
CLINICAL PRACTICE GUIDELINES
ON THE
MANAGEMENT OF OSTEOARTHRITIS

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Ministry of Health Malaysia



Malaysian Society of Rheumatology



Academy of Medicine of Malaysia

**Clinical Practice Guidelines on the
Management of Osteoarthritis**

2002

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FOREWORD

Osteoarthritis (OA) is a very common disease worldwide. Although it can affect people of all ages, its prevalence increases sharply in the elderly. The symptoms range from mild to severe and disability increases as OA progresses. Therefore it has important social and economic consequences.

OA is seen and managed by doctors in various disciplines, ranging from general practitioners to physicians and orthopaedic surgeons. Because of this, a consensus is needed to guide doctors in managing OA along evidence based principles.

In order to achieve this, we gathered a multidisciplinary team comprising medical and paramedical personnel from the public, academic and private sectors to contribute to this Clinical Practice Guidelines on the management of OA. I hope the result is an up to date, easily readable and comprehensive booklet which will be useful in the everyday practice of doctors and personnel involved in the management of OA.

I would like to thank everyone involved in the production of this CPG for making it possible.



Dr. Hj. Azmillah Rosman
Chairperson of
CPG on the management of OA

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1. INTRODUCTION AND OVERVIEW OF OSTEOARTHRITIS

Osteoarthritis (OA) is the commonest form of arthritis found worldwide. It is responsible for the largest burden of joint pain and is the single most important rheumatological cause of disability and handicap.

The term osteoarthritis was coined by Joluk Spender of England in 1886 as a preferable term for rheumatoid arthritis. It was first introduced to refer to the condition presently understood as OA and differentiated from rheumatoid arthritis by Archibald Garrod in 1907.

OA is currently understood to be a process rather than a disease which may be triggered by diverse constitutional and environmental factors.

The factors influencing the expression, prevalence and distribution of OA in populations are complex and interactive. Race, genetics, bodybuild, obesity, gender, occupational use, repetitive use and previous injury have all been shown to have an influence. Latitude and climate have no significant influence.

Age is the most powerful predictor of OA with the prevalence of OA rising steeply with advancing age at all joint sites. The estimated prevalence of symptomatic knee OA in populations above the age of 65 is 30%. Women are twice as likely to suffer from knee OA as men. The COPCORD study in Malaysia showed that 9.3% of adult Malaysians complained of knee pain with a sharp increase in pain rate to 23% in those over 55 years of age and 39% in those over 65 years.^{1,2}

The exact prevalence of OA is difficult to determine because of the lack of use of standardised criteria. In epidemiological studies OA is often described by radiological criteria, however radiological disease especially when mild, has poor correlation with the presence of pain.

In all populations studied so far the prevalence of knee OA is higher than that of hip OA but this is more marked in Asian populations.³ The joints commonly involved in OA are shown in Figure 1.

Pain is the most important presenting symptom of OA. The cause for pain is often unclear and is likely to vary in severity, location and precipitating cause between individuals. By the time a person seeks help for pain caused by OA the likelihood of disability related to squatting, climbing or walking is high.⁴

Although there is no known cure for OA, current treatments aimed at educating the patient, controlling pain, increasing fitness and strengthening surrounding muscles can improve joint mobility and limit functional impairment. Disease modifying therapies which limit the disease progression and encourage repair are being explored. When these modalities fail to limit pain and disability and OA disrupts the patient's life, joint surgery is an option.

Primary prevention by reduction of obesity and avoidance of undue trauma during sport and repetitive knee bending while carrying heavy loads at work are obvious strategies.

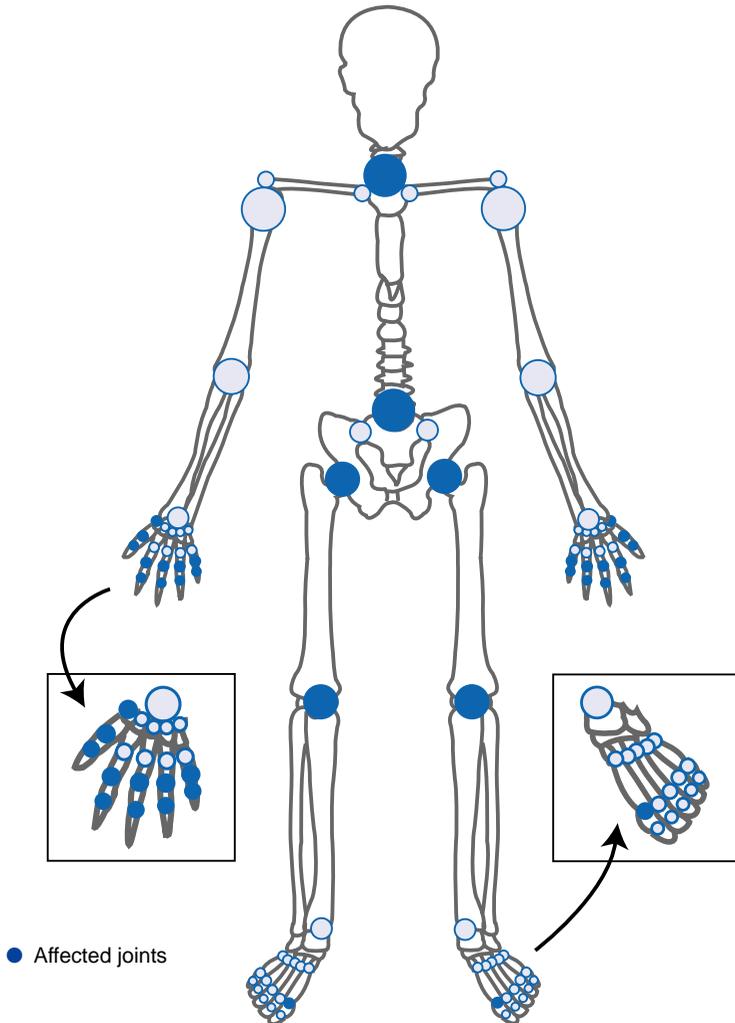


Figure 1 Joints commonly involved in OA

2. PATHOPHYSIOLOGY

OA can be best described as joint failure.⁵ The first change in OA is probably biomechanical stress that feeds back onto the cartilage surface and subchondral bone. This may lead to biochemical changes in the tissues. The moment an injury occurs, there is an attempt at joint repair. This may be an anti-inflammatory response with cellular infiltrate and a fibroblastic response with the formation of fibrocartilage. The repair process incorporates a bone response in the form of bony osteophytes. There may be a synovial effusion followed by some thickening of the synovium.

As the damage leads to further biomechanical disturbance, there may be muscle wasting due to a combination of disuse, effusion related neurogenic feedback and other mechanisms. Later on other definitive changes occur i.e. subchondral bony sclerosis, osteophytic proliferation and cartilage loss, all reflected in the classic X-ray appearance of joint space narrowing, subchondral sclerosis and osteophytes.

The synovial fluid usually comprises a macrophage infiltrate with some lymphocytes. It is not usual for the fluid to have a predominant polymorphonuclear response in the absence of concomitant disease like crystal shedding, inflammatory joint disease or sepsis.⁶



Figure 2 X-Ray of OA knees



Figure 3 OA knees with varus deformity

3. DIAGNOSIS

Classification

There are several different methods by which osteoarthritis can be classified. It can be classified by the joints involved and the localization within the joint. For example, in knee OA, there may be medial, lateral and/or patello-femoral involvement. It can also be classified by aetiology as shown below.⁷

Primary: Idiopathic

Primary OA includes generalised OA, a condition associated with Heberden's nodes and polyarticular disease, especially in the hand, with a female preponderance and a high prevalence in first degree relatives.^{8,9}

Secondary

- 1) Metabolic: e.g. acromegaly, haemochromatosis, chondrocalcinosis
- 2) Anatomic: e.g. slipped femoral epiphysis, Legg-Perthes disease, congenital dislocation of the hip, leg length inequality, hypermobility syndromes, avascular necrosis
- 3) Traumatic: e.g. major joint trauma, fracture through a joint or osteonecrosis, joint surgery
- 4) Inflammatory: e.g. rheumatoid arthritis, psoriatic arthropathy and septic arthritis.

Risk Factors

1. *Susceptibility Factors*

Advancing age

Obesity: in bilateral knee OA¹⁰

Heredity: especially generalized OA

Reproductive variables: female preponderance, postmenopausal state

Hypermobility

2. *Mechanical Factors*

Major injury: fracture, meniscal tear, cruciate ligament damage

Joint shape: e.g. Legg-Perthes disease, malalignment of biomechanical axis

Occupational: e.g. knee OA in manual worker, hip OA in farmers

Symptoms

Most people have pain during and after activity

Stiffness: “gelling” after inactivity, usually < 30 minutes

Loss of movement: difficulty with certain tasks, pain worse at the extremes of movement

Feelings of insecurity and instability of the joint

Functional limitations and handicap¹¹

Signs

Tenderness around the joint margins

Firm swellings around the joint margins (Herberden’s nodes / Bouchard’s nodes)

Crepitus on movement

Effusions may occasionally be present

Restricted, painful movement

Quadriceps muscle wasting (knee OA)

Deformity (varus deformity knee)

Instability

Investigations

Plain radiographs

Single view of the affected joint may be able to establish diagnosis and severity and also monitor disease progression. **Weight bearing films of the knee are required (AP view, standing)**. A lateral film of the knee will show patello-femoral OA. Additional single view of hands and feet may aid differential diagnosis.

Classical plain x-ray findings are: osteophytes, joint space narrowing, subchondral bone sclerosis, subchondral cysts and malalignment. Note: radiological findings do not always correlate with symptoms; patients with abnormal X-rays may be asymptomatic.

Blood investigations

When the diagnosis of OA is certain, blood tests are not necessary. If inflammatory markers (ESR, CRP) are checked, they are likely to be normal, or only mildly elevated.

Synovial fluid findings

Gross appearance	Clear
Viscosity	High
WCC/mm ³	200 – 10,000
% polymorphs	< 50%
Crystals	Negative

Diagnostic Criteria

The American College of Rheumatology Criteria for OA of the Hip and Knee

Hip:¹²

Pain in the hip (usually in the groin) for most days of the prior month



And 2 of the following:

ESR < 20 mm/hour

Radiographs of femoral and /or acetabular osteophytes

Radiographs of hip joint space narrowing (superior, axial, and/or medial)

Knee:¹³

Pain in the knee for most days of the prior month



And 1 of the following:

Over 50 years of age

Less than 30 minutes of morning stiffness

Crepitus on active movement and osteophytes

Pitfalls in the diagnosis

Patients with typical features of OA may be erroneously diagnosed as having

- Rheumatoid arthritis: because the rheumatoid factor (RF) is positive in low titre
- Systemic lupus erythematosus: because the anti-nuclear antibody (ANA) is positive in low titre
- Connective tissue disease: because the ESR is mildly elevated

The prevalence of RF and ANA positivity rises with age and the ESR also rises with age. Therefore, the diagnosis of these other conditions must be made on clinical grounds and not just on a blood test.

However, patients with OA can develop other rheumatic complaints such as gout, pseudogout, septic arthritis and soft tissue rheumatism such as bursitis, which have to be treated in addition to their OA.

4. MANAGEMENT

The management of OA involves a multidisciplinary approach with the aim to relieve symptoms and improve joint function. It involves non-pharmacological and pharmacological therapy. In certain cases, surgery is indicated.

PATIENT EDUCATION

Patients with OA should be informed of their diagnosis and the nature of the disease and its progression discussed. Patients who have an understanding of the disease and its natural history cope better and report less pain.^{14,15} The most important goal is to instill a positive attitude.

WEIGHT REDUCTION

Overweight patients should aim to lose weight. Weight loss decreases pain substantially in those with knee OA. Losing 5 kg of weight reduces the force on the knee by 15 - 30 kg with each step.¹⁶

PHYSIOTHERAPY

Physiotherapy should be started as soon as possible to improve joint mobility, increase muscle strength, reduce pain and prevent further disability.

All patients should participate in an exercise programme to mobilise the joints and strengthen the surrounding muscles.

(A) *Exercise Programme*

Exercise programmes should be individualised. A combination of exercises including range of movement (ROM), strengthening and low impact aerobic exercises are appropriate.

There are 2 types of exercise programmes :

- *Range of motion exercises and strengthening exercises*^{18,19}

Isometric exercises are recommended initially, followed by progressive resistance exercises and a combination of open and closed chain exercises. These exercises should be done daily. (See opposite page)

- *Aerobic programme*^{19,20}

Aerobic exercises that can be recommended include walking for 30 minutes 3 times per week, biking, swimming, aerobic dance and hydrotherapy.

Figure 4
 Quadriceps-strengthening Exercise

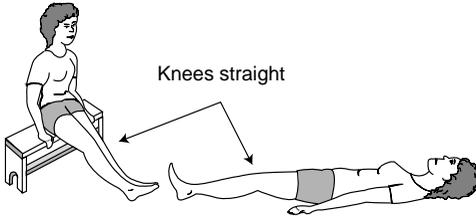


Figure A

Figure B

1. Sit on a firm surface (figure A) or lie flat in bed (figure B).
2. Perform this exercise in either of the following positions:
 - a) Sit in a chair (figure A) with your legs straight, heels on the floor or on a footstool. Squeeze your thigh muscles, pushing your knees downward the floor.
 - b) Lie in bed (figure B) with your legs straight and squeeze your thigh muscles, pushing the back of your knees into the bed.
3. Hold this position for a full 5 seconds. Use a clock or watch with a second hand, or count: one-one thousand, two-one thousand, three-one thousand, four-one thousand, five one-thousand.
4. Relax the muscles.
5. Begin your strengthening program with 10 repetitions, holding each contraction for a full 5 seconds. Perform this exercise 7 times daily and increase the number of repetitions you perform with each set by three to five daily during the first week.
6. By the end of the week, you should be able to perform 15 repetitions per set. This is the maximum number of repetitions you should perform in a set. (Total per day = 15 repetitions per set · 7 sets = 105)
7. If your arthritis is causing knee pain, apply heat to your knees for 15 or 20 minutes prior to performing your exercises.
8. If your knee is swollen after exercise, apply ice pack and reduce the number of repetitions the next time.

Figure 5
 Quadriceps-strengthening exercise concentrating on the vastus medialis oblique muscle

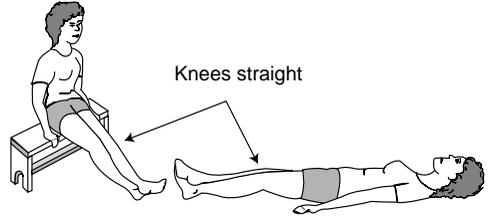


Figure C

Figure D

1. Sit on a firm surface (figure C) or lie flat in bed (figure D).
2. Cross your ankles with right leg above and left leg below. Legs should be stretched out straight.
3. With your heels on the floor or on the bed, push down with right leg, push up with left leg, squeezing your ankles together. (Pretend that you're squeezing a tennis ball between your ankles.) There should be little actual movement except for the muscle tightening.
4. Hold this position for a full 5 seconds. Use a clock or watch with a second-hand, or count: one-one thousand, two-one thousand, three-one thousand, four-one thousand, five one-thousand.
5. Relax the muscles.
6. Reverse the position of the legs so that the leg that was on top is now at the bottom.
7. Repeat steps one, two, and three.
8. Begin your strengthening program with 10 repetitions, holding each contraction for a full 5 seconds. Perform this exercise 7 times daily and increase the number of repetitions you perform with each set by three to five daily during the first week.
9. By the end of the week, you should be able to perform 15 repetitions per set. This is the maximum number of repetitions you should perform in a set. (Total per day = 15 repetitions per set · 7 sets = 105)
10. If your arthritis is causing knee pain, apply heat to your knees for 15 or 20 minutes prior to performing your exercises.
11. Caution: in most patients, these knee exercises will not cause joint pain or increase the pain from your arthritis. If, however, you have significant pain lasting more than 20 minutes after you perform these exercises, decrease the number of repetitions by five per set. Maintain this number of repetitions until your knee discomfort subsides. Then, each day thereafter, increase the number of repetitions by three per set until you reach a maximum of 15 per set.

Adapted from Brandt¹⁷

(B) *Joint Protection*

Assisted Walking Device.

In hip and knee OA, the proper use of a walking stick in the contra-lateral hand reduces forces through these joints by as much as 50%.²¹ Canes should be of correct height. The top of the cane handle should reach the patient's wrist when the patient is standing with the arms at the side. Shoes with good shock-absorbing properties are recommended.

Knee Brace

The use of a knee brace has been shown to lessen the load in the degenerative knee²² and may be appropriate in patients with medial compartment arthrosis and varus malalignment.

^{23,24}

Patellar taping

Medial patella taping in patello-femoral OA followed by quadriceps exercises has been shown to reduce pain and improve function.²⁵

(C) *Pain Relief Modalities*

• *Thermal Modalities*

Thermal modalities may be beneficial in decreasing pain, increasing flexibility and reducing swelling. Some thermotherapy modalities used are hot packs, shortwave diathermy and ultrasound. Heat therapy is not recommended for acutely inflamed joints.

• *Transcutaneous Electrical Nerve Stimulation (TENS)*

TENS has significant benefit in pain relief if treatment duration is more than 4 weeks. Both high frequency and strong burst mode TENS have shown benefit.²⁶

OCCUPATIONAL THERAPY

Occupational therapy helps correct and minimise the dysfunction in lifestyle by improving function through the use of adaptive equipment.

Energy Conservation Techniques

- Plan the task to be done. Give allowance for rest periods.
- Alternate a heavy task with a light task to minimize fatigue.
- When feeling fatigue, stop and rest.
- If the task can be broken down, plan it that way to save energy.
- If pain persists, stop the task. Rest the joints in a splint if necessary.²⁷

Indications for Splinting

Splints are used to improve function, correct position or deformity and reduce pain.

Examples : Protective carpometacarpal splint and knee extension splint.²⁷

Footwear modification

Useful shoe modifications include heel raise, medial arch support and lateral weight shift, lateral arch support and medial weight shift as well as metatarsal arch support.

Stress Management

Pain is often combined with muscle spasm and other signs of stress. Relaxation activities and structured relaxation techniques can help decrease pain which can be taught by the occupational therapist. The patient may need to reschedule activities to accommodate pain peaks.²⁸

DRUG THERAPY

Types of Pharmacological Therapy

Oral	Intra-articular Injection	Topical
Analgescic non - opioid (paracetamol) opioid (codeine, tramadol)	glucocorticoid hyaluronan	methylsalicylate NSAIDs capsaicin
NSAIDs non-selective COX-2 selective others		

Oral therapy

Analgesics

- Non-opioid analgesics:**

These should be used as the first line treatment in OA, e.g. paracetamol. Their efficacy may be as good as that of NSAIDs^{29,30,31} with fewer side effects. Regular dosing of paracetamol may be necessary because of the short half-life. The maximum recommended dose is 4g/day.

- Opioid analgesics:**

Opioids such as codeine, may be used as adjunct therapy when symptoms are inadequately relieved. It can also be used as an alternative for patients with contraindications to other analgesics or NSAIDs.

Side-effects of opioids include nausea and vomiting, constipation, urinary retention, mental confusion, drowsiness, respiratory depression and physical dependence. The dose of opioids used should be titrated such that there is a minimal risk of dependence. Tramadol is a synthetic opioid and does not cause respiratory or CNS depression except in an overdose.^{32,33} Combination drugs such as paracetamol with codeine are also available.

Non Steroidal Anti-inflammatory Drugs (NSAIDs)

NSAIDs provide symptomatic relief from the pain and inflammation associated with OA but do not arrest its progression.

Choice of an NSAID.

- There are no “safe” NSAIDs. The lowest possible dose of NSAID should always be used.
- Large variations are possible in the response of individuals to different NSAIDs.
- If symptoms are not relieved with one NSAID, another class should be tried. (See Appendix 1) The analgesic effect is achieved within 1 week and anti-inflammatory effect within 3 weeks.³⁴
- **Combination therapy with more than one NSAID should never be used. There is no benefit in combination therapy and the incidence of side effects may be additive.**

Dosing

It is reasonable to prescribe NSAIDs on an as needed basis, rather than in a fixed daily dose as pain control may be comparable and toxicity is likely to be lower. If this approach is ineffective, the NSAIDs may be prescribed on a regular basis for a limited period (eg. 3 weeks and review if necessary) Paracetamol or a non-opioid analgesic can be used as “rescue” medication during episodic increases in joint pain, rather than increasing the dosage of the NSAID.

Side effects of NSAIDs.

- Gastrointestinal (GI) intolerance
- Gastrointestinal ulceration, perforation and bleeding
- Blockade of platelet aggregation leading to a bleeding tendency.
- Renal impairment and interstitial nephritis
- Hypersensitivity reactions (e.g. in asthmatic patients)
- Other side effects include drowsiness, dizziness, tinnitus, fluid retention.

Special Precautions

- Caution is required when prescribing NSAIDs to those with renal, cardiac or hepatic impairment, hypertension and pregnancy. Those who are allergic to one NSAID may also be allergic to others.

Issues of GI safety and NSAIDs

NSAIDs are known to cause erosions and ulcers throughout the whole GI tract. Clinically, these may be silent or may present as dyspepsia, upper GI bleeding or ulcer perforations.

An overall rate of 0.73% per year for significant GI events was reported with 0.50% per year for upper GI tract and 0.23% per year for lower GI tract complications.³⁵ In a meta-analysis, Gabriel et al³⁶, calculated an odds ratio of 5.5 for serious GI events in elderly patients when comparing NSAID users vs. non-users.

Pathogenesis of NSAID gastropathy

NSAIDs cause damage to the GI tract through two actions: a topical irritant effect and more importantly an inhibition of prostaglandin secretion in the GI tract. The latter effect is predominantly due to cyclooxygenase 1 (COX 1) iso-enzyme inhibition and is responsible for ulcer formation. (see Appendix 2)

Risk factors for upper GI tract complications³⁶ are

Age > 65

- Comorbid medical conditions
- Oral glucocorticoids
- History of peptic ulcer disease
- History of upper GI bleeding
- Anticoagulants

Treatment of NSAID ulcers

The most effective therapy for NSAID-induced ulcers are the acid-suppressing agents: proton-pump inhibitors (PPIs) and H2 antagonists. Lancaster-Smith et al³⁷ showed that in those who discontinued NSAIDs, 95% of ulcers healed with a standard dose of ranitidine (which was 30% higher than in those who could not discontinue NSAIDs). More recent studies have shown that the PPIs are superior in ulcer healing to H2 antagonists and misoprostol.³⁸

Prevention of NSAID induced ulcers

Antacids are widely prescribed with NSAIDs, but have no effect in preventing the occurrence of ulcers and may in fact mask their presence by suppressing symptoms. H2 antagonists in conventional doses have been shown to reduce the incidence of duodenal but not gastric ulcers, when co-prescribed with NSAIDs.³⁹ In those patients with OA who are at high risk of gastropathy co-treatment with PPIs is recommended. In addition, other agents have also been shown to be useful, e.g. famotidine 40 mg daily and misoprostol, a prostaglandin E1 analogue.

Patients at high risk for serious GI complications should receive prophylactic anti-ulcer treatment. Those with serious co-morbid illness who are at increased risk of mortality if an ulcer complication occurs, should also be considered for prophylactic anti-ulcer therapy. COX-2 selective inhibitors need to be considered in such patients.

COX- 2 Selective Inhibitors

These are drugs which selectively inhibit the COX- 2 enzyme, which is the enzyme shown to be induced during inflammation. Due to this selectivity, these drugs have minimal effect on COX-I, the housekeeping enzyme which yields protective prostaglandins, especially in the GI tract. Hence, this class of drugs should provide analgesic and anti-inflammatory effects without the well-known GI tract adverse effects of conventional NSAIDs.

Rofecoxib (12.5mg and 25mg od), celecoxib (200mg od) and meloxicam (7.5mg od) are as efficacious in OA as existing non-selective NSAIDs but have significantly less GI toxicity.⁴²

The use of these agents has been shown to reduce GI tract complications and perforations, ulcers and bleeds^{43,44} by up to 54%⁴⁵ compared with non-selective NSAIDs.

Co-prescription of low-dose aspirin with standard NSAIDs in patients with history of cardiovascular or cerebrovascular disease is known to increase the risk of GI complications. If an NSAID is considered, it is preferable to use a COX-2 selective agent even though at present there is no published data to support it.

The same cautions must be exercised as with non-selective NSAIDs.

Prescribing in the elderly

Physiological changes in renal and liver function are associated with aging. It is, therefore important to be cautious in prescribing some of the drugs commonly used for the symptomatic relief of osteoarthritis in the elderly patient.

Paracetamol is comparable in efficacy to low or high dose ibuprofen⁴⁶ in symptomatic control of pain and should be used in preference to NSAIDs.^{47,48} NSAIDs should be used with caution, as there is an increased likelihood of gastropathy^{49,50}, deterioration of renal function⁵¹, development of oedema and precipitation of cardiac failure in susceptible individuals.⁵²

The COX-2 selective inhibitors are preferred due to their improved GI side effect profile. Use of these drugs also needs careful monitoring in the elderly patient .

Tramadol can lead to constipation and confusion in the elderly patient if the dose is not titrated carefully.

Intraarticular Therapy

This mode of therapy should be performed only by a practitioner trained in the procedure.

Glucocorticoids

In acute exacerbation of knee OA, intraarticular glucocorticoids^{53,54} can be used after aspiration of a joint effusion. This may provide short term pain relief. Sterile technique is important and long acting glucocorticoids (e.g. triamcinolone, methyl prednisolone) are used. Synovial fluid should be sent for gram stain and culture if infection is suspected. After intraarticular injection, patients should be advised to rest for 24 – 48 hrs. Ideally this should be followed by quadriceps strengthening exercises.

It is not advisable to repeat intraarticular injections at less than 3 monthly intervals.⁵⁵ If not effective initially, repeat injections are not likely to be of benefit. **Systemic glucocorticoids have no role in the management of osteoarthritis.**

Hyaluronan

Hyaluronan is a glycosaminoglycan found in synovial fluid. Viscosupplementation i.e. restoration of viscous and elastic properties of pathologic synovial fluid in OA of the knee has been proposed as a form of treatment.

Results of some clinical trials have shown that administration of hyaluronic acid is superior to placebo in patients with osteoarthritis of the knee.^{56,57} It is generally well tolerated and offers pain reduction as well as functional improvement.

Although there is reasonable evidence to support the efficacy of viscosupplements in patients with OA of the knee, several issues require further exploration i.e. the cost effectiveness of their use in clinical practice, the profile of patients most likely to benefit and the optimal regimen for repeat treatment courses (clinicians should follow the manufacturer's recommendations).

Topical Therapy

Topical NSAIDs, methylsalicylate liniment (LMS), capsaicin and NSAID-containing medicated plasters are useful options in the treatment of OA⁵⁸.

Others

Glucosamine

Glucosamine sulphate has been shown to be useful in relieving pain and improving function in patients with mild to moderate OA.^{59,60} It may retard joint space narrowing and modify disease progression in medial compartment OA but this awaits further confirmation.

Ginger extract and acupuncture may be useful in pain control.^{61,62} However the lack of properly controlled randomised trials makes it difficult to recommend these forms of treatment.

There is no scientific evidence to support the use of the myriad of gadgets and alternative remedies available in the community.^{63,64}

Surgical options

Patients who have refractory pain in spite of medical therapy and/or progressive limitation in activities of daily living should be referred to the orthopaedic surgeon for evaluation to consider surgery

Surgical options include

- Arthroscopic debridement
- Ligamentous reconstruction
- Osteotomy
- Unicompartmental arthroplasty
- Total joint arthroplasty
- Arthrodesis

Total joint arthroplasty is by far the best option in the older age groups (above 60 years).

Parameters useful in selecting the best surgical option are:

- survivorship associated with a given procedure
- complications of the procedure

Before deciding the best surgical option for a patient, important factors to consider are the patient's age, the joints affected, the timing of the surgery and the expertise available.

Arthroscopic debridement^{65,66}

This method provides transient relief of symptoms in mild-to-moderate knee osteoarthritis but does not alter the arthritic process. Patients must be warned about the potential complications and the possibility of a need for subsequent reconstructive surgery.

*Ligamentous reconstruction of the knee joint*⁶⁷

The goals of this procedure are to provide pain relief, and restoration of joint stability. The patients must be counseled that this is a salvage procedure .

Patients with recurrent episodes of symptomatic instability despite comprehensive conservative treatment programme are likely to benefit.

*Osteotomy*⁶⁸

The goal of osteotomy is to provide pain relief and functional improvement. Patients below 60 years of age with unicompartmental knee pain and varus deformity in the absence of patello-femoral symptoms may benefit. Osteotomy has also been used in the hip to alleviate symptoms and delay definitive surgery.

Unicompartmental arthroplasty^{69,70}

Traditionally, this surgical option is for patients with unicompartmental arthritis of the knee who are more than sixty years of age and have a sedentary lifestyle. It is also an alternative to tibial osteotomy or total knee arthroplasty in patients younger than sixty years. Patients with mild to moderate angular deformity and no ligamentous laxity of the knee joint may benefit. The technique is demanding and good results have been reported only in centres of excellence.

*Total joint arthroplasty*⁷¹

Total joint arthroplasty is the mainstay of surgical treatment for osteoarthritis of the knee, hip and glenohumeral joints. Most patients have complete pain relief and near-normal function following successful surgery. Total joint replacement has limited durability beyond 15 years and durability depends largely on the level of physical activity.

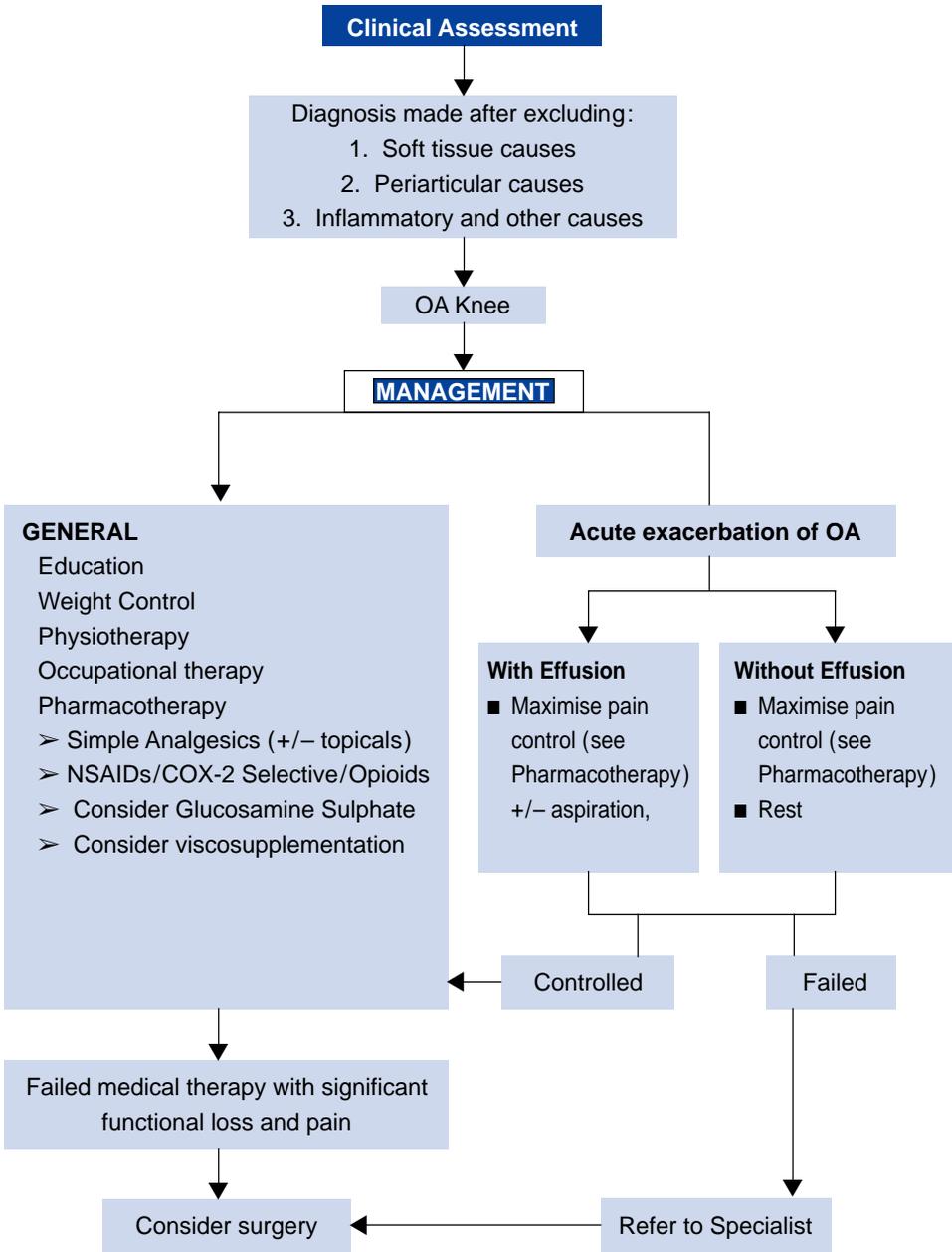
Arthrodesis

Arthrodesis has been shown to effectively alleviate pain and is most commonly performed in the spine, and in small joints of the wrists, hands and feet. In the knee and hip it serves only as a salvage therapy.

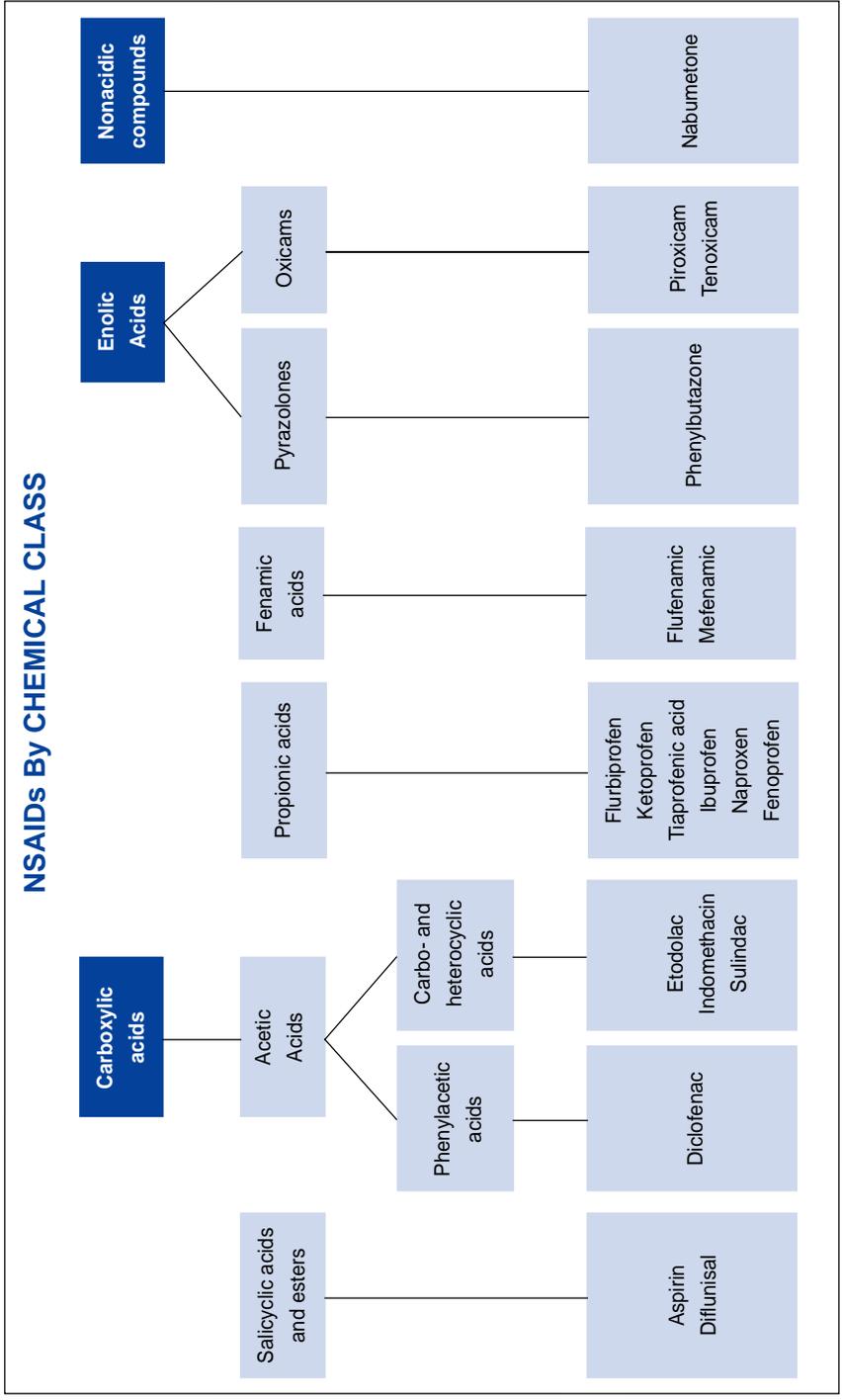
5. PRIMARY PREVENTION

Primary prevention is theoretically possible if all the risk factors (refer page 4) are modified. Many of these risk factors are of particular importance in weight-bearing joints. Prevention of obesity, weight reduction in the obese and health education pertaining to joint protection techniques (including avoidance of trauma to the joints) are recommended as measures for primary prevention. Currently there is no data available to recommend the intake of any preparation to prevent osteoarthritis. An important aspect of primary prevention is to identify those individuals at risk.⁷²

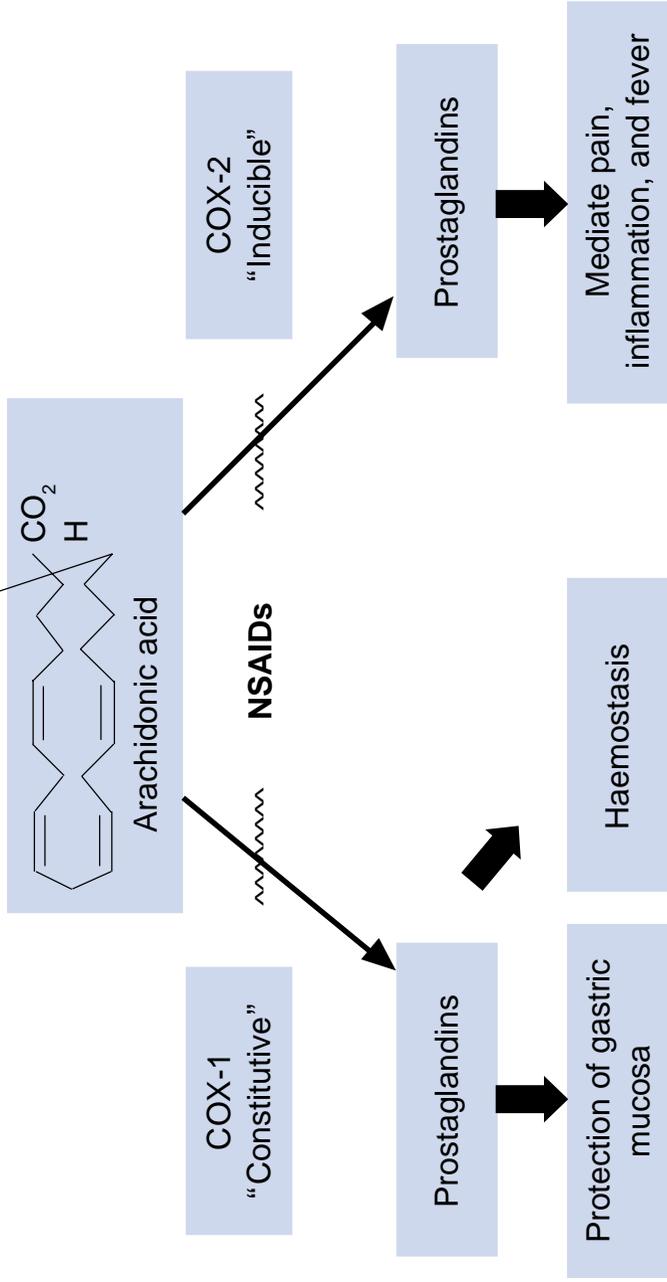
6. ALGORITHM OF MANAGEMENT OF KNEE OSTEOARTHRITIS



Appendix 1



Mechanism of Action of NSAIDs



Appendix 3

Recommendations concerning interventions in the management of osteoarthritis are shown in the following table:

Intervention	Category of evidence
Patient education	1A
Exercise	1B
Analgesic	1B
NSAIDs	1A
COX-2	1B
Topical/periarticular	1B
IA steroid	1B
Opioid	1B
IA hyaluronic acid	1B
Lavage	1B
Patellar taping	1B
Weight reduction	1B
Insoles	2A
Arthroscopic debridement	1B
Osteotomy	3
Joint replacements	3

Categories of evidence⁷³

Category	Evidence from
1A	meta-analysis of randomised controlled trials
1B	at least one randomised controlled trial
2A	at least one controlled study without randomisation
2B	at least one type of quasi-experimental study
3	descriptive studies eg. comparative study, correlation studies, or case-control studies
4	expert committee reports or opinions and/or clinical experience of respected authorities

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