G. Sudhakara Rao et al



CODEN [USA]: IAJPBB

ISSN: 2349-7750

INDO AMERICAN JOURNAL OF PHARMACEUTICAL SCIENCES

http://doi.org/10.5281/zenodo.2589037

Available online at: <u>http://www.iajps.com</u>

A Case Study

A CASE REPORT ON STUDY OF CONSERVATIVE MANAGEMENT OF OSTEOARTHRITIS, A COMMON MUSCULO SKELETAL PROBLEM IN A COMMUNITY AT A TERTIARY CARE HOSPITAL

¹G. Sudhakara Rao*, ²G. Sandeep Kumar, ³Ch. Suman, ⁴V. Sowndarya, ⁵Y. Nagaprasanna ¹SIMS College of Pharmacy, Mangaldas Nagar, Guntur. A.P.

Article Received: January 2019	Accepted: February 2019	Published: March 2019
Abstract:		
It is an observational prospective study a	nd the study was planned and carried	l out for a period of six months and
150 II I All de	and the second	

152 cases were collected. All the patients were systematically interviewed, and their socio-demographic details were noted. In this study, we have collected 152 cases of osteoarthritis. Among 152 cases observed males 36 (23.6%) females 116(76.3%). Out of which 22 were smokers and 132 were nonsmokers and DM were 29, Hypertension were 25 and thyroid were 23. And people between the age 41-50 are mostly affected with osteoarthritis and followed by 51-60 of age i.e. 38.8% and 26.3% respectively, 62.4% were prescribed with analgesics and 49% using NSAIDS, 81% were prescribed with calcium supplements, 55.7% were prescribed with antibiotics, and 49.8% were prescribed with steroids. Four main strategies for the conservative treatment of osteoarthritis exist that must be used before indicating surgical treatment are medical treatment, physical medicine, intra-articular injections and self-management education programs.

Corresponding author:

Dr. G. Sudhakar Rao

Associate Professor, SIMS college of Pharmacy,Guntur. Mobile: +91- 9642843077, E-mail: gerapatisudhakararao@gmail.com.



Please cite this article in press G. Sudhakar Rao et al., A Case Report On Study Of Conservative Management Of Osteoarthritis, A Common Musculo Skeletal Problem In A Community At A Tertiary Care Hospital., Indo Am. J. P. Sci, 2019; 06(03).

INTRODUCTION:

Osteoarthritis

Osteoarthritis (OA) is one of the most common causes of knee pain and it affects millions of people around the world. Musculoskeletal disorders have a major impact on the health of the aging population. It is one of the leading causes of disability in our nation along with heart diseases, cancer and diabetes. Regarding the "work" place younger people get osteoarthritis from joint injury. Osteoarthritis of knee is a complex multifactorial process primary care like injury and deformity being major of pain and disability thus it has a substantial impact on functioning and activities of daily living the estimated population prevalence being varying from 4-30% depending on age, gender and disease. The risk of osteoarthritis of knee is comparable in men and women up to about 50yrs of age however after 50 years of age the incidence and prevalence of osteoarthritis of the knee increase more rapidly in women than in. It usually presents with pain over the knee joint which aggravates on squatting, sitting cross legged and climbing stairs other symptoms such asslight pain, numbness, tingling and radiating pain are rare. The physical examination is marked by joint line tenderness, crepitus and painful range of movements. In severe cases fixed flexion, deformity, restricted range of movements with vargus or valgus deformity can be present. The pathological features are characterized by progressive cartilage destruction, subchondral cyst formation with sclerosis of the surrounding bone, osteophyte formation and capsular fibrosis. The histological appearance is characterized by increased cellularity, appearance of clusters of chondrocytes. In late stages there is a complete loss of cartilage in some areas exposing the bone.

Musculoskeletal disorders have a major impact on the health of the aging population. Osteoarthritis is the most common joint diseases, increasing in frequency and severity in all aging populations. OA occurs due to change in dynamic equilibrium between the break down and repair of joint tissues is over whelmed. Traditionally OA has been considering a disease of articular cartilage. Recently due to improvement in MRI imaging, there is an increase of understanding of the other tissues in the pathophysiology of OA.OA involves the entire joint organ, including the subchondral bone, menisci, ligaments, muscle, capsule and synovium.

Anatomy

The knee is the largest and most complex joint of the body actually consisting of three joint within a single synovial cavity. It is formed by fusion of lateral femorotibial, medial femorotibial, and femoral patellar joints.

Types

It is compound synovial joint, incorporating to condylar joints between the condyles of the femur and tibia and one saddle joint between the femur and patella.

Articularsurface

- The knee joint is formed by
- a) The condyles of the femur
- b) The condyles of the tibia
- c) The patella

The femur condyles articulate with the tibial condyles below and behind and with the patella.

Fibrous capsule

Fibrous capsule is very thin and is deficient anteriorly where it is replaced by the quadriceps femora's, the patella and the ligamentous patella. [1-5]

Bursae

Anterior

- 1. Sub cutaneous prepatellor bursa
- 2. Sub cutaneous infra patellar bursa
- 3. Deep infra patellar bursa

Lateral

- 1. A bursa deep to the lateral head of the gastrocnemius
- 2. A bursa between the fibular collateral ligament and biceps femora's
- 3. A bursa between the fibular collateral ligament and the tendon of the popliteus

Medial

- 1. A bursa deep to the head of the gastrocnemius
- 2. A bursa between the tibia collateral ligament and the tendons of Sartorius, gracillus and semi tendinosus.
- 3. A bursa between the tendons of semi membranous and the medial tibial condyle.

Menisci

Menisci are fibro cartilaginous discs that located over the tibial condyles. The medial meniscus is a C in shape the lateral meniscus it is thick the periphery and thin towards centrally. The shape of menisci increases the concavity of the tibial condyles in which the femoral condyles. Menisci place an important part in distributing weight bearing forces, in reducing friction between the joint segments and serving as shock absorber.

Knee joint capsule

The capsule of the knee joint is cylindrical shape and it encloses the entire femoral and tibial condyles. it is concave anteriorly it is having a large anterior recess for patella in which patella will articulates. Posteriorly,the capsule is attached proximally to the posterior margins of the femoral condyles and inter choncylar notch and distally to the posterior tibial condyles the capsule is reinforced posteriorly by a number of muscles. Anteriorly, the patella, the tendon of the quadriceps muscle superiorly, and the patella ligament inferiorly complete the anterior portion of the joint capsule. [6-10]

Osteoarthritis

Osteoarthritis is a slowly progressive, degenerative disease of synovial joint occurring late in life it is the second most frequently reported chronic condition after lower back ache.

Epidemiology

The prevalence of OA increases with age. Generally, OA is uncommon under the age of 35years with 0.1% of people affected between the ages of 25-34years, but 80% of people affected above the age of 55 years. It occurs in all the population irrespective of race, climate or geographical location. Obesity is the strongest modifiable risk factor and has been shown to particularly affect the knees. Trauma or injury due to diseases, such as rheumatoid arthritis, will predispose a joint to developing osteoarthritis. A strongest genetic component is thought to be present; an inherited defect in type II collagen genes is linked to the development of early-onset polyarticular osteoarthritis.

OA may develop in any joint, but most commonly affects the knees. In 2005, it was estimated that over 26 million people in the world had OA. The prevalence of OA however varies greatly depending on the age, sex and geographical area studied. The incidence of knee OA increases with age, and women have higher rates than men, especially after the age of 50 years. A leveling off or decline occurs at all joint sites around the age of 80 years.

PATHOPHYSIOLOGY:

Osteoarthritis is a degenerative joint disease that may cause cartilage loss and morphological damage to other joint tissues, more biochemical changes occur in the earliest stages of osteoarthritis progression. However, during onset of osteoarthritis, the collagen matrix becomes more disorganized and there is a decrease in proteoglycan content within cartilage. The breakdown of collagen fibers results in a net increase in water content. This increase occurs because there is an overall loss of proteoglycans (and thus a decreased osmotic pull), it is outweighed by a loss of collagen. Without the protective effects of the proteoglycans, the collagen fibers of the cartilage can become susceptible to degradation and thus exacerbate the degeneration. Inflammation of the synovium and the surrounding joint capsule can also occur, though often mild. This can happen as breakdown products from the cartilage are released into the synovial space, and the cells lining the joint

attempt to remove them. Other structures within the joint can also be affected. The ligaments within the ioint become thickened and fibrotic and the menisci can become damaged and wear away. Menisci can be completely absent by the time a person undergoes a joint replacement. New bone outgrowths, called "spurs" or osteophytes, can form on the margins of the joints, possibly in an attempt to improve the congruence of the articular cartilage surfaces in the absence of the menisci. The subchondral bone volume increases and becomes less mineralized (hypo mineralization). All these changes can cause problems functioning. The pain in an osteoarthritic joint has been related to thickened synovium and subchondral bone lesions. [11-14]

Symptoms

- **Pain.** Your joint may hurt during or after movement.
- **Tenderness.** Your joint may feel tender when you apply light pressure to it.
- **Stiffness.** Joint stiffness may be most noticeable when you wake up in the morning or after a period of inactivity.
- **Loss of flexibility.** You may not be able to move your joint through its full range of motion.
- **Grating sensation.** You may hear or feel a grating sensation when you use the joint.
- **Bone spurs.** These extra bits of bone, which feel like hard lumps, may form around the affected joint.¹⁵⁻¹⁷

Diagnosis

During the physical exam, checking for tenderness, swelling or redness, and for range of motion in the joint.

- X-rays. Cartilage doesn't show up on X-ray images, but cartilage loss is revealed by a narrowing of the space between the bones in your joint. An X-ray may also show bone spurs around a joint. Some people may have X-ray evidence of osteoarthritis before they experience any symptoms.
- Magnetic resonance imaging (MRI). An MRI uses radio waves and a strong magnetic field to produce detailed images of bone and soft tissues, including cartilage. An MRI isn't commonly needed to diagnose osteoarthritis but may help provide more information in complex cases Laboratory tests
- **Blood tests:** Although there is no blood test for osteoarthritis, certain tests like ESR & CRP may help rule out other causes of joint pain, such as rheumatoid arthritis.

• Joint fluid analysis. Your doctor may use a needle to draw fluid out of the affected joint. Examining and testing the fluid from your joint can determine if there's inflammation and if your pain is caused by gout or an infection.¹⁸⁻²⁰

Treatment

Currently, the process underlying osteoarthritis cannot be reversed, but symptoms can usually be effectively managed with lifestyle changes, physical and other therapies, medications, and surgery. Exercising and achieving a healthy weight are generally the most important ways totreat osteoarthritis. Osteoarthritis is a chronic (long-term) disease. There is no cure. but treatments are available to manage symptoms. Long-term management of the disease will include several factors:

- Managing symptoms, such as pain, stiffness and swelling
- Improving joint mobility and flexibility
- Maintaining a healthy weight
- Getting enough of exercise

(a) EARLY TREATMENT

It is aimed at symptomatic pain relief. The principles are to maintain movements and muscle strength, protect the joint from over load, relieve pain and modify daily activities

This is achieved by:

Physical Activity

- One of the most beneficial ways to manage OA is to get moving. While it may be hard to think of exercise when the joints hurt, moving is considered an important part of the treatment plan. Studies show that simple activities like walking around the neighborhood or taking a fun, easy exercise class can reduce pain and help maintain (or attain) a healthy weight.
- Strengthening exercises build muscles around OA-affected joints, easing the burden on those joints and reducing pain. Range-of-motion exercise helps maintain and improve joint flexibility and reduce stiffness. Aerobic exercise helps to improve stamina and energy levels and also help to reduce excess weight. Talk to a doctor before starting an exercise program. ²¹⁻²³
 (b) Intermediate Treatment

Tab 1. Madiantiana	aammanly ug	d for actor	anthritic
1 ab.1: Medications	commoniv use	ea for osteo	Darthritis

TVPICAL DOSACE
I II ICAL DOSAGE
650 to 1,000 mg four times per day
oso to 1,000 mg four times per duy
250 to 500 mg two times per day
250 to 500 mg two times per duy
1 200 mg per day
1,200 mg por <i>day</i>
150 to 200 mg two times per day
<u> </u>
200 mg per day
50 mg two to three times per day
400 to 600 mg three times per day

(i) Nonsteroidal anti-inflammatory drugs (NSAIDs)

Classification

A. Nonselective COX inhibitors (traditional NSAIDs)

1. Salicylates: Aspirin.

2. Propionic acid derivatives:Ibuprofen,Naproxen, Ketoprofen, Flurbiprofen.

3. Fenamate:Mephenamic acid.

4. Enolic acid derivatives: Piroxicam, Tenoxicam.

5. Acetic acid derivatives:Ketorolac,Indomethacin, Nabumetone.

6.

Pyrazolonederivatives:Phenylbutazone,Oxyphenbut
azone.B. Preferential COX-2 inhibitors
Nimesulide, Diclofenac, Aceclofenac,Meloxicam,
Etodolac.C. Selective COX-2 inhibitors
Celecoxib, Etoricoxib, Parecoxib.D.Analgesic-antipyretics with poor
antiinflammatory action
1.Paraaminophenolderivative:
Paracetamol(Acetaminophen).

Metamizol

2.Pyrazolonederivatives: (Dipvrone).Propiphenazone.

3. Benzoxazocinederivative:Nefopam.

Aspirin

Aspirin is acetylsalicylic acid. It is rapidly converted in the body to salicylic acid which is responsible for most of the actions. Other actions are the result of acetylation of certain macromolecules including COX. It is one of the oldest analgesic- antiinflammatory drugs and is still frequently used.

Mechanism of action

Analgesia PGs induce hyperalgesia by affecting the transuding property of free nerve endings so that stimuli that normally do not elicit pain are able to do so. NSAIDs do not affect the tenderness induced by direct application of PGs, but block the pain sensitizing mechanism induced by bradykinin, $TNF\alpha$, interleukins (ILs) and other analgesic substances primarily by inhibiting COX-2. This constitutes the peripheral component of the analgesic action of NSAIDs. They are, therefore, more effective against associated inflammation pain. Lately the centralcomponent of analgesic action of NSAIDs has also been shown to involve inhibition of PG synthesis in the spinal dorsal horn neurones as well as in brain.

Pharmacokinetics

Aspirin is absorbed from the stomach and small intestines. Its poor water solubility is the limiting factor in absorption micro fining the drug-particles and inclusion of an alkali (solubility is more at higher pH) enhances absorption. However, higher pH also favours ionization, thus decreasing the diffusible form.

Aspirin is rapidly deacetylatein the gut wall, liver, plasma and other tissues to release salicylic acid which is the major circulating and active form. It is ~80% bound to plasma proteins and has a volume of distribution ~0.17 L/kg. Entry into brain is slow, but aspirin freely crosses placenta. Both aspirin and salicylic acid are conjugated in liver with glycine to form salicyluric acid (major pathway). They are also conjugated with glucuronic acid. Few other minor metabolites are also produced. The metabolites are excreted by glomerular filtration and tubular secretion. Normally, only 1/10th is excreted as free salicylic acid, but this can be increased by alkalinisation.

The plasma $t\frac{1}{2}$ of aspirin as such is 15–20 min, but taken together with that of released salicylic acid, it is 3–5 hours. However, metabolic processes get saturated over the therapeutic range; $t\frac{1}{2}$ of anti-

inflammatory doses may be 8–12 hours while that during poisoning may be as high as 30 hours. Thus, elimination is dose dependent.

Adverse effects

Nausea, vomiting, epigastric distress, increased occult blood loss in stools.Rashes, fixed drug eruption, urticaria, rhinorrhoea, angioedema, asthma and anaphylactic reaction.

Interactions

1. Aspirin displaces warfarin, naproxen, sulfonylureas, phenytoin and methotrexate from binding sites on plasma proteins: toxicity of these drugs may occur. Its antiplatelet action increases the risk of bleeding in patients on oral anticoagulants.

2. Aspirin at analgesic doses inhibits tubular secretion of uric acid and antagonizes uricosuricaction of probenecid. Tubular secretion of methotrexate is also interfered.²⁴⁻²⁶

Oxaceprol

Oxaceprol is an anti-inflammatory drug used in the treatment of osteoarthritis. It is derived from L-proline, a DNA-encoded amino acid. The active effect oxaceprol is to inhibit the adhesion and migration of white blood cells.

Mechanism of action

Oxaceprol (N-acetyl-L-hydroxyproline) is an amino acid derivative. It reduces leucocytes extravasation as well as the adhesion of leucocytes to capillaries. It prevents leucocyte infiltration into the joints, thus inhibiting an early step of inflammatory cascade and presenting a novel class of anti-inflammatory agents.²⁷⁻³⁰

METHODOLOGY:

1. Study Site

The study entitled "To study the conservative management of osteoarthritis and a common musculoskeletal problem in a community at a tertiary care hospital" was carried out in outpatient department of tertiary care hospital of ANDHRA HOSPITALS, Vijayawada, Andhra Pradesh.

2. Study Method

It is an observational prospective study.

3. Study Duration

The study was planned and carried out for a period of six months from September 2017 to February 2018

4. Sample Size

A total of 152 cases including both males and females.

5. Source of Data

- All the relevant data required were collected from
- Patient's case notes

- Treatment charts
- X ray reports
- Interviewing patients or patient's care takers
- Any other relevant sources The study was conducted in the outpatient department of Andhra hospital, Vijayawada, Andhra Pradesh in India, for a period of 6 months.

All the patients were systematically interviewed and their socio-demographic details were noted

6. Study Criteria

a) Inclusion criteria

- Both genders
- Above 45 years of age
- Patients with one or more disease
- Patients who are physically fit to answer the questions
- Patients who gave consent for study

Exclusion criteria

- Patients with co-morbid illness
- Patient with other illicit drug use other than alcohol
- Patient who were in pain state
- Patients who require attention for medical problems
- Patients without reliable information

7. Study Design

i) Collection of data

The data were collected and recorded in a specially designed data entry format. Prior to data collection, written consent from the patient/bystander was obtained on a patient consentform. Patient/bystander were also well informed about the study, its objective etc. through a patient consent form after informing about the current study through patient information form.

Hospital Name:			CASE NO:	
Patient Name:	Age:	Weight:	BMI:	
Gender: Male/Female				
Name of the consultant:				
Chief complaints:				
Past medical history:				
Social history:				
Pain scoring:				
Extremely severe (gre	ater than or equal	to 14 points),		
Very severe (11 to 13	points), Severe (8	to 10 points),		
Moderate (5 to 7 po	oints),			
Mild involvement (1 to 4 point	ts).			
Type of pain:				
Shooting	Throbbing	S	welling	
Numbness	Stiffness	C	Others	
Exercise:				
Do you exercise regularly?				
Yes		No		

Tab.2: Data collection form assessing the management of osteoarthritis

Prescription:

Data collector name and signature:

ii) Data Analysis

variables a resultant data was represented in bar graphs and The obtained data during the ward rounds were thoroughlypie charts.

analysed to evaluate inappropriateness in drug using. Data

analysed also included the results on patient's demographicsRESULTS AND DISCUSSION:

[age, gender, co-morbidities, poly pharmacy, social history, In this study, we have collected 152 cases of osteoarthritis. etc.] by using appropriate statistical stools. The appropriate treatment and conservative management for osteoarthritis are observed.

8. Statistical Analysis

1. Quantitative Analysis

This study was analyzed using Microsoft Excel. Frequencies and percentages were calculated and tabulated for categorical

Tab3: Sample

Category	Number
Non smokers	132
Smokers	22
Diabetes mellitus	29
Hypertension	25
Thyroid	23
Physiotherapy	102
Surgery	08
Total patients initially considered	152

Tab.3 shows the total sample of the study, out of the total 150 patients, initially considered for the study, were excluded based on the fixed criteria. He final study sample was of which 22 were smokers and 132 were nonsmokers, DM were 29, Hypertension patients were 25 and thyroid patients were 23.

Gender wise distribution

Among 152 cases observed for the study; as shown in Tab.4 and figure 4, 23.6% were males and 76.3% were females.

Tab.4: Gender wise distribution of patients

GENDER	FREQUENCY	PERCENTAGE
Male	36	23.6%
Female	116	76.3%



AGE WISE DISTRIBUTION:

Among 152 cases observed they are divided according to their age in Tab.5 and Fig.5 people between the age 41-50 are mostly affected with osteoarthritis and followed by 51-60 of age i.e. 38.8% and 26.3% respectively.

AGE IN YEAR	NO.OF PATIENTS	PERCENTAGE
31-40	9	5.9
41-50	59	38.8
51-60	40	26.3
61-70	26	17.1
71-80	15	9.8
81-90	3	1.9

Tab.5: Age wise distribution



Smokers and Non-Smokers

Among 152 cases of osteoarthritis, Tab.6, and Fig.6, differentiates the smoker and non-smokers and relates the risk factor of smoking in osteoarthritis. Among 152 cases 14.7% are smokers and 85.5% are non-smokers.

Tab.6: Relation between Smokers and Nonsmokers

Variable	
Smokers	Non smokers



Co-Morbidities

Among 152 cases of osteoarthritis, Tab.7 and Fig.7, differentiates the diabetes, hypertension and thyroid. Among 152 cases 19% are diabetes, 16.4% are hypertension and 15.1% are thyroid.

Tab.7: Patients with co-morbidities

CO-MORBITIES	FREQUENCY	PERCENTAGE
Diabetes	29	19.0%
Hypertension	25	16.4%
Thyroid	23	15.1%



NSAIDS & ANALGESICS:

Among 152 cases of osteoarthritis, Tab.8 and Fig.8, differentiates the use of NSAIDS and Analgesics in osteoarthritis compared with other drugs tramadol, aceclofenac and piroxicam are prescribed more.

S.NO	NAME OF THE DRUG	NO.OF PATIENTS	PERCENTAGE-%
1.	Tramadol	35	23
2.	Aceclofenac	30	19.7
3.	Paracetamol	30	19.7
4.	Piroxicam	22	14.4
5.	Mefenamic acid	18	11.8
6.	Diclofenac	11	7.2
7.	Naproxen	10	6.5
8.	Thiocolchicoside	8	5.2
9.	Indomethacin	6	3.9

Tab.8: NSAIDS and Analgesics

PROTON PUMP INHIBITORS & CALCIUM SUPPLEMENTS:

Among 152 cases of osteoarthritis, Tab.9 and Fig.9, differentiates the proton pump inhibitors and calcium supplements. Among 152 cases 15 patients were prescribed with rabeprazole, 53 patients were prescribed with pantoprazole and 124 were prescribed with calcium supplements.

NAME OF THE DRUG	NO.OF PATIENTS	PERCENTAGE
Rabeprazole	15	9.8
pantoprazole	53	34.8
Calcium	124	81.5

Tab.9: Proton Pump Inhibitors & Calcium Supplements

ANTIBIOTICS & STEROIDS:

Among 152 cases of osteoarthritis, Tab.10 and Fig.10, differentiates the use of Antibiotics and steroids in osteoarthritis. Among 152 cases cephalexin, levofloxacin, methyl prednisolone and deflazacort were used with a percentage of 28.2%, 15.1%, 28.2% and 11.8% respectively.

Tab.10: Antibiotics & Steroids

NAME OF THE DRUG	NO.OF PATIENTS	PERCENTAGE
Cephalexin	43	28.2
Levofloxacin	23	15.1
Fusidic acid	18	11.8
Methyl prednisolone	43	28.2
Deflazacort	18	11.8
Methotrexate	15	9.8

The current study shows that the osteoarthritis is a common degenerative joint disease. In this study we have considered a total of 152 cases of osteoarthritis patients out of which 36 (23.6%) were males and 116 (76.3%) were females. We have also considered the smokers and co-morbidities conditions like hypertension, diabetes and thyroid of 16.4%, 19.0% &15.1%. The conservative management of osteoarthritis we have considered physiotherapy, medicinal treatment, intra-articular injections and surgery. In medicinal treatment we have considered anti-inflammatory, NSAIDS. Analgesics, antibiotics, calcium supplements and corticosteroids. In this study the use of tramadol, aceclofenac, piroxicam, thiocolchicoside and cephalexin has greater importance in the treatment of osteoarthritis. The use of intra articular injections like methyl prednisolone and methotrexate are used in the case of chronic osteoarthritis.Intra-articular injections of corticosteroids relieve pain for weeks, while intra-articular injections of hyaluronic acid (viscosupplementation) relieve pain for months. In this study among 152 cases tramadol(23%), aceclofenac(19.7%), piroxicam(14.4%), thiocolchicoside(5.2%), cephalexin(28.2%),methylprednisolone(28.2%) and methotrexate(9.8%) were used. Out of 152 cases 102(67.1%) patients have done physiotherapy along with medicinal treatment. Out of 152 cases 8(5.2%) patients have gone to surgery along with medicinal treatment. The use of medicinal treatment ranks first in the conservative management of osteoarthritis followed by physiotherapy, intra articular injection and surgery.

CONCLUSION:

Osteoarthritis is one of the most common joint diseases that occurs world widely. Pain, tenderness, reduced range of movement is the major symptoms of osteoarthritis. Four main strategies for the conservative treatment of osteoarthritis exist that must be used before indicating surgical treatment: medical treatment, physical medicine, intra-articular injections and self-management education programs.

REFERENCES:

- Ayanniyi O, Duncan FA, Adeniyi AF. Leprosy: Knowledge and Attitudes of physiotherapists 4 Ayanniyi O, Dosumu OJ, Mbada CE. Pattern and Physiotherapy Management of Shoulder Pain: A 5-Year Retrospective Audit of a Nigerian Tertiary Hospital. Med Sci 2016;5:12-26.
- 2. Bollet AJ, Nance JL (July 1966). "Biochemical Findings in Normal andOsteoarthritic articular cartilage Invest. 45 (7): 1170-77.
- 3. Jump up to: a b Brocklehurst R, Bayliss MT, Maroudas A, Coysh HL, Freeman MA, Revell PA, Ali SY(January 1984). "The composition of normal and osteoarthritic articular cartilage from human knee joints. With special reference to unicompartmental replacement of and osteotomy the knee". J Bone Joint Surg Am. 66 (1): 95-106.
- Jump up Mankin HJ, Thrasher AZ (January 1975). "Water content and binding in normal and osteoarthritic human cartilage". J Bone Joint Surg Am. 57 (1): 76–80.
- Jump up to Venn M, Maroudas A (April 1977). & quot; Chemical composition and swelling of normal and osteoarthrotic femoral head cartilage. I. Chemical composition". Ann. Rheum. Dis. 36 (2):121-29.
- 6. Jump up Madry H, Luyten FP, Facchini A (2012). "Biological aspects of early

osteoarthritis". KneeSurg.SportsTraumato 1.Arthrosc. 20 (3): 407–22.

- Jump up Englund M, Roemer FW, Hayashi D, Crema MD, Guermazi A (2012). "Meniscuspathology,osteoarthritis and the treatment controversy". Nat. Rev. Rheumatol. 8 (7):412–19.
- Jump up Hill CL, Gale DG, Chaisson CE, Skinner K, Kazis L, Gale ME, Felson DT (2001). "Kneeeffusions, popliteal cysts, and synovial thickening: association with knee pain in osteoarthritis". J.Rheumatol. 28 (6): 1330–37.
- Jump up Felson DT, Chaisson CE, Hill CL, Totterman SM, Gale ME, Skinner KM, Kazis L, Gale DR(3 Apr 2001). "The association of bone marrow lesions with pain in knee osteoarthritis". Ann Intern Med. 134 (7): 541–49.
- Brosseau L, Rahman P, Toupin-April K, Poitras S, King J, Angelis GD, et al. A Systematic Critical Appraisal for Non-Pharmacological Management of Osteoarthritis Using the Appraisal of Guidelines Research and Evaluation II Instrument.PLoS One 2014; 9(1):829-86.
- Mohammed S, Bermejo JL, Souares A, Sauerborn R, Dong H.Assessing responsiveness of health care services within a health insurance scheme in Nigeria: users' perspectives. BMCHealthServ Res 2013;13(502):1-13.
- Ayanniyi O, Dosumu OJ, Mbada CE. Pattern and Physiotherapy Management of Shoulder Pain: A 5-Year Retrospective Audit of a Nigerian Tertiary Hospital. Med Sci 2016;5:12-26.
- Fernandes L, Hagen KB, Bijlsma JW, AndreassenO, Christensen P, Conaghan PG, et al. EULAR recommendations for the nonpharmacological core management of hip and knee osteoarthritis. Ann Rheum Dis 2013;72:1125-35.
- Ayanniyi O, Duncan FA, Adeniyi AF. Leprosy: Knowledge and Attitudes of physiotherapists in Nigeria. Disabil, CBR Inclusive Dev 2013;24(1):41-55.
- Urbach D, Nebelung W, Weiler HT, Awiszus F. Bilateral deficit of voluntary quadriceps muscle activation after unilateral ACL tear. Med Sci Sports Exerc 1999;31:1691-6.
- 16. Li RC, Maffulli N, Hsu YC, Chan KM. Isokinetic strength of the quadriceps and hamstrings and functional ability of anterior cruciate deficient knees in recreational athletes. Br J Sports Med 1996;30:161-4.

- Chmielewski TL, Stackhouse S, Axe MJ, Snyder-Mackler L. A prospective analysis of incidence and severity of quadriceps inhibition in a consecutive sample of 100 patients with complete acute anterior cruciate ligament rupture. J Orthop Res 2004;22:925-30/.
- 18. Hopkins JT, Ingersoll CD.Arthrogenic muscle inhibition: a limiting factor in joint rehabilitation. J Sport Rehabil 2000;9: 135-59.
- 19. Palmieri RM, Weltman A, Edwards JE, Tom JA, Saliba EN, Mistry DJ, et al. Pre-synaptic modulation of quadriceps arthrogenic muscle inhibition. Knee Surg Sports TraumatolArthrosc 2005;13:370-6.
- 20. Mizner RL, Petterson SC, Stevens JE, Vandenborne K, SnyderMackler L. Early quadriceps strength loss after total knee arthroplasty.The contributions of muscle atrophy and failure of voluntary muscle activation. J Bone Joint Surg Am 2005;87: 1047-53.
- Angelozzi M, Madama M, Corsica C, Calvisi V, Properzi G, McCaw ST, et al. Rate of force development as an adjunctive outcome measure for return-to-sport decisions after anterior cruciate ligament reconstruction. J Orthop Sports PhysTher 2012;42:772-80.
- 22. Bryant AL, Clark RA, Pua YH. Morphology of hamstring torquetime curves following ACL injury and reconstruction: mechanisms and implications. J Orthop Res 2011;29:907e14. 13. Maffiuletti NA, Pensini M, Martin A. Activation of human plantar flexor muscles increases after electromyostimulation training. J ApplPhysiol 2002;92:1383-92.
- Sakamoto AC, Teixeira-Salmela LF, de Paula-Goulart FR, de MoraisFaria CD, Guimara es CQ. Muscular activation patterns during active prone hip extension exercises. J ElectromyogrKinesiol 2009;19:105-12.
- 24. Barry BK, Warman GE, Carson RG.Agerelated differences in rapid muscle activation after rate of force development training of the elbow flexors.Exp Brain Res 2005;162: 122-32.
- 25. Bryant AL, Clark RA, Pua YH. Morphology of hamstring torquetime curves following ACL injury and reconstruction: mechanisms and implications. J Orthop Res 2011;29:907-14.
- 26. Pincivero DM, Campy RM, Salfetnikov Y, Bright A, Coelho AJ. Influence of contraction intensity, muscle, and gender on median frequency of the quadriceps femoris. J ApplPhysiol 2001;90:804-10.
- 27. Risberg MA, Holm I. The long-term effect of 2 postoperative rehabilitation programs after anterior cruciate ligament reconstruction: a

randomized controlled clinical trial with 2 years of follow-up. Am J Sports Med 2009;37:1958-66.

- 28. Marder RA, Raskind JR, Carroll M. Prospective evaluation of arthroscopically assisted anterior cruciate ligament reconstruction. Patellar tendon versus semitendinosus and gracilis tendons. Am J Sports Med 1991;19:478-84.
- 29. Roberts D, Fride'n T, Stomberg A, Lindstrand A, Moritz U. Bilateral proprioceptive defects in patients with a unilateral anterior cruciate ligament reconstruction: a comparison between patients and healthy individuals. J Orthop Res 2000;18: 565-71.
- Ihara H, Nakayama A. Dynamic joint control training for knee ligament injuries. Am J Sports Med 1986;14: 309-15