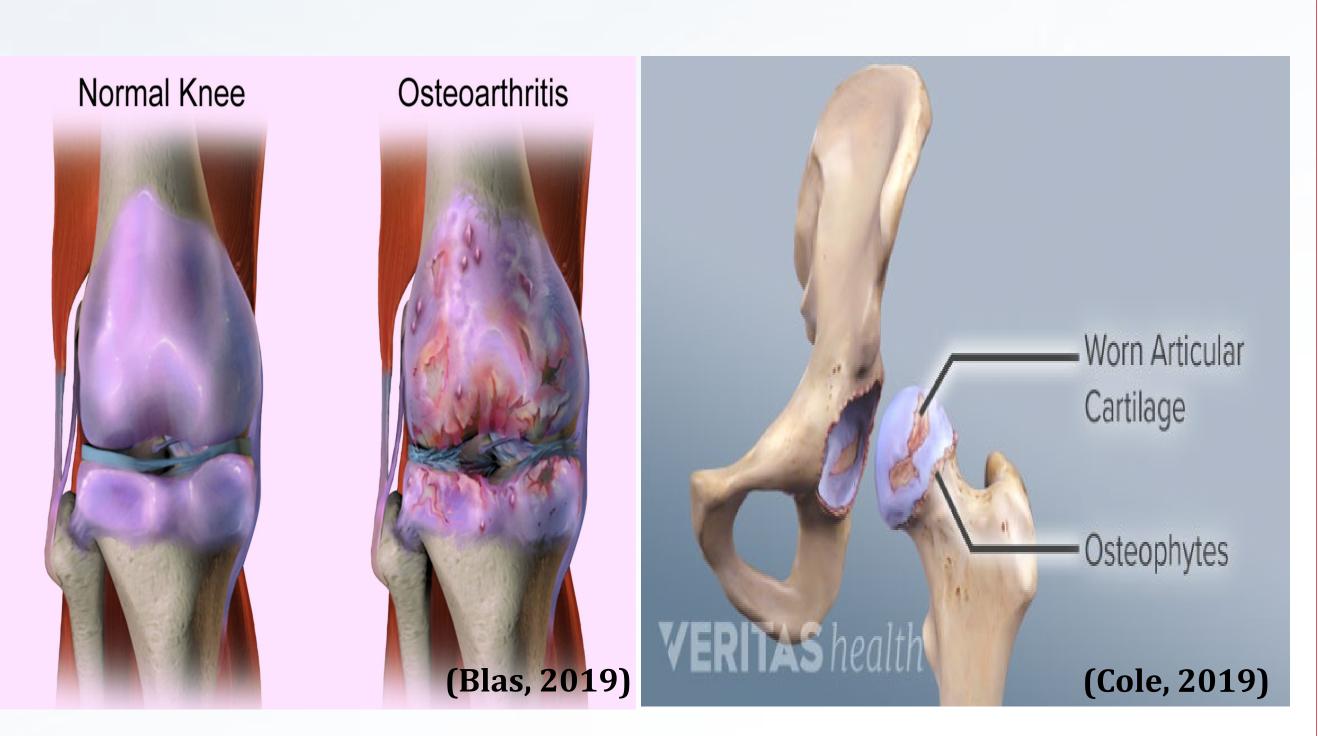
Introduction

Osteoarthritis (OA) is the primary cause of chronic pain, affecting approximately Twenty-seven million people in the United States. OA is a disease of the synovial joint, mostly causing pain in hips, knees, hands and facet joints of the spine (Miller, R., Tran, P., Obeidat, A., Raghu, P., Ishihara, S., Miller, R. & Malfait, A., 2015). In humans, knees are the largest synovial joint. In early stages pain is described as "sharp" but chronically progresses into a "dull/achy" pain debilitated many, once active individuals. Treatment options consist of pharmacological and as well as surgical interventions. "OA is the principal diagnosis associated with total knee replacement", (Miller, 2015, p.318).

Human beings are living longer than ever before. This fact broadens the patient population of those affected by OA. Anybody can be affected from the progressive wear and tear and degeneration of joints over a life-span. Being older than 65, obese, and of African American descent puts those at greater risk for developing OA (Mora, J., Przkora, R. & Cruz-Almeida, Y., 2018).

Presentation of Case

A 62-year-old African American male presents to the office complaining of progressively worse dull-achy pain in his left knee that is really stiff. He was physically active in his younger years but his exercise has slowly come to a halt. He has been diagnosed with type two diabetes mellitus and has a BMI of 30. He states that his knee pain inhibits him from exercise. An x-ray showed visible osteophytes. A joint aspiration and MRI indicate the After exhausting all traditional treatments, including NSAIDS and intra-articular corticoid injections, the pain still persists. Patient is instructed to lose some weight through dietary modifications and light exercise as tolerated. Surgical intervention will be the next treatment option. A primary left total knee arthroplasty has been scheduled



Treatment	OARSI	ACR	AAOS
Exercise (land and water based)	Appropriate	Strong recommendation	Strong recommendation
Transcutaneous electrical nerve stimulation (TENS)	Uncertain	Conditional recommendation	Inconclusive
Weight control	Appropriate	Strong recommendation	Moderate recommendation
Chondroitin or Glucosamine	Not appropriate for disease modification, Uncertain	Recommended against use	Recommended against use
Acetaminophen	Without comorbidities: appropriate	Conditional recommendation	Inconclusive
Duloxetine	Appropriate	No recommendation	No recommendation
Oral NSAIDs	Without comorbidities: appropriate With comorbidities: not appropriate	Conditional recommendation	Strong recommendation
Topical NSAIDs	Appropriate	Conditional recommendation	Strong recommendation
Opioids	Uncertain	No recommendation	Recommended only tramado
Intra-articular corticosteroids	Appropriate	Conditional recommendation	Inconclusive
Intra-articular viscosupplementation	Uncertain	No recommendation	Recommended against use

(Mora, Przkora, & Cruz-Almeida, 2018)

Abbreviations: OARSI, Osteoarthritis Research Society International; ACR, American College of Rheumatology; AAOS, American Academy of Orthopedic Surgeons; TENS, transcutaneous electrical nerve stimulation; NSAIDs, non steroidal antinflammatory drug.

Aerobic/endurance	Exercise modalities	Balance/proprioceptive	Stretching
	Resistance/strength training		
Include activities like walking,	Isometric, isotonic, isokinetic,	This includes modalities such as	This group will specifically
climbing stairs, and cycling. They	and dynamic modalities have been	Tai Chi, using slow and gentle	help with patient's range of
can decrease joint tenderness	studied. Most of them targeting	movements to adopt different	motion and flexibility.
while improving functional status	quadriceps, hip abductors,	weight baring postures while using	
and respiratory capacity. Cycling	hamstrings, and calf muscles. They	breathing techniques.	
is especially attractive to patients	improve strength, physical function,		
given the low impact profile.16,18	and pain levels, with similar efficacy		
One study showed a reduction of	and outcomes than aerobic		
10-12% on the physical disability	exercises.		
and the knee pain questionnaires. 16			

Implications for Nursing Care

- Risk factors:
 - Age
 - Prior joint injury
 - Genetics

Obesity

- Males African American
- Chief complaint-Pain
- Early onset- "sharp"
- Later stages- chronic- "dull/achy"
- Neuroanatomy of knee joint
- Sensory and sympathetic peripheral nerve fibers innervate
- Sensory cell bodies located in the dorsal root ganglion L3-
- Tissue injury/remodeling characteristics, molecules exiting nociceptors
- Classic molecules- prostaglandins, bradykinins Additional molecules-cytokines, chemokines
- Education
 - Diet (vitamins, glucosamine sulfate, phytochemicals)
 - Pain management
 - Weight management
 - Psychological (support groups, psychologist, meditation, holistic medicine)

Phytochemicals from food (reviewing 5879 articles, Five RCT's and Four cross-sectional

- Attributes found to decrease the inflammatory process, decreasing progression of OA
 - Antioxidants
 - Vitamin A
 - Vitamin (Vitamin E

Phytochemicals

- Fruits
- Vegetables Grains
- Beans Other plants
- physical activity

Conclusion

30 million adults in the U.S. are affected by OA. Prevention, delay of progression and symptom management are multifactorial and vary slightly amongst each individual. In general, maintaining a healthy diet, taking vitamins and low-impact regular exercise are big contributors to slowing the progress, decreasing the risk of OA. There are many innovative treatment options available to manage pain to optimize quality of life.

(Mora, Przkora, & Cruz-Almeida, 2018)

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