OSTEOARHRITIS OF THE KNEE
(GUIDELINE FOR TREATMENT)

Noverial
Fakultas Kedokteran, Universitas Andalas
Email : noverial@hotmail.com

ABSTRACT

As OA is associated with aging, the impact of this disease will continue to grow over the next decade. With no cure, physicians and patients alike attempt to control the pain, stiffness, and other adverse effects. Most of the treatments of OA address the symptoms rather than the cause of the disease. They have been targeted to decrease pain and inflammation while preserving independence and quality of life as much as possible. Presently OA has no cure, and physicians have little hope that a cure will be available shortly, treatment strategies focus on treating the disease symptoms with minimal side effects. Interestingly, physicians show a greater interest in more effective, safer drugs than a "super-drug" that would cure OA. OA is a major cause of pain and disability in the elderly. Pain caused by inflammation of bursae and cartilage, subchondral fracture, distention and instability of the capsule and cartilage, osteophyte formation, spasm of the muscle. Progression of this disease in the knee may take many years. Once established however, the joint may remain in a stable condition for many years. Triad at large; pain, disability and structural change.

***Key words** : osteoarthritis, knee, disease, structural change*

INTRODUCTION

Osteoarthritis (OA) is a degenerative joint disease characterized by a mixture of degradative and reparative processes resulting in cartilage erosion subchondral bone remodeling marginal osteophyte formation, synovial inflammation and capsular fibrosis (Fig. 1).[1,2,3]

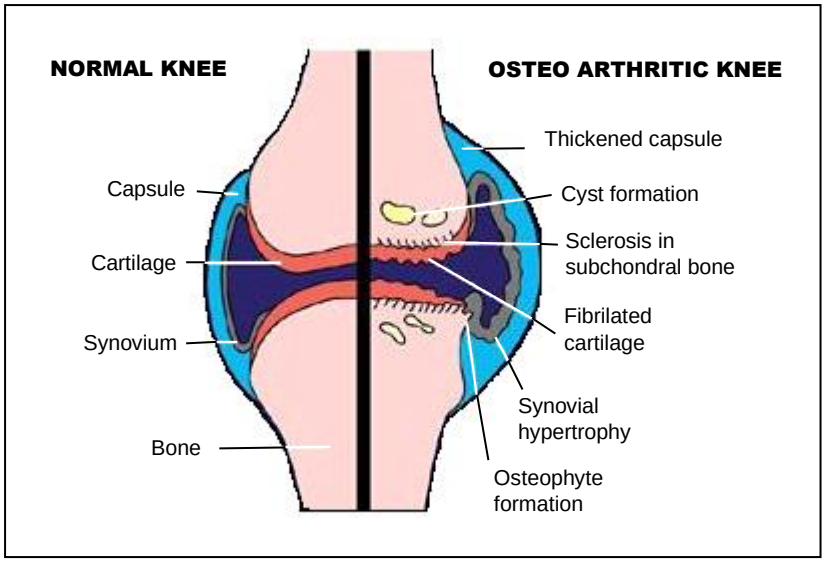


Fig 1. Normal knee vs OA knee

OA is not a single disease but rather the end result of a variety of disorders leading to the structural or functional failure of 1 or more joints. Osteoarthritis involves the entire joint including the nearby muscles, underlying bone, ligaments, joint lining (synovium), and the joint cover (capsule).[4]

Although the causes of OA of the knee are not always known, biomechanical stresses affecting the articular cartilage and subchondral bone and biochemical changes in the articular cartilage and synovial membrane are important in its pathogenesis. Current evidence suggest that both mechanical and biochemical factors play an important role in its progression. [5,6]

As OA is associated with aging, the impact of this disease will continue to grow over the next decade. With no cure, physicians and patients alike attempt to control the pain, stiffness, and other adverse effects. Most of the treatments of OA address the symptoms rather than the cause of the disease. They have been targeted to decrease pain and inflammation while preserving independence and quality of life as much as possible. Presently OA has no cure, and physicians have little hope that a cure will be available shortly, treatment strategies focus on treating the disease symptoms with minimal side effects. Interestingly, physicians show a greater interest in more effective, safer drugs than a “super-drug” that would cure OA..[5]

OA is a major cause of pain and disability in the elderly. Pain caused by inflamation of bursae and cartilage, subchondral fracture, distention and instability of the capsule and cartilage, osteophyte formation, spasm of the muscle. Progression of this disease in the knee may take many years. Once established however, the joint may remain in a stable condition for many years. Triad at large; **pain, disability, and structural change**. [7,8]

Herein, we present guidelines for the medical and surgical management of patients with OA of the knee. Differences in approach and treatment strategies which are unique to OA of the knee.

PATHOPHISIOLOGY

Although the etiology of OA is unknown, the pathology and pathogenesis of OA has been extensively studied. Researcher has shown that it appears to result from an imbalance between the synthesis and degradation of articular cartilage, its extracellular matrix, and subchondral bone resulting in loss of integrity. Cartilage is responsible for absorbing stress placed on a joint and providing a smooth, friction-reducing surface to ease joint movement. It is able to perform these roles because of its composition consisting of chondrocytes and a matrix of collagen and proteoglycan. Cartilage is maintained through a process of synthesis and degradation. In people with osteoarthritis, this process appears to be off balance leading to decreased production or increased degradation.[9,10]

The key characteristics of an OA joint are swelling, fibrillation, erosion and eventual loss of articular cartilage, together with remodeling of underlying bone resulting in subcondral sclerosis, bone cysts, an increase in metaphyseal bone and development of osteophytes (spurs). The end point of OA is eburnation, in which the focal loss of cartilage at the articulating surface of a bone reaches the stage where the underlying bone becomes exposed and subjected to increasingly localized overloading.[9,10]

The primary enzymes responsible for the degradation of cartilage are the matrix metalloproteinases (MMPs). These enzymes are secreted by both synovial cells and chondrocytes. IL-1 is a potent pro-inflammatory cytokine that, in vitro, is capable of inducing chondrocytes and synovial cells to synthesize MMPs. IL-1 may not only actively

promote cartilage degradation, but may also suppress attempts at repair, in OA. In addition to these effects, IL-1 induces nitric oxide production, chondrocyte apoptosis, and prostaglandin synthesis, which further contribute to cartilage deterioration.[9,10]

Matrix metalloproteinase enzymes, various cytokines, and nitric oxide have all been implicated in increased matrix degradation, since they are found in increased concentrations in osteoarthritic joints. Many compounds including growth factors influence cartilage synthesis. Insulin-like growth factor 1 and transforming growth factor beta may play a role in reduced production of cartilage, but their role is still unproven. In addition to this imbalance, other factors seem to play a part in the destruction of cartilage including metabolic, biomechanical, immunologic, and genetic influences. These changes result in cartilage that is fibrillated (as opposed to being smooth) and hypertrophied, inflamed in the underlying synovium, and has developed bony prominences known as osteophytes. Additionally, other surrounding structures may be involved. Subchondral bone may become brittle and develop cysts and microfractures, which can result in bone that is less efficient as a shock absorber.

MMPs and pro-inflammatory cytokines (e.g., IL-1) appear to be important mediators of cartilage destruction in OA. Synthesis and secretion of growth factors and of inhibitors of MMPs and cytokines are apparently inadequate to counteract these degradative forces. Progressive cartilage degradation and OA result.[9,10]

RISK FACTOR

SYSTEMIC RISK FACTORS

Age

Before age 45, osteoarthritis occurs more frequently in males (although it is not even common in younger adults). After age 55, it develops more often in females. In a 2000 study, 33% of women had osteoarthritis compared to 25% of men.

Ethnicity

Preliminary studies have shown conflicting evidence on the development of OA in African-Americans and in Caucasians, but differences in rates and radiographic features of the disease have been observed.[9]

Estrogen deficiency

Current evidence suggests that estrogen may have a protective effect on the development of OA, with an inverse relationship between OA and osteoporosis. Women with high lifetime exposure to endogenous (produced internally as opposed to exogenous or taken as Knee and OA Hip.[9]

Nutritional factors

Evidence indicates that continuous exposure to oxidants contributes to the development of many common age-related diseases, including OA. The intake of micronutrient antioxidants could be postulated to protect against OA. Relationships with intake levels of Vitamin C and D have also been found.[8]

Genetics

OA in all its heterogeneous forms appears to be strongly genetically determined. Candidate genes for common forms of OA include the vitamin D receptor gene, the insulin-like growth factor genes, the cartilage oligomeric protein genes, and the HLA region.[8]

Biochemical markers of cartilage or bone metabolism.

Bone-derived collagen cross-links in urine studies have demonstrated that candidate markers for cartilage turnover can be detected, and thus help identify persons at high risk for disease occurrence and progression.

LOCAL BIOMECHANICAL RISK FACTORS[7,8,9,11]

Obesity

Recent US studies have shown that being overweight pre-dates the development of disease and increases the risk for radiographic progression. Studies suggest that for OA Knee, this risk is greater for women than for men.

Mechanical environment of the joint

Alterations in the mechanical environment of the joint adversely affect load distribution.

Knee laxity (displacement or rotation of the tibia with respect to the femur)

Studies suggest that a portion of the increased laxity evident in an affected knee precedes the development of OA and may predispose to the disease.

Proprioception (the conscious and unconscious perception of joint position and movement).

Patients with OA have less proprioceptive accuracy than age-matched controls, suggesting the deficit precedes disease development.

Knee alignment (knee position in reference to the hip and ankle)

Malalignment predicts worse surgical outcomes, but its role in the natural history of OA has been minimally considered.

Loading of articular cartilage

Reductions in cartilage stiffness, resulting in cartilage erosion, have been shown to have a linear correlation with increasing stages of OA.

Acute joint injury and joint deformity

Apparent risk factors for postraumatic OA include high body mass, high level of activity, residual joint instability or malalignment, and persistent articular surface incongruity.

Occupational factors

Repetitive tasks, overworking the joints, and fatiguing muscles that protect the joints increase the risk for OA in those joints.

Sports participation

Epidemiologic studies have demonstrated that participation in certain competitive sports increases the risk for OA. Sports activities that appear to increase the risk of OA include those that demand high-intensity, acute, direct joint impact as a result of contact with other participants, playing surfaces, or equipment (eg, football and soccer). Early diagnosis and effective treatment of joint injuries, with complete rehabilitation, should decrease the risk of OA.

Muscle weakness

Longitudinal studies suggest that quadriceps muscle weakness not only results from painful OA Knee, but also is itself a risk factor for structural damage to the joint.

DIAGNOSIS

Clinical Finding

Diagnosis of OA is generally based on clinical findings. Patients are usually complain of pain and stiffness in affected joint(s), which is exacerbated with activity and relieved by rest. Early morning stiffness, is typically less than 30 minutes.

Phisical Examination

Physical examination of the affected area may demonstrate tenderness, crepitating, bony or soft tissue swelling, muscle wasting, advanced; gross deformity, bony hypertrophy, subluxation, loss of motion. Synovial swelling occasionally occurs and can be found on examination. The physician should inspect the surrounding soft tissue and bursal areas as well, to exclude periarticular disease.[11]

Laboratorium

Laboratory tests help rule out arthritis due to infection, inflammatory disorders, or endocrine and metabolic disease. The erythrocyte sedimentation rate should be normal or only mildly elevated; any notable elevation should prompt a search for polymyalgia

rheumatica, an underlying malignancy, or chronic infection. Radiographic evidence of chondrocalcinosis should prompt investigation of serum levels of calcium, phosphorus, magnesium, and thyrotropin (TSH). Joint fluid is usually bland or shows mild inflammation, demonstrated by fluid that contains fewer than 200 white blood cells/microliter; crystals should be absent. Newer assays of cartilage are not diagnostically useful in OA.[12]

Radiology

The radiographic hallmarks of osteoarthritis include nonuniform joint space loss, osteophyte formation, cyst formation and subchondral sclerosis. The initial radiographs may not show all of the findings. At first, only minimal, nonuniform joint space narrowing may be present. The involved joint spaces have an asymmetric distribution. As the disease progresses, subluxations may occur and osteophytes may form. Subchondral cystic changes can occur. These cysts may or may not communicate with the joint space, can occur before cartilage loss and have a sclerotic border. Subchondral sclerosis or subchondral bone formation occurs as cartilage loss increases and appears as an area of increased density on the radiograph. In the advanced stage of the disease, a collapse of the joint may occur.[13]

When evaluating patiens with OA of knee, AP and lateral radiographs allow an adequate evaluation of the medial and lateral joint spaces. Roentgenogram should also include 45 degree PA view of the knee (Rosenberg view) and skyline view of patella. To adequately assess the joint spaces, the AP view should be obtain with patient in a standing position. The lateral view also allows evaluation of the patellofemoral joint; however, an additional view, can offer even more information about this joint space. occurs *(Figures 2c and 2d)*, and osteophytes are seen anteriorly and medially at the distal femur and proximal tibia, and posteriorly at the patella and the tibia.[13]

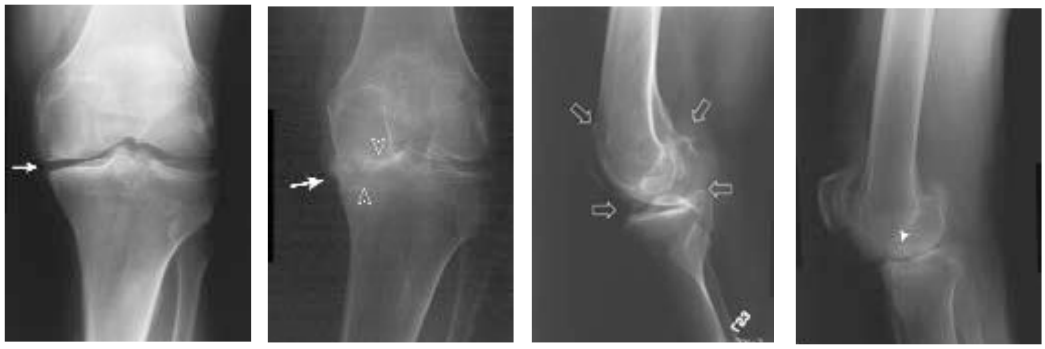


Fig 2. Osteoarthritis of the knees. *(A)* Anteroposterior view of the left knee of patient 1 shows medial joint space narrowing *(arrow). (B)* Lateral view of the left knee shows sclerosis with marked osteophyte formation *(arrows).* The osteophytes are best seen in this view. *(C)* Patient 2 has marked osteoarthritic changes with medial joint space narrowing *(white arrow)* causing a varus deformity of the knee and collapse of the joint space with destruction of the medial cartilage and the subchondral cortex *(open arrowheads)*. *(D)* Subchondral cysts *(solid arrowhead)* are noted.

CLASSIFICATION

System proposed for the classification of OA are base on radiographic criteria, clinical criteria or combination of both. Radiographic criteria propsed in 1957 by Kellgren and Lawrence remain the principal method for defining OA and were adopted by World Health Organization in 1961.[14]

Many studies tried to classify stage or grading of OA according to clinical staging, radiological grading, and arthroscopy grading.

Clinical staging divide into four stages:

Stages I minimal pain and swelling.

Stage II (mild) pain with extra activities.

Stage III (moderate) swelling loss of range of motion, pain with regular activities.
Stage IV(severe) swelling/warmth, loss of range of motion, pain at rest.

The first standardized method to determine radiographic knee OA was developed by Kellgren and Lawrence. This system was base on a global assessment combining several feature.

They divide into five grade in our modification:

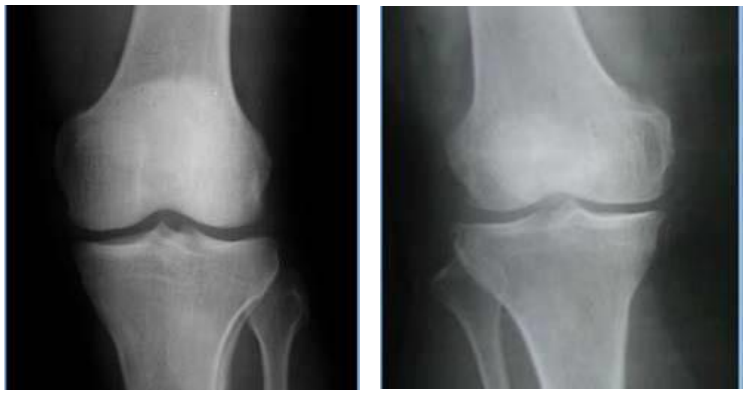
Grade 0 no radiological changes.

Grade 1 small osteophyte, no joint space narrowing.

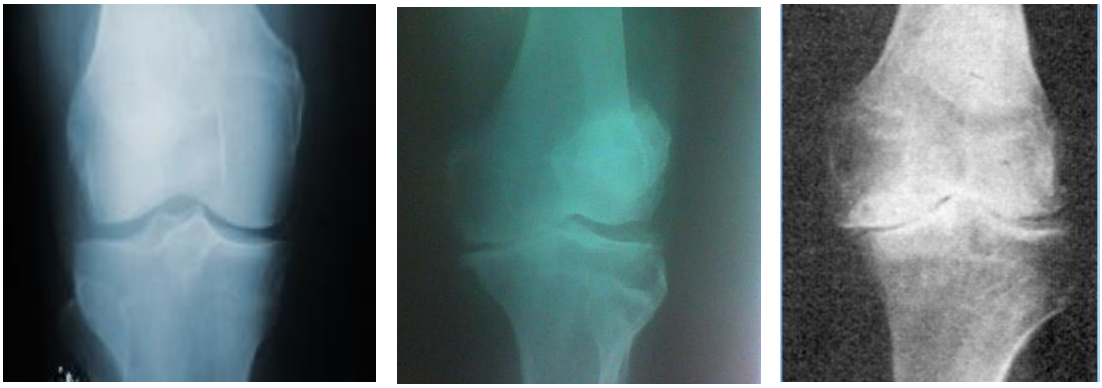
Grade 2 Osteophyte, subchondral sclerosis, cyst.

Grade 3 moderate to large osteophyte, unilateral joint space narrowing.

Grade 4 complete loss of joint space, obvious deformities, joint dislocation.



Grade 0 Grade 1



Grade II Grade III Grade IV

Fig 3. Kellgren and Lawrence Clasification (Modification)

Another radiological grading by Koshino divided OA into:

Grade 0 no abnormal finding.

Grade 1 subchondral cyst, sclerosis.

Grade 2 decrease joint space.

Grade 3 severe joint space narrowing,

Grade 4 complete loss of joint space,

Grade 5 joint dislocation.

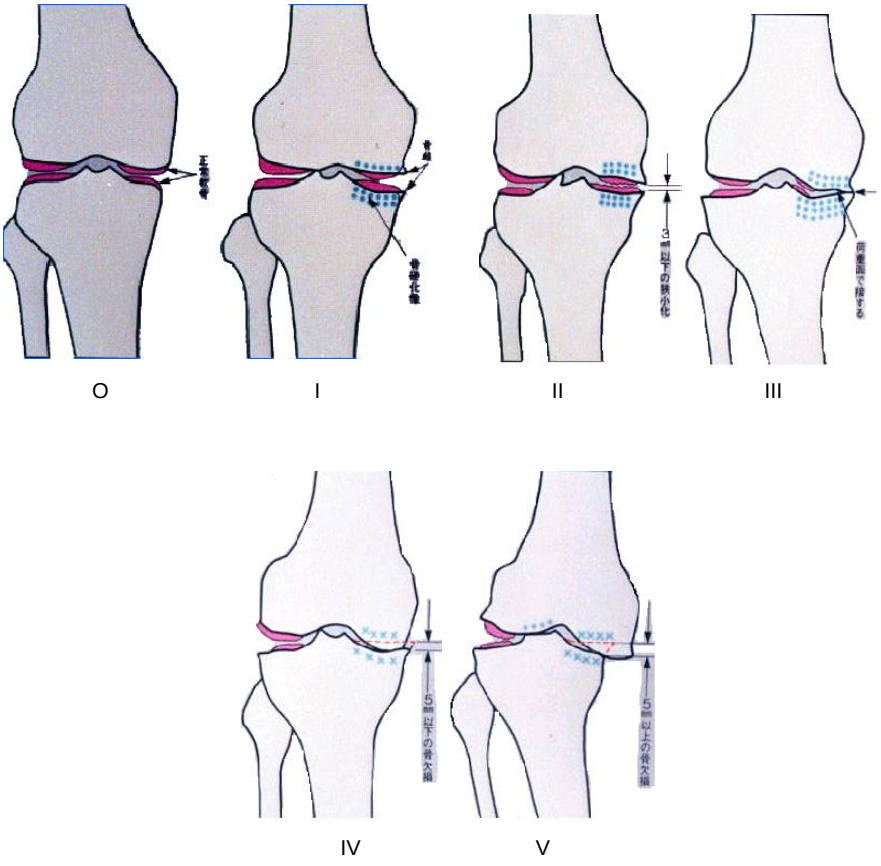


Fig 4. Classification by Koshino

The classification system used to determine eligibility for arthroscopy was designed by Jackson.[15] Arthroscopic grading divide into:

Stage 1 softening

Stage 2 fibrilation,

Stage 3 fragmentation

Stage 4 eburnation.

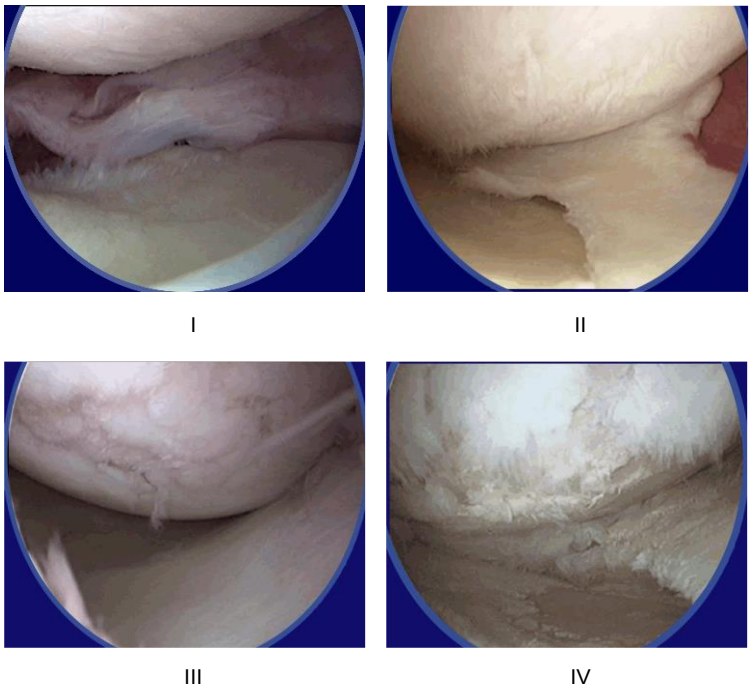


Fig 5. Arthroscopic Grading by Jackson

MANAGEMENT

Managements of OA are challenging. When planning the management for osteoarthritis of the knee, the patient's age, degree of functional disability, level of pain, and expectations for outcomes of the treatment need to be considered. A thorough history and a clinical examination focusing on determining levels and the location of pain and stiffness are essential. Of specific interest is the patient's assessments of levels of

pain and stiffness after prolonged periods of rest or sleep and an accurate physical assessment of loss of joint function, effusion, and scope of functional impairment.

Treatment choices should be individualized and patient centered, agreed on by the patient and doctor in a mutual discussion. Practice parameters/clinical practice guidelines are systematically developed statements, based on current professional knowledge, that assist practitioners and patients to make decisions about appropriate health care for specific clinical circumstances. Deviations from clinical practice guidelines may be justified by individual circumstances. As such, treatment guidelines should be used precisely as that guidelines. Decisions on individual patient treatment remains an art and not a science; therefore, treatment must be based on individual patient needs and professional judgment.

Considerations in choosing therapy:

- Individual and patient centered
- Severity of OA
- Professional knowledge and skill of the surgeon
- Facilities available
- Socioeconomic factor

The aims of therapy are provided adequate pain relief, improvement of joint mobility, minimize functional impairment.

Management divides into non pharmacologic therapy, pharmacologic therapy, and surgery.

NONPHARMACOLOGIC THERAPY

Nonpharmacologic therapy is aimed at reducing modifiable risk factors and educating patients about their disease and its management. These interventions include involving patients in support groups and self-help courses and referring patients to physical and occupational therapists to learn various exercises and mechanisms of joint protection and energy conservation.

Nonpharmacology include:

1. Patient education
2. Physical therapy
3. Occupational therapy
4. Weight lost

Patient education

Patient education appears to be effective in assisting patients with OA to manage their disease, both in terms of pain control and the necessity for medical visits. Patient who understands his/her illness is better able to manage pain and use medication. Material education such as literature audiocassette, computer, which explain the disease and it management, emphasizing the role of weight reduction and exercise.[7,8,9]

A meta-analysis of 10 trials that contrasted patient education whit the therapeutic effects of non-steroidal anti inflammatory drugs confirmed a significant beneficial effect of education on joint pain. The method was only around 20% as effective as non-steroidal anti-inflammatory drugs, but there was some evidence for a synergistic effect of both interventions.[8,9]

Self-management programs with health education such as the Arthritis Self-Management Program, which consists of 6 2-hour sessions, have been reported to decrease patient pain and the number of visits to physicians.[8]

Physical therapy

Physical therapy is a mainstay of the treatment of osteoarthritis. Two main approaches are used by physiotherapists: muscle strengthening programmes specific for certain joints and general aerobic conditioning. Specific exercise are important, e.g. quadriceps-strengthening exercises for knee osteoarthritis. Application of heat to the affected joint prior to exercise makes it more comfortable for the patient. Patients with osteoarthritis should avoid unsupervised weight bearing exercises as such activities may aggravate cartilage damage. Ideally, all newly diagnosed patient with osteoarthritis of the knee should be seen by a physiotherapist.[9,16]

Occupational therapy

Occupational therapists have an important role to play in advising patients on how to protect their joints from further damage. They can provide a range of devices that can be used in the home to assist with the activities of daily living, such as personal hygiene, dressing and household chores. An occupational therapist can provide assistive and ergonomic devices as well as instruction on joint protection techniques. Use of orthotic inserts, knee braces, and canes and walkers can improve gait and relieve pain.[8]

Although occupational therapy provides a means of educating patients and social support, there few evaluations of specific interventions such as the provision of walking aids, orthoses, and splints in controlled studies.

Weight loss

A study of 21 obese elderly men and women with knee osteoarthritis randomised to either a diet and exercise group or diet alone group found that the former group lost more weight but both groups had similar improvements in self reported disability, knee pain intensity, and frequency after six months.[7]

PHARMACOLOGIC THERAPY

Analgesics

Pain is the main reason why patients with osteoarthritis seek help from health care professionals. However, drug treatment is an adjunct, not a substitute for other types of treatment. As osteoarthritis has only a minor inflammatory component, paracetamol is now accepted as first-line therapy in uncomplicated osteoarthritis.[7]

The dose of paracetamol 1 g four times a day. It is safe and well tolerated, especially in older age groups. Paracetamol/opiate combinations such as coproxamol

may be used if paracetamol alone is unhelpful. Stronger opiates should be avoided if at all possible. Both the American College of Rheumatology and European League Against Rheumatism guidelines recommend this as initial therapy.[8]

Non-steroidal anti-inflammatory drugs

It has been suggested that the efficacy of NSAIDs in osteoarthritis relates to their action as analgesics and not as anti-inflammatory drugs. If it is necessary to use an NSAID to manage osteoarthritis, the following points should be borne in mind. As published studies have failed to identify a difference in the relative efficacy of different NSAIDs, individual choice should be based on relative safety, patient acceptability and cost. Therefore, ibuproven should be used first line because of its good safety profile and low costs.[17]

In patients with renal insufficiency, NSAIDs should be avoided whenever possible or used in very low doses if the benefits are expected to outweigh the risks. In such cases, serum creatinine, urea and electrolytes must be monitored regularly.[17]

Topical NSAIDs

Over the past few years there has been considerable debate about the safety and efficacy of topical NSAIDs. However, a recent systematic review of 86 trials involving over 10,000 patients showed that four topical NSAIDs (ketoprofen, felbinac, ibuprofen and piroxicam) were significantly more effective than placebo for pain relief and that this efficacy was not just related to a rubbing action. The most common side effects of topical NSAIDs are cutaneous reactions, such as urticaria, pruritus, irritation and contact dermatitis.[9,17]

As a clear role for topical NSAIDs in the treatment of osteoarthritis has yet to be defined, their routine use for this condition remains unjustified. However, they may be a safer alternative to oral NSAIDs in elderly patients who have an inflammatory component to their osteoarthritis. In the meantime, data are still needed to confirm their efficacy compared with simple analgesics, such as paracetamol, and topical rubifacients. If it is necessary to use a topical NSAID, then choice should be based on the cheapest available preparation.[7,8]

Intra-articular corticosteroids

Systemic corticosteroids have no role in the management of osteoarthritis. However, injections of intra-articular corticosteroids can be used successfully to reduce pain and relieve inflammation (synovitis) associated with acute flare-ups. They also have a place in treating patients awaiting surgery and in enabling patients with severe pain to participate more easily in an exercise programme. Corticosteroids may cause direct cartilage injury and accelerate cartilage loss and so repeated intra-articular injection is probably not justified. It is recommended that a joint should not be injected more frequently than every three months. The appropriate dose will vary with the joint involved and the corticosteroid used. For example, an intra-articular dose of methylprednisolone acetate ranges from 4 to 10mg for a small joint to 20 to 80mg for a large joint (such as the knee), depending on the volume of the effusion.[8,17]

SURGERY

Surgery is used where medical therapy has reached its limits. The purpose of surgical treatment is to reduce pain, increase function and improve symptoms overall.

Until some five decades ago arthrodesis (fusion of the joint) was the only surgical option. Although it relieved pain, the resulting stiff knee remained a functional disability. The 1950s and 1960s witnessed major developments in the surgical management of osteoarthritis of the knee. Surgical debridement, realignment osteotomy and prosthetic arthroplasty were introduced. [18]

Arthroscopy

Arthroscopy is a minimally invasive alternative to traditional surgical options such as osteotomy and arthroplasty. It's usually the first line of surgical treatment for OA knee.[5]

The use of arthroscopy in the treatment of the arthritic knee remains controversial. Patients should fully understand that arthroscopy, in this context, is at best a procedure to buy time and provide some pain relief. Moseley and associates shows no

long-term effects of the procedure, whether it is performed solely as an arthroscopic washout or is associated with debridement. However, if the patient's symptoms result from mechanical problems, such as loose bodies or meniscal tears, arthroscopy does play a role. The success and cost-effectiveness of meniscal repair for middle-aged patients is debatable: for most patients from this group, the treatment will be partial excision of meniscal tears and removal of cartilage debris. (Medscape Update on Surgical Management) [19,20]

Jackson was designed eligibility for arthroscopy management according to arthroscopic staging. Stage I patients have mild pain and swelling without mechanical symptoms and are treated conservatively. Stage II and III patients have pain, swelling, with mechanical symptoms often due to a meniscal tear. These patients are candidates for arthroscopic debridement, but as Jackson believe, “less is better” when performing debridement. Remove only the loose flaps of articular cartilage or flaps of meniscus. No procedure other than debridement when done in this study group. Stage IV is devided into early and late cases. The early cases with pain, swelling, joint space narrowing, fragmented articular cartilage, and small areas of exposed bone can still benefit with arthroscopic debridement. Late stage IV cases with extensive areas of exposed bone and malaignment due to the loss of articular cartilage were treated with realignment osteotomy or total joint arthropasty. [15]

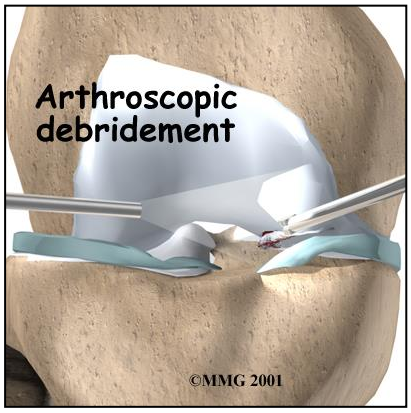


Fig. 6. Schematic of arthroscopy

Osteotomy

Osteotomy ("bone cutting") is a procedure in which a wedge of bone is removed from a damaged joint. Weight is shifted from an area where there is damaged cartilage to an area where there is more or healthier cartilage. In osteoarthritis, cartilage breakdown in the knee often is much greater in the inner part of the knee joint, often resulting in a bowlegged appearance.

The goal of tibial osteotomy is anatomic (tibiofemoral) alignment of 7° to 10° valgus or a mechanical axis passing through 30% to 40% of the lateral tibial plateau. Stable fixation, which allows early range of motion and weight bearing should be obtained. A midline incision, which can be used for subsequent conversion to TKA, is preferable. The procedure can be performed using either a closing lateral osteotomy, which is most common, or an opening medial osteotomy.

The indications for tibial osteotomy include the following:

- a young patient (less than 50 years of age) with a vigorous lifestyle or a job that involves heavy labor
- primarily medial compartment pain
- varus malalignment of the knee
- relative preservation of the lateral compartment and patellofemoral joint.

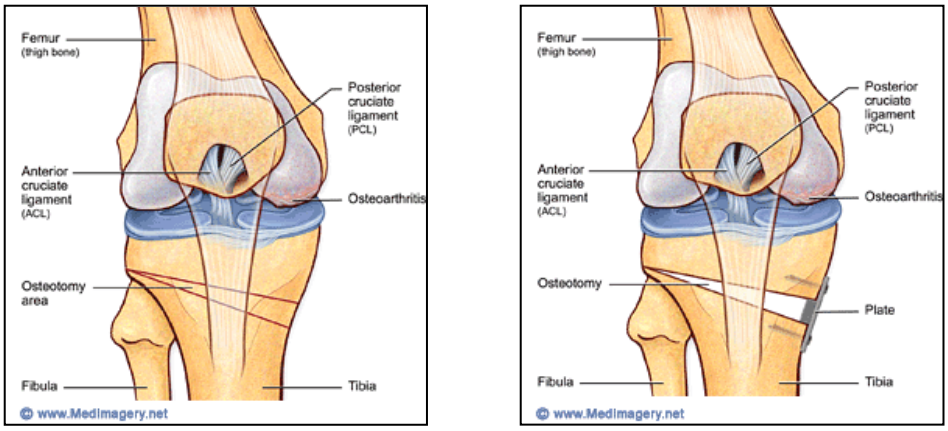


Fig 7. Schematic for osteotomy surgery

The procedure is contraindicated in patients with flexion contractures greater than 15° or suffering from inflammatory arthritis.[21]

This prosedure use to moderate to severe OA pain caused from the knee being out of alignment (Clinical stage III to IV). Principally indicated for unicompartmental arthritis and corresponding malalignment, or for symptomatic posttraumatic malunions about the knee associated with OA. Tibial osteotomy is an option for some patients who have a relatively small varus angulation (less than 10 degrees) and stable ligamentous support.

The use of high tibial osteotomy has decreased in recent years due to the success of TKA. However, studies evaluating high tibial osteotomy continue to demonstrate good outcomes. Koshino et all found after a mean duration of follow-up of 6.6 years, the medial opening-wedge osteotomy of the proximal part of the tibia provided satisfactory clinical results for patients with osteoarthritis of the knee. [22]

Total Knee Arthroplasty

Total knee arthroplasty is recommended for patients with more severe varus, or any valgus, deformity and ligamentous instability (Grade IV). It is also indicated for patients who have had ineffective pain relief following a tibial osteotomy. Total knee arthroplasty is an accepted surgical treatment for painful unicompartmental osteoarthritis of the knee in older patients, and the prevalence of total knee arthroplasty is increasing. Total knee arthroplasty has also performed well in younger patients. When patients of any age with unicompartmental osteoarthritis and with any diagnosis are poor candidates for other types of nonoperative and operative treatment, total knee arthroplasty is an option. To our knowledge, the results of total knee arthroplasty in patients with unicompartmental arthritis of the knee are no different than those in patients with bicompartmental or tricompartmental degenerative arthritis. Total knee arthroplasty is, of course, the final solution for many people, providing pain free and functioning joints for up to 20 years.

Giles R. Scuderi, MD, stressed that this procedure is not often used in active, middle-aged patients, owing to the potential for aseptic loosening, a result of wear debris generated during their active lives. Concern about the potential need for numerous revision operations in the course of a lifetime has kept practitioners from choosing TKA for patients other than those over the age of 60 years.

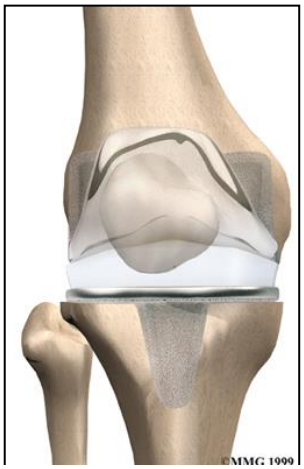


Fig 8. Schematic for total knee arthroplasty

NEW TECHNIQUE AND FUTURE TREATMENT OF OSTEOARTHRITIS

Presently OA has no cure, and physicians have little hope that a cure will be available shortly for therapies. Most therapy has been targeted to decrease pain and inflammation while preserving independence and quality of life as much as possible rather than modify the disease itself.

Development of disease-modifying therapy must be based on an understanding of the complex underlying biological processes, and this requires a wide range of competence that cannot easily be gathered into one research group or clinical discipline. Initiatives promoting collaborative efforts are therefore paramount.

The causes of osteoarthritis (OA) continue to elude biologists and translational clinical scientists; however, treatment of this very common disorder continues to be expanded.

Until recently, the only methods of repair of such surface damage to a joint have been by procedures such as drilling or microfracture of the subchondral bone or

abrasion arthroplasty. All these procedures aim to create bleeding in the joint that will produce a clot on the surface of the exposed bone. Under the stimulus of movement and load bearing, the clot will undergo metaplasia to fibrocartilage, the cells being derived from the bone marrow. However, this cartilage is deficient in type II collagen and normal proteoglycans and therefore functions for only a limited period.

Microfracture

With this procedure, puncture holes are made surgically in the bone beneath the damaged cartilage in order to create bleeding and clot formation. Once a clot fills in the area of cartilage defect, over time the clot tissue will be transformed into a type of scar cartilage (fibrocartilage). This tissue acts as a patch, like spackling an area of chipped paint. The new tissue does not have the mechanical integrity of normal cartilage, however, and will often degenerate in 3-5 years. After surgery weight bearing is avoided for 4-6 weeks to allow the clot to mature, and a passive motion machine is used 6 hours/day to stimulate healing. Full healing takes approximately 6 months.

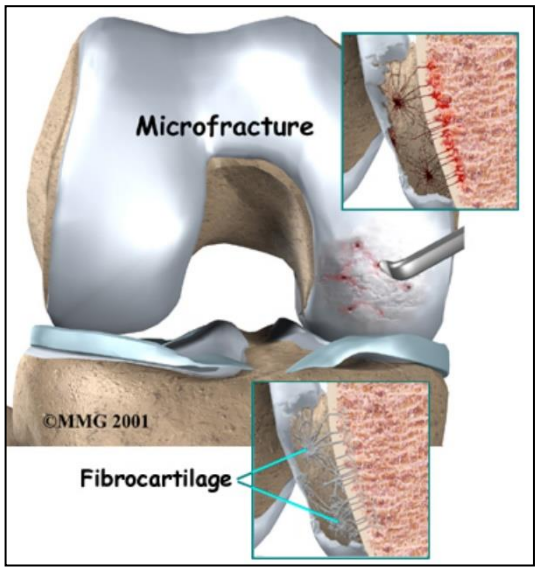


Fig. 9 Schematic of microfracture procedure

Mosaicplasty

Mosaicplasty for osteochondral grafting involves harvesting a small osteochondral graft, either from a non-weight-bearing donor area (superior medial margin of femoral notch) of the same knee joint (autograft) or using allografts, and then implanting the graft into areas of focal cartilage defect on the weight-bearing surface. However, the technique is only effective in knee joints with localized areas of cartilage loss, and is not usually used in joints with more generalized osteoarthritis. Result 86%-90% satisfied short term (Imhoff, Jacob, et all), 43% satisfied at 18 months (Gambardella)[5]

Osteochondral Autograft

This relatively new procedure replaces a localized area of damaged cartilage with a plug of cartilage and bone from a donor site in the same knee. Sort of like a hair transplant. This technique seems to be an option for small localized defects especially those smaller than 1 or 1.5 centimeters. Since this procedure is new there are still some unanswered questions about its long term effectiveness and about the possible detrimental effects on the donor site.

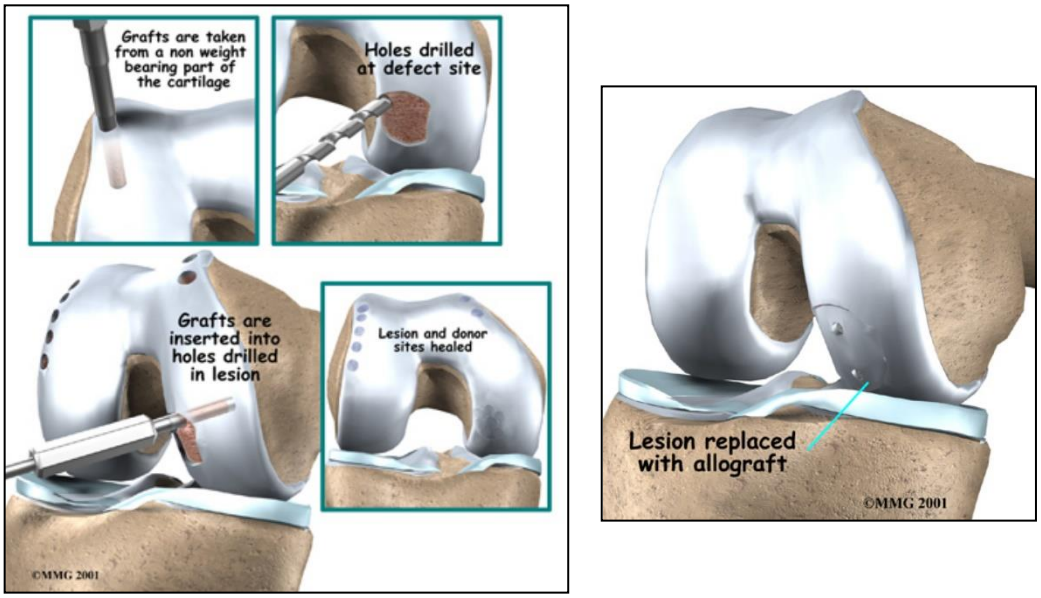


Fig 10. Schematic for Osteochondral Autograph

Chondrocyte Implantation

Dr Halbrecht was one of the first surgeons in the United States to perform the technique of chondrocyte implantation. This new technique for the treatment of cartilage surface damage is the ability to clone the patients own cartilage and reimplant the cells back into the joint. This technique involves taking a small sample of cartilage from the joint, extracting the cartilage cells and growing new cells in the laboratory using cell culture techniques. These cells are then reimplanted into the damaged area of the joint and kept in place with a patch of periostium, the skin-like lining of the adjacent bone. After surgery the leg is kept non weight bearing for 4-6 weeks and a continuous passive motion machine is used for 2 weeks to stimulate cartilage growth. Full recovery takes 6 months-1 year. Up to ten year data is currently available on this technique with 80-90% of patients achieving good-excellent results.

A new technique is currently under investigation in Norway that injects a polymer into the arthritic knee. The polymer then hardens slowly as it conforms to the contour of the joint and acts as a new spacer. Human trials have just begun and this technique is not approved for use in the United States at this time. It is not clear what will happen to this polymer over time or what the long term consequences may be. [23]

Several centers are working experimentally to develop a method for inserting cartilage cells into the joint embedded in a gel-like absorbable matrix. This is currently only being done in animal models.

An alternative source of cartilage cells in the future may well be human stem cells taken from the bone marrow of the patient, which could be modified by culture conditions to produce cartilage cells and matrix. This method has the theoretical advantage of providing unlimited numbers of cells and thus would avoid the need to harvest from the patient's joint, reducing the surgery required and the possible damage produced by harvesting. Xenografts derived from animal sources might also be used and could be stored in tissue banks for use when required.[26,27]

REFFERENCE

1. Takahashi K et al. The effects of hyaluron on matrix metalloproteinase-3, interleukin-1β, and tissue inhibitor of metalloproteinase-1 gene expression during the development of osteoarthritis, J OA Research Society International 1999;7:182-90.
2. Adams EM, Atkinson MH, Lussier AJ. The role of viscosuplementation with hylan G-F 20 in the treatment of osteoarthritis of the knee. Osteoarthritis and cartilage 1995;3:213-226.
3. Frizziero L, Govoni E, Bacchini P. Intraarticular hyaluronic acid in the treatment of osteoarthritis of the knee: clinical and morphological study. Clin and Exp Rheu 1998;16:441-449
4. Peat G, Thomas E, Handy J, Wood L. The Knee Clinical Assessment Study – CAS(K). A prospective study of knee pain and knee osteoarthritis in the general population. BMC 2004;5:4
5. Jackson RW. Defining the role of arthroscopy in the treatment of osteoarthritis. Medscape. 2002. http://www.medscape.com/viewprogram/264_pnt
6. Losada CJ, Altman RD. Current concepts on the management of osteoarthritis (oa). Cutting Edge Reports. Sponsored by Novartis. Available at http://www.rheuma21st.com
7. Hirsh JM, Lozada JC. Medical management of osteoarthritis. JCOM J Clin Outcomes Manage 2001;8(2):51-9.
8. Cheng SN. Medical management of osteoarthritis. JAOA 2002;15(1):6-10.
9. Haq I, Murphy E, Darce J. Osteoarthritis. Posgrad Med J. 2003;79:377-383.
10. Hinton R, Moody RL, Davis WA. Osteoarthritis: Diagnosis and Therapeutic Considerations. American Family Physician, 2002
11. Morehead K, Sack KE. Osteoarthritis: What therapies for this disease of many causes? Postgrad Med 2003;114(5):11-7
12.
13. Swagerty D, Hellinger D. Radiographic assessment of osteoarthritis. Am Fam Physician 2001;64:279-86
14. Sangha O. Epidemiology of rhematic diseases. Rheumatology 2000;39(suppl.2):3-12
15. Jackson RW, Dieterichs C. *Arthroscopy*. 2003;19(1):13-20 Available at: http://www.medscape.com/viewarticle/450692_print

16. Dieppe P. Osteoarthritis: time to shift the paradigm. BMJ 1999;318:1300-1
17. Wood J. Osteoarthritis and its management. The Phar J 1999;262(7046):744-746
18. Satku K. Unicompartmental Knee Arthroplasty: Is it a Step in the Right Direction? – Surgical Options for Osteoarthritis of the Knee. Singapore Med J 2003 Vol 44(11) : 554
19. Allgood P. Arthroscopy lavage for the knee osteoarthritis. Steer. 2003;3(3). Available at: http://www.wihrd.soton.ac.uk/projx/signpost/steers/STEER_2003(3).pdf
20. Moseley JB, O'Malley K, Petersen NJ, et al. A controlled trial of arthroscopic surgery for osteoarthritis of the knee. *N Engl J Med* 2002; 347: 81-88.
21. Barret WP. Alternatives to Total Knee Arthroplasty. Medscape. 2000. Available at: http://www.medscape.com/viewprogram/519_pnt
22. Koshino T, Murase T, Sayto T. Medial opening-wedge high tibial osteotomy with use of porous hydroxyapatite to treat medial compartment osteoarthritis of the knee. JBJS 2003;85(1):75-85.
23. Uthman I, Raynauld JP, Haraoui B. Intrarticular therapy in osteoarthritis. Postgrad Med J 2003;79:449-53.
24. Hochberg MC et al. Guidelines for the medical management of osteoarthritis. Journal American College Rheumatology. 1995vol 38(11):1541-1546
25. Grainger R, Cicuttini FM. Medical management of osteoarthritis of the knee and hip joints. MJA 2004; 180 (5):232-236
26. Heinegård D, Lidgren L, Saxne T. Recent developments and future research in the Bone and Joint Decade 2000 –2010. Bulletin of the World Health Organization 2003, 81
27. Halber JL. Osteoarthritis Injuries - The Future. Institute for Arthroscopy and Sports Medicine 2004. Availble at: http://www.iasm.com/future.html