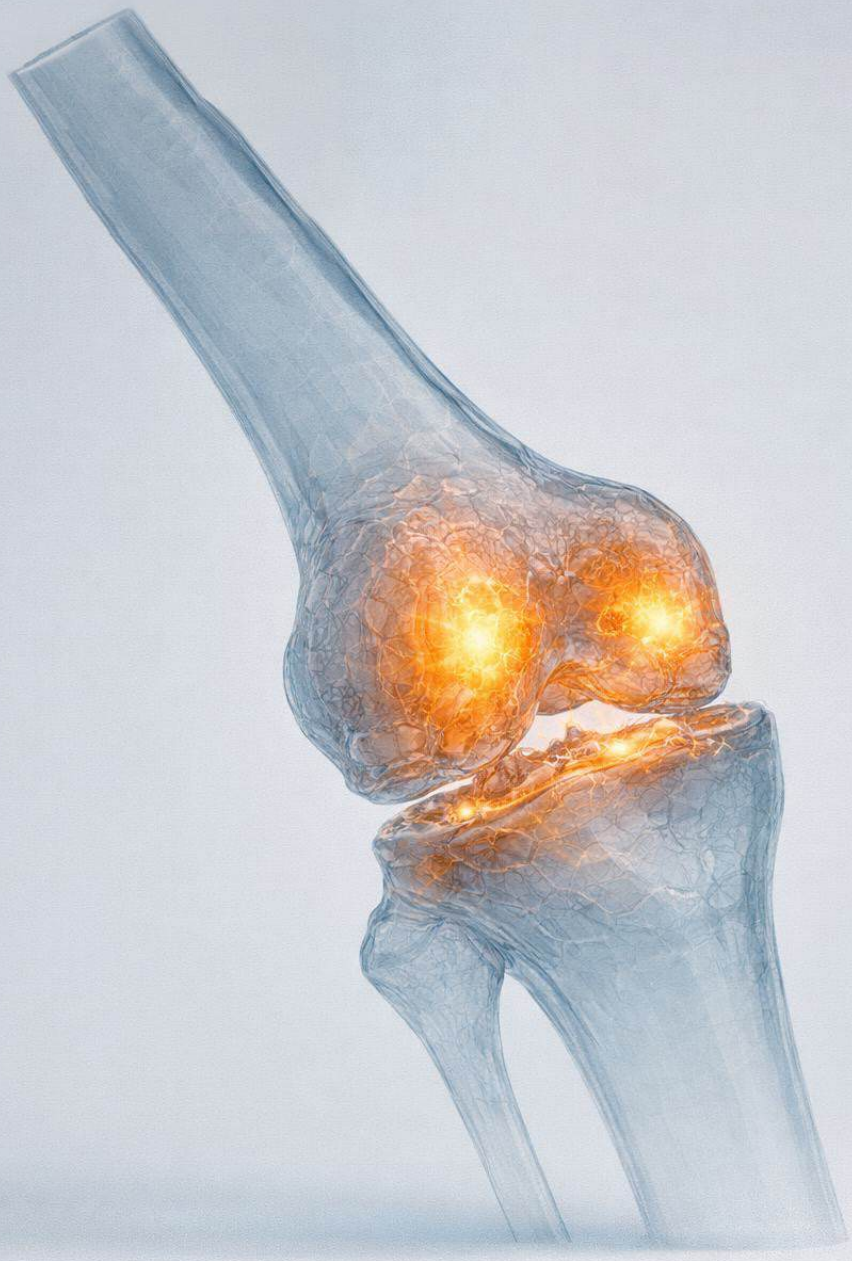
OSTEOARTHRITIS

National Standard Treatment Guideline



Ministry of Health
Republic of Maldives

ADB

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Japan Fund for Prosperous and Resilient Asia and the Pacific

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World Health Organization
Maldives

National Standard Treatment Guidelines

- Acid Peptic Disease
- Acute Anxiety
- Acute Pancreatitis
- Acute Psychosis
- Acute kidney Injury
- Arrhythmia
- Chronic Liver Disease
- Chronic Pancreatitis
- Chronic kidney disease
- Congenital Heart Diseases
- Dementia
- Depression
- Diabetes Mellitus Type 1
- Diabetes Mellitus Type 2
- Gestational Diabetes
- Epilepsy
- Heart Failure
- Hyponatremia
- Hypernatremia
- Hypokalemia
- Hyperkalemia
- Interstitial Lung Disease
- Liver Failure
- Obesity
- Obstructive Sleep Apnoea
- Osteoarthritis
- Ovarian Cancer
- Pneumonia
- Stroke
- Upper Gastrointestinal bleed
- Unstable Angina

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Technical Lead and Editor

Dr. Sangeeta Sharma
Professor, Neuropsychopharmacology, Institute of Human Behaviour & Allied Sciences (IHBAS) & President (Honorary), Delhi Society for Promotion of Rational Use of Drugs (DSPRUD), New Delhi, India

Technical Contributors and Reviewers for STGs

Maldivian Contributors

INTERNAL MEDICINE

Dr. Fathimath Nadia
Senior Consultant in Internal Medicine,
Indira Gandhi Memorial Hospital (IGMH), Male', Maldives

Dr. Moosa Murad
Senior Consultant in Internal Medicine,
Indira Gandhi Memorial Hospital (IGMH), Male', Maldives

Dr. Ibrahim Hassan
Senior Specialist Registrar Internal Medicine,
Medical Director of Kulhudhuffushi Regional Hospital.

Dr. Aminath Munaza
Consultant in Internal Medicine, Hulhumale' Hospital

Dr. Mihuna Ibrahim
Consultant in Internal Medicine, Hulhumale' Hospital

Dr. Ahmed Zooshan
Consultant in Internal Medicine, Indhira Gandi Memorial Hospital

Dr. Shivir Sharma Dahal
Consultant in Internal Medicine, ADK Hospital

Dr. Quraisha Haneef
Consultant in Internal Medicine, ADK Hospital.

Dr. Muhammad Asad UR Rehman Khan
Consultant in Internal Medicine, Tree Top Hospital.

ENDOCRINOLOGY

Dr. Ibrahim Faisal
Consultant in Endocrinology, Indhira Gandi Memorial Hospital.

Dr. Mariyam Niyaz
Consultant in sub specialist in Endocrinology,
Indhira Gandi Memorial Hospital.

Dr. Mohamed Shiruhan
Consultant in sub specialist in Endocrinology,
Indhira Gandi Memorial Hospital.

NEPHROLOGY

Dr. Ahmed Abdulla
Consultant Sub specialist in Nephrology,
Indhira Gandi Memorial Hospital.

RHEUMATOLOGY

Dr. Ibrahim Sujau
Consultant Sub specialist in Rheumatology,
Indhira Gandi Memorial Hospital.

Dr. Sariu Ali Didi
Consultant in Rheumatology, ADK Hospital.

PSYCHIATRY

Dr. Shanooha Mansoor
Consultant in Psychiatry, Indhira Gandi Memorial Hospital.

Dr. Shooga Moosa
Consultant in Psychiatry, Indhira Gandi Memorial Hospital.

Dr. Abdulla Nazim
Consultant in Psychiatry, Indhira Gandi Memorial Hospital.

GASTROENTEROLOGY

Dr. Abdullah Isneen Hilmy
Consultant Sub specialist in Gastroenterology,
Indhira Gandi Memorial Hospital.

PULMONOLOGY

Dr. Mohamed Ismail
Consultant in Pulmonology, Indhira Gandi Memorial Hospital

ORTHOPEDICS

Dr. Ahmed Azim Abdul Shukoor
Consultant in Orthopedics, Hulhumale' Hospital

CARDIOLOGY

Dr. Mohamed Shaneez Najmy
Consultant in Cardiology, Indhira Gandi Memorial Hospital

Dr. Migdhaadh Shareef
Consultant in Cardiology, Indhira Gandi Memorial Hospital

Dr. Aishath Eleena
Consultant in Pediatric Cardiology, Indhira Gandi Memorial Hospital

EMERGENCY MEDICINE

Dr. Fahira Ahmed Rasheed
Consultant in Emergency Medicine,
Indhira Gandi Memorial Hospital

ENT

Dr. Ahmed Shifaz
Consultant in Otolaryngology,
Indhira Gandi Memorial Hospital

OBSTETRICS & GYNAECOLOGY

Dr. Hawwa Inaya Abduraheem
Consultant in Obstetrics and Gynaecology,
Hulhumale' Hospital

Dr. Aminath Juhaina Hameed
Consultant in Obstetrics and Gynaecology,
Hulhumale' Hospital

Dr. Nashwa Samir Hussein Abdulla
Consultant in Obstetrics and Gynaecology,
Medica Hospital

Dr. Shirmeen Mohamed
Consultant in Obstetrics and Gynaecology,
Indhira Gandi Memorial Hospital

PAEDIATRICS

Dr. Abbasa Abdul Hamid
Consultant sub specialist in Paediatric Neurology, Hulhumale' Hospital

Dr Ismail Ejaz Ali
Consultant in Paediatrics, ADK Hospital

Dr.Nusaiba Farouk Hassan
Consultant in Paediatrics, Indhira Gandi Memorial Hospital

Dr. Amany Naseer
Consultant in Paediatrics and Medical Director of
Addu Equatorial Hospital

RADIOLOGY

Dr. Basma Ibrahim Sobir
Consultant in Radiology, Indhira Gandi Memorial Hospital

DENTAL

Dr. Nadeema Rasheed
Consultant in Orthodontics,
Indhira Gandi Memorial Hospital.

MEDICAL OFFICERS

Dr. Suha Abdul Shakoor
Medical Officer, B. Atoll Hospital

Dr. Aishath Maurisha
Medical Officer, Gan Regional Hospital

Dr. Mohamed Hishaam
Medical Officer, Shaviyani Atoll Hospital

Dr. Aishath Shurooq Waheed
Medical Officer, Shaviyani Atoll Hospital

DSPRUD contributors

ENDOCRINOLOGY

Dr. SV Madhu
Director Professor, Department of Endocrinology, Center for Diabetes, Endocrinology and Metabolism, UCMS & GTB Hospital, New Delhi

NEPHROLOGY

Dr. Anil Yadav
Additional Medical Superintendent (Admin), Department of Medicine, UCMS & GTB Hospital, New Delhi.

Dr. Likhita V
Senior Resident and Postgraduate Nephrology Trainee, CMC Vellore.

PSYCHIATRY

Dr. R.K. Chadda
Former Professor & Head, Department of Psychiatry, AIIMS, New Delhi; Consultant, Amrita Hospital, Faridabad, Haryana.

Dr. Amit Khanna
Associate Professor, Department of Psychiatry, IHBAS, New Delhi.

NEUROLOGY

Dr. Suman Kushwaha
Professor, Department of Neurology, IHBAS, New Delhi.

Dr. Mridula Rastogi
Assistant Professor, Department of Neurology, IHBAS, New Delhi.

Dr. Manoj Kumar Sharma
Professor, Department of Hepatology, Institute of Liver and Biliary Sciences (ILBS), New Delhi.

Dr. Monika Jain
Head, Gastroenterology, Balaji Action Hospital, New Delhi.

Dr. Ekta Gupta
Professor, Dept of Clinical Virology, Nodal Officer WHO CC, ILBS, New Delhi

PULMONOLOGY

Dr. Anup R Warrier
Senior Consultant, Infectious Disease Specialist, Aster Medicity, Kochi, Kerala, India.

Dr. Amit Mandal
Pulmonologist & ICU Specialist, Senior Director, Paras Hospital, Panchkula, Haryana, India.

Dr. Manvir Bhatia
Founder, Neurology & Sleep Centre; Vice President, Indian Society of Sleep Research

Dr. Ashok Rajput
Chief Consultant & Pulmonologist, Morpheus Lung & Sleep Clinic, CK Birla Hospital, New Delhi.

Dr. Rajendra Prasad
Director Medical Education & Professor, Respiratory Medicine, Era University, Lucknow, Uttar Pradesh, India.

Dr. Nikhil Gupta
Associate Professor, Department of Medicine, Dr. Ram Manohar Lohia Institute of Medical Sciences, Lucknow, Uttar Pradesh, India.

ORTHOPEDICS

Dr. Sumit Sural
Director Professor & Head, Department of Orthopedics, MAMC & LN Hospital, New Delhi.

Dr. N.V. Kamat
Former Director General Health Services, Govt. of NCT Delhi; Executive Vice President, DSPRUD

CARDIOLOGY

Dr. M.S.S. Mukharjee
Senior Interventional Cardiologist, Pulse Heart Center, Hyderabad

Dr. Neeraj Nishchal
Additional Professor, Department of Medicine, AIIMS, New Delhi.

Dr. Prerna Garg
Senior Resident, Cardiology, AIIMS, New Delhi

Dr. R. Krishna Kumar
Pediatric Cardiology, Amrita Hospital, Kochi

Dr. Aashima Dabas
Professor, Pediatrics, LN Hospital, New Delhi

EMERGENCY MEDICINE

Dr. Vanitha Rajagopalan
Assistant Professor, Critical & Intensive Care, Department of Anesthesiology, AIIMS, New Delhi

OBS & GYNAE ONCOLOGY

Dr. Amita Suneja
Former Director Professor, Department of Obstetrics & Gynaecology, UCMS & GTB Hospital, Delhi.

Dr. Poonam Joon
Deputy Medical Superintendent, Head of Department Obstetrics & Gynaecology, Sanjay Gandhi Memorial Hospital; Secretary, DSPRUD, New Delhi

Endorsed by

Uza. Thasleema Usman
Commissioner of Quality Assurance
Ministry of Health, Male', Maldives

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GUIDELINES DEVELOPMENT METHODOLOGY

The development of the Maldives Standard Treatment Guidelines (STGs) followed a structured, evidence-informed, and consensus-driven methodology adapted from internationally accepted guideline-development standards and the Delhi Society for Promotion of Rational Use of Drugs (DSPRUD) model. The process combined systematic evidence retrieval, critical appraisal, contextual adaptation, and multidisciplinary expert review to ensure feasibility, clinical relevance, and national ownership.

1. Determining Scope and Priority Conditions

Priority clinical conditions were identified through consultation with national programme managers, specialty clinicians, and health-system stakeholders. Selection criteria included: (i) major causes of morbidity and mortality, (ii) observed variation in clinical practice or prescribing patterns, (iii) potential to improve patient outcomes, and (iv) the feasibility of implementation across health-facility levels in Maldives. The final list of diseases reflected national epidemiology, service-delivery capacity, and essential-medicine availability.

2. Identification of Existing Evidence and Source Guidelines

A targeted search strategy was used to identify high-quality existing clinical guidelines. Searches were conducted across international guideline repositories (e.g., WHO, NICE, SIGN and other intergovernmental bodies, international and national guideline repositories, specialty societies and professional associations.

3. Quality Appraisal of Source Guidelines

Retrieved guidelines were screened for transparency of development, methodological rigour, clarity of recommendations, applicability to health-system reality, editorial independence. Guidelines were included if they met the Institute of Medicine (IOM) definition of a clinical guideline and addressed treatment or management of priority conditions. Guidelines that did not meet minimum quality standards, review articles, diagnostic criteria, or technical standards were excluded.

4. Adoption, Adaptation, and Contextualization

The guideline-development team employed an adopt–adapt–contextualize model:

- **Adoption**: High-quality recommendations that aligned with Maldivian health-system realities were retained without modification.
- **Adaptation**: Recommendations were modified when local considerations such as diagnostic capacity, medicine availability, workforce skills, referral pathways, or cost constraints affected feasibility.

- **Contextualization**: Where evidence was absent or inconclusive, conditional recommendations were formulated based on expert consensus, with explicit consideration of pragmatism, safety, and local workflows.
  Medicines were selected in alignment with the Maldives National Essential Medicines List (NEML), based on suitability, efficacy, safety, and availability.

5. Expert Consensus and Multidisciplinary Input

Draft recommendations were initially prepared by experts from the DSPRUD, India, providing a strong methodological foundation for the process. Building on this, a collaborative and participatory process brought together clinicians from internal medicine, paediatrics, obstetrics-gynaecology, surgery, emergency medicine, endocrinology, cardiology, general practitioners, and public health representing different levels of healthcare. Consensus was achieved through moderated discussions, iterative revisions, and resolution of divergent views. For topics lacking strong evidence, recommendations were derived from expert clinical judgment grounded in extensive practice experience.

6. Drafting, Peer Review, and Validation

Each guideline section was organized in a standard format including key clinical features, essential investigations, non-pharmacological management, pharmacological therapy (with step-up/step-down options where relevant), referral criteria, paediatric considerations, and follow-up requirements. Drafts were peer-reviewed by senior clinicians and national experts. Reviewer comments were systematically integrated to strengthen clarity, accuracy, and applicability.

7. Addressing Conflicts of Interest

All contributors declared the absence of conflicts of interest. Individuals with potential or perceived conflicts were excluded from authorship or decision-making roles.

8. Updating and Future Revisions

The STGs were conceptualized as a living document. Future updates will incorporate new scientific evidence, changes in essential-medicine availability, national programme priorities, and user feedback from clinicians. Periodic review cycles will ensure the continued relevance and reliability of recommendations.

9. Distinctive Features of the Guidelines

Developed through a collaborative process involving a large group of multidisciplinary experts from different levels of healthcare, the guidelines incorporate the following distinctive features:

- **Diagnostic Assumption and Confirmation**: While assuming that an initial diagnosis has been established by the healthcare provider, the guidelines provide essential information for confirming diagnoses. This includes a comprehensive overview of major signs and symptoms, descriptions of confirmatory tests, and clear guidance on practices that are prohibited, discouraged, or unreliable—promoting evidence-based medicine supported by relevant references.
- **Comprehensive Treatment Approach**: The guidelines offer a systematic, up-to-date framework for managing medical conditions across the continuum of care. They begin at the primary care level and extend to secondary and tertiary care, incorporating protocols for treatment response assessment and referral criteria as integral components.
- **Diverse Treatment Modalities**: Recommendations encompass both non-pharmacological and pharmacological interventions and surgical intervention where applicable, providing flexibility for individualized treatment plans. Cautionary notes are included where necessary to ensure safe and effective use of therapies.
- **Assessment and Referral Criteria**: Clear criteria and goals for evaluating patient response to treatment are provided, along with guidance on when referral to higher levels of care is warranted ensuring continuity and comprehensiveness in patient management.

ACKNOWLEDGEMENTS

The Government of the Republic of Maldives is committed to ensuring universal access to quality health services for all citizens. The Constitution of Maldives mandates the progressive realization of rights, including the right to good standards of health care for the population. In line with this national commitment, standardized quality health services are regarded as the foundation of a strong and equitable healthcare system.

This important work would not have been possible without the cooperation and support of many individuals and institutions. We express our sincere appreciation to the Honourable Minister of Health, Abdullah Nazim Ibrahim, for his leadership, commitment, and continuous guidance throughout the development process. We are grateful to WHO and ADB for their significant contribution, support, and technical assistance.

Our heartfelt gratitude is extended to the technical lead and editor, Dr. Sangeeta Sharma, Professor, Neuropsychopharmacology, IHBAS and President, Delhi Society for Promotion of Rational Use of Drugs (DSPRUD), and her team. We express our deepest appreciation to the Maldivian and DSPRUD experts and contributors who played a pivotal role in this process. Their technical expertise and dedication to adapt the standards to the Maldivian context have been instrumental in the development and finalization of these guidelines. The time, experience, generous sharing of knowledge and insights contributed by all parties have not only enriched the work but also have been invaluable in making these standards practical, locally acceptable, and aligned with the needs of the resident population.

It is important to acknowledge the immense efforts, involvement, timely coordination, collaboration, and dedication of the Quality Assurance and Regulation Division team who made it possible for these Clinical Treatment Guidelines to come into existence.

Uza. Thasleema Usman

Commissioner of Quality Assurance
Ministry of Health, Male', Maldives

OSTEOARTHRITIS

QUICK REFERENCE GUIDE

Osteoarthritis (OA) is the most common form of arthritis, primarily affecting adults over 50 years, with women being more frequently affected than men. It causes significant morbidity through chronic pain, stiffness, and functional limitation, often leading to reduced mobility and quality of life. While not directly fatal, OA increases risks of disability and secondary complications, but early diagnosis and lifestyle interventions can slow progression and improve outcomes.

Definition

Osteoarthritis is a chronic, degenerative joint disease of the whole joint (cartilage, bone, synovium, ligaments, muscle). Phenotypes include knee, hip, hand (distal interphalangeal (DIP), proximal interphalangeal (PIP), first carpometacarpal), spine; post, traumatic; obesity, associated; malalignment, driven.

Causes, risk factors & triggers

- Mechanics: malalignment, prior injury, high/repetitive load.
- Biology: age, related matrix change; low, grade inflammation.
- Risks: age >50, female sex, obesity, genetics, congenital/acquired deformity, inflammatory arthritis.
- Triggers: overuse spikes, weight gain, cold/low barometric pressure.

Evaluation for diagnosis

- Clinical features: activity, related deep ache; short “gelling” stiffness (<30 min); crepitus; bony enlargement; limited range of motion; joint, line tenderness.
- Physical exam: look/feel/move; assess alignment, gait, strength (quadriceps).
- Laboratory investigations (to exclude): erythrocyte sedimentation rate (ESR)/ C, reactive protein (CRP) usually normal; rheumatoid factor (RF)/anti, cyclic citrullinated peptide (anti, CCP) or serum urate only if alternate diagnoses suspected.
- Imaging: X-ray first line (joint, space narrowing, osteophytes, subchondral changes). Weight, bearing knee views preferred. Magnetic resonance imaging (MRI) only if symptoms persist with normal/equivocal X-ray or to assess soft tissue; ultrasound (US) for effusion/injection guidance. (NICE)

Classification / severity assessment

- Radiographic: Kellgren-Lawrence grades 1-4.
- Symptoms/function: Visual Analogue Scale (VAS) or Numeric Rating Scale (NRS) for pain; Western Ontario and McMaster Universities Osteoarthritis...

Index (WOMAC) or Lequesne index for function/disability.

- Rule: treat symptoms and function, not films.

Differential diagnosis

- Inflammatory arthritis (rheumatoid, psoriatic), calcium pyrophosphate dihydrate (CPPD) disease, gout.
- Septic arthritis (hot red joint), avascular necrosis (AVN), fracture, meniscal/ligament injury, bursitis/ tendinopathy, referred pain (lumbar spine/hip).

Management goals & principles

- Reduce pain and stiffness; improve function and participation; slow progression; minimize harm; delay/ avoid surgery.
- Multimodal, stepwise, individualized; non, drug care for everyone; medicines as adjuncts. (NICE)

Approach to Management

1. All patients: education; structured exercise; weight management if overweight; review comorbidities/ medications; flare plan.
2. Escalation: add topical then oral medicines if needed; consider injections for short-term relief; refer if persistent disability or deformity.
3. Surgery: for severe pain/disability after maximal conservative care.

Non-pharmacological interventions

- Exercise (core): land, based strengthening plus aerobic; balance; range of motion. Home-based or group formats acceptable; reassure that initial pain rise is common and safe.
- Weight loss: structured diet plus activity for knee/hip OA with overweight/obesity.
- Education & self-management: pacing, activity modification, joint protection.
- Aids/adaptations: cane in contralateral hand, knee bracing for uni-compartmental symptoms, proper footwear/insoles; home/work modifications.
- Modalities: heat/cold as adjuncts.
- Avoid/limited: acupuncture/dry needling (do not offer routinely).

Pharmacological therapy

Use the lowest effective dose for the shortest duration; review risks every visit.

1. **Topical first**: Topical non-steroidal anti-inflammatory drug (NSAID): e.g., diclofenac 1% gel 2-4 g to knee/hand up to four times daily. Indicated for knee/hand OA; favorable safety. Or Topical capsaicin: 0.025-0.075% cream to affected joint 3-4 times/ day; burning/erythema common first 1-2 weeks.
2. Oral options (if persistent symptoms / multiple joints)

- Oral NSAIDs short courses: Paracetamol (acetaminophen) 500-1000 mg every 6-8 hours (max 3 g/day) Or ibuprofen 200-400 mg three to four times daily, naproxen 250-500 mg twice daily. Cautions: gastrointestinal (GI), renal, cardiovascular (CV) risk; co-prescribe proton pump inhibitor (PPI) if GI risk; avoid in chronic kidney disease and high CV risk.
- Adjunct in neuropathic pain component (selected cases), Pregabalin: 75 mg at night (or 50 mg twice daily); titrate to 150-300 mg/day as needed (max 600 mg/day). Adjust for renal impairment. If unsuitable, amitriptyline (10-25 mg at night, titrate) or gabapentin (100-300 mg three times daily, titrate).
- Tramadol (short term, rescue): 25-50 mg every 6 hours as needed (max 200-300 mg/day). Risks: sedation, falls, nausea, dependence-use sparingly, avoid in older/frail if possible.
- Severe pain unresponsive to NSAIDs/steroids (short-term use) Opioids: avoid, if possible; consider only short, infrequent rescue when all else unsuitable. (NICE)

3. Intra-articular therapies (short-term relief for flares)
   - Corticosteroid injection: triamcinolone acetonide 40 mg Intra-articular (IA) or methylprednisolone acetate 40 mg IA; limit frequency (e.g., ≤3-4/year); screen for diabetes control and infection.
   - Hyaluronic acid: do not offer routinely (variable benefit, cost). (NICE)

Not recommended / uncertain: Glucosamine/chondroitin (not recommended by several guidelines); platelet, rich plasma and stem, cell injections lack consistent evidence for routine use. (NICE)

Assessment of response, review, and adjustment

- Follow, up: 4-6 weeks after starting/changing therapy; then every 3-6 months if stable; sooner for High-risk (long-term NSAIDs, uncontrolled pain).
- Track: pain (VAS/NRS), function (WOMAC/Lequesne), mobility, adverse effects, adherence, satisfaction.
- Step-up: persistent moderate-severe pain despite optimal non-drug plus first-line drugs; declining function; radiographic progression with symptoms.
- Step-down: stable ≥3-6 months, reduced analgesic need, patient preference to minimize meds.

Referral (tiered)

- Primary to Secondary care: uncertain diagnosis/atypical features; failure after 3-6 months of conservative care; progressive functional loss; recurrent effusions; imaging or injections needed but unavailable.
- Secondary to Tertiary/Orthopedics: severe OA (e.g., Kellgren-Lawrence 3-4) with refractory pain/disability ± ...

deformity/instability/malalignment; surgical candidacy; complex comorbidities.

- Document & Send: diagnosis, imaging, treatments tried (including intra, articular), scores, comorbidities, patient goals.

Complications

- Pain/disability, deconditioning, falls; work loss; medication harms (GI bleed, renal/CV events; steroid, related hyperglycemia); peri, operative risks if surgery required; anxiety/depression.

Patient education: objectives & caregiver instructions

- OA is chronic but manageable; control is the goal and not cure.
- Set targets: pain relief, mobility, participation.
- Emphasize on movement: daily low, impact exercise; pace activities; protect joints.
- Set weight goals if overweight, sleep and mood care.
- Medicine safety: follow doses; avoid unsupervised NSAIDs or duplicate products as in various Fixed dose combinations containing NSAIDs or over-the-counter products.
- Explain Red flags: hot/red swollen joint, fever, severe or rapidly worsening pain, seeking urgent care.
- Emphasize use aids and home/work adaptations; engage in shared decisions; track symptoms.

INTRODUCTION

Arthritis is inflammation of one or more joints that causes pain, stiffness, and loss of function. Osteoarthritis is the most common type, affecting about 600 million people, more than twice the 1990 estimate with knees leading the burden. Most patients are women and over 55, though obesity and prior injury put younger adults at risk; in Southeast Asia the load is high, and up to 60% of older Thais have clinical or radiographic knee OA. Arthritis isn't directly fatal, but inactivity and weight gain from painful joints raise cardiovascular and metabolic risks. There's no cure, yet early diagnosis, exercise, based rehabilitation, weight control, and careful analgesic use slow progression and cut disability. Standardized care is essential to reduce variation, avoid excessive NSAIDs, ensure physiotherapy, and trigger timely referral.

SCOPE AND INTENDED USERS

These guidelines cover diagnosis and non-surgical care of adult osteoarthritis across primary, secondary, and tertiary settings. They outline case detection, evaluation, simple grading, pharmacologic and non, pharmacologic treatment, patient education, referral pathways, and when to consider surgery (without surgical technique details.

Intended users

The primary target audience comprises general practitioners, primary, care physicians, family physicians, specialists, community health workers, and physiotherapists, often the first point of contact for individuals with OA symptoms. The content is also relevant to nurses, rehabilitation specialists, and policy makers involved in designing service delivery protocols for musculoskeletal care.

Applicability by healthcare level:

- **Primary care**: Focuses on early detection through history and examination, use of simple diagnostic tools, initiation of conservative measures (e.g., analgesia, exercise programs, lifestyle modification), and identification of red flags that require urgent referral.
- **Secondary care**: Expands on the primary, level approach by adding access to imaging (e.g., X-ray), targeted laboratory tests, and specialist consultations for confirmation of diagnosis, escalation of therapy, or administration of Intra-articular injections.

- **Tertiary care**: Reserved for complex, refractory cases and surgical planning (e.g., total joint replacement, osteotomy). Includes multidisciplinary evaluation to optimize patient readiness for surgery.

DEFINITIONS

OA is a chronic, progressive, degenerative joint disorder that primarily affects synovial joints. It is characterized by the breakdown and eventual loss of articular cartilage, remodeling of the underlying subchondral bone, formation of osteophytes (bony outgrowths), and varying degrees of synovial inflammation. These structural alterations lead to pain, stiffness, reduced range of motion, and functional impairment.

OA is traditionally considered a “wear and tear” condition; however, it is now recognized as a multifactorial disease involving mechanical, metabolic, genetic, and inflammatory components. It can be classified as:

- **Primary (idiopathic) OA**: Occurs without an identifiable cause, often age, related and influenced by genetic predisposition.
- **Secondary OA**: Develops as a consequence of known factors such as joint injury, congenital or developmental abnormalities, inflammatory arthritis, metabolic disorders, or previous joint surgery.

CAUSES, RISK FACTORS & TRIGGERS

OA is a multifactorial disease resulting from the interplay between mechanical forces, biochemical processes, and age, related changes in joint tissues. While the exact initiating event may vary, the disease represents a failure of the joint’s capacity to repair and maintain itself in the face of cumulative micro, injury.

Causes/risk factors/triggers	Mechanism	Modifiable?	Practical actions
Mechanical wear and tear	Chronic loading erodes avascular cartilage; limited regeneration → cumulative damage	Partly	Activity pacing, low, impact exercise, ergonomics, weight reduction
Biochemical mediators (MMPs, aggrecanases driven by IL, 1β, TNF, α)	Enzyme, mediated breakdown of collagen/proteoglycans weakens matrix	Partly	Treat comorbid inflammation, exercise therapy, guideline, directed meds
Aging	Reduced proteoglycans, increased collagen cross, links; altered subchondral bone and synovial fluid	No	Strengthen peri, articular muscles, symptom control, fall prevention
Age >50 years	Strongest predictor; prevalence rises with age	No	Early detection; joint, friendly activity

Female sex (post, menopause)	Hormonal influences on cartilage/bone	No	Strength/conditioning; address osteoporosis where present
Obesity	Higher joint load; adipokine, driven low, grade inflammation	Yes	Weight loss, nutrition support, aerobic + strength training
Prior joint injury	Meniscal/ligament tears, Intra-articular fractures disrupt mechanics	Partly	Early rehab, neuromuscular training, protect from re, injury
Repetitive joint use	Squatting, kneeling, heavy lifting, high-impact sports accelerate wear	Yes	Task modification, ergonomics, cross, training, rest cycles
Genetics	Variants in cartilage/collagen/ BMP pathways ↑ susceptibility	No	Family risk counseling; early lifestyle measures
Congenital anatomical anomalies	DDH, coxa vara/valga alter joint reaction forces	No (baseline)	Optimize mechanics; orthotics; corrective surgery if indicated
Acquired malalignment	Varus/valgus knee increases compartment load	Partly	Bracing, targeted physio, weight loss; consider realignment surgery
Inflammatory arthritis (secondary OA)	Synovitis and structural damage lead to OA	Partly	Diagnose/tx primary disease early; DMARDs/urate control as appropriate

Triggers of Flare or Pain Exacerbation

Even in established OA, symptoms often fluctuate. Common triggers for acute worsening include:

- Overuse - Increased physical activity beyond the joint's tolerance, especially repetitive or high-impact movements, can provoke pain and swelling.
- Weight gain - Even modest increases in body mass can significantly raise joint loading, especially on the knees (where each extra kilogram of body weight translates into roughly four kilograms of extra force per step).
- Weather changes - Many patients report increased pain and stiffness with drops in barometric pressure or exposure to cold and damp conditions. While mechanisms are unclear, theories include altered joint fluid pressure and sensitization of pain receptors.

EVALUATION FOR DIAGNOSIS

Domain	OA, positive cues	Red flags / differentials)
Confirm OA clinically, exclude other causes, gauge impact	Mechanical joint pain worse with use, brief stiffness after rest (<30 min), crepitus, bony enlargement, reduced range of motion, joint line tenderness; typical joints: knee, hip, DIP/PIP, 1st CMC	Prolonged morning stiffness, hot red joints, systemic features
Pain & stiffness: Character, onset, activity/rest relation	Pain worsens with use, eases with rest; gradual onset; "gelling" after inactivity <30 min	Morning stiffness >30-60 min, nocturnal inflammatory pain, fever
Function: Activities of daily living (ADLs): stairs, squatting, walking, grip/pinch	Limits on rising from chair, climbing stairs; jar opening difficulty (hand OA)	Rapid functional decline out of proportion to findings
Site involved: Knee, hip, hand distribution	Knee: weight, bearing pain; Hip: groin/thigh pain, shoes on/off hard; Hand: DIP/PIP/1st CMC pain	Widespread small, joint swelling (RA), MCP predominance, acute 1st MTP (gout)
Exam - Inspection & palpation Look/feel/move the joint	Crepitus; joint, line tenderness; bony enlargement	Warmth, erythema, marked effusion, diffuse boggy synovitis
Exam - Range of movement (ROM) & strength Active/ passive ROM; muscle bulk	End, range restriction; quadriceps wasting in knee OA	Neurologic deficits, severe instability, locking from loose body/meniscal tear
Exam Hands , DIP/PIP/1st CMC nodes	Heberden (DIP) and Bouchard (PIP) nodes	Synovitis of MCPs/WRISTs (think RA/psoriatic)
Labs (to exclude) Order only if atypical/inflammatory features ESR/CRP (selective); RF/anti, CCP if RA suspected; serum urate if gout possible; CBC if indicated	ESR/CRP usually normal or mildly ↑	High ESR/CRP → inflammatory arthritis/infection; anemia/ infection on CBC

CONFIRMATION OF DIAGNOSIS

The diagnosis of OA is primarily clinical, based on characteristic symptoms and physical examination findings, and supported when available by imaging studies. Laboratory tests play an exclusionary role rather than a confirmatory one. The aim at this stage is to confirm the degenerative nature of the joint disease, rule out inflammatory, infective, or crystalline arthropathies, and determine the severity of structural damage.

Component	Purpose	When to use	Key findings	Limitations / notes
X-ray (plain radiography)	First-line imaging to support diagnosis and stage severity	If diagnosis uncertain, to grade severity, monitor progression, or before procedures	Joint space narrowing, osteophytes, subchondral sclerosis, subchondral cysts	Correlate with symptoms; early OA may have normal X-ray

Weight, bearing X-ray views	Better assessment of lower, limb joint space	Knee/hip OA evaluation and pre, op planning	More accurate joint space loss on standing AP knee/ hip views	Ensure correct positioning and compartment views
MRI	Problem, solving and soft, tissue assessment	Persistent symptoms with normal or equivocal X-ray; suspected meniscal/ligament injury; to differentiate OA from AVN or inflammatory arthritis	Cartilage loss, bone marrow lesions, synovitis, meniscal and ligament pathology	Not routine for OA diagnosis; higher cost and limited access
Ultrasound	Adjunct and procedure guidance	To detect effusion/ synovial thickening; to guide injections	Effusion, synovial hypertrophy, osteophytes; dynamic assessment	Limited for cartilage and deep structures; operator dependent

CLASSIFICATION / SEVERITY ASSESSMENT CRITERIA

The classification of OA severity serves two essential purposes: guiding treatment decisions and providing a standardized framework for monitoring disease progression. Severity assessment should integrate structural findings from imaging, functional impairment in daily activities, and symptom burden reported by the patient. Always treat the patient, not the film; symptoms and images often don't align.

1. Radiographic Severity - Kellgren-Lawrence (K-L) Grading System

Grade	Radiographic Features	Interpretation
0	No radiographic features of OA	Normal
1	Doubtful joint space narrowing, possible osteophyte formation	Possible OA
2	Definite osteophytes, possible joint space narrowing	Mild OA
3	Multiple osteophytes, definite joint space narrowing, some sclerosis, possible bony deformity	Moderate OA
4	Large osteophytes, marked joint space narrowing, severe sclerosis, definite bony deformity	Severe OA

2. Functional Severity

Western Ontario and McMaster Universities Osteoarthritis Index (WOMAC): The WOMAC index is a validated, patient, reported questionnaire widely used to assess pain, stiffness, and physical function in OA, particularly of the knee and hip. Domains and Scoring include:

- **Pain** - 5 questions (e.g., pain during walking, using stairs, in bed, sitting/lying, standing upright)
- **Stiffness** - 2 questions (morning stiffness, stiffness later in the day)
- **Physical function** - 17 questions (difficulty with daily activities such as bending, walking, rising from a chair)

Responses are scored on a Likert or visual analog scale (VAS), with higher scores indicating greater severity. Scores can be expressed as raw values or normalized to a scale of 0-100.

Functional Severity - Lequesne Algo functional Index: The Lequesne index is another validated tool for assessing OA severity, especially in the hip and knee. It combines pain/ discomfort, maximum walking distance, and activities of daily living into a singl e score (0-24).

3. Combined Severity Assessment

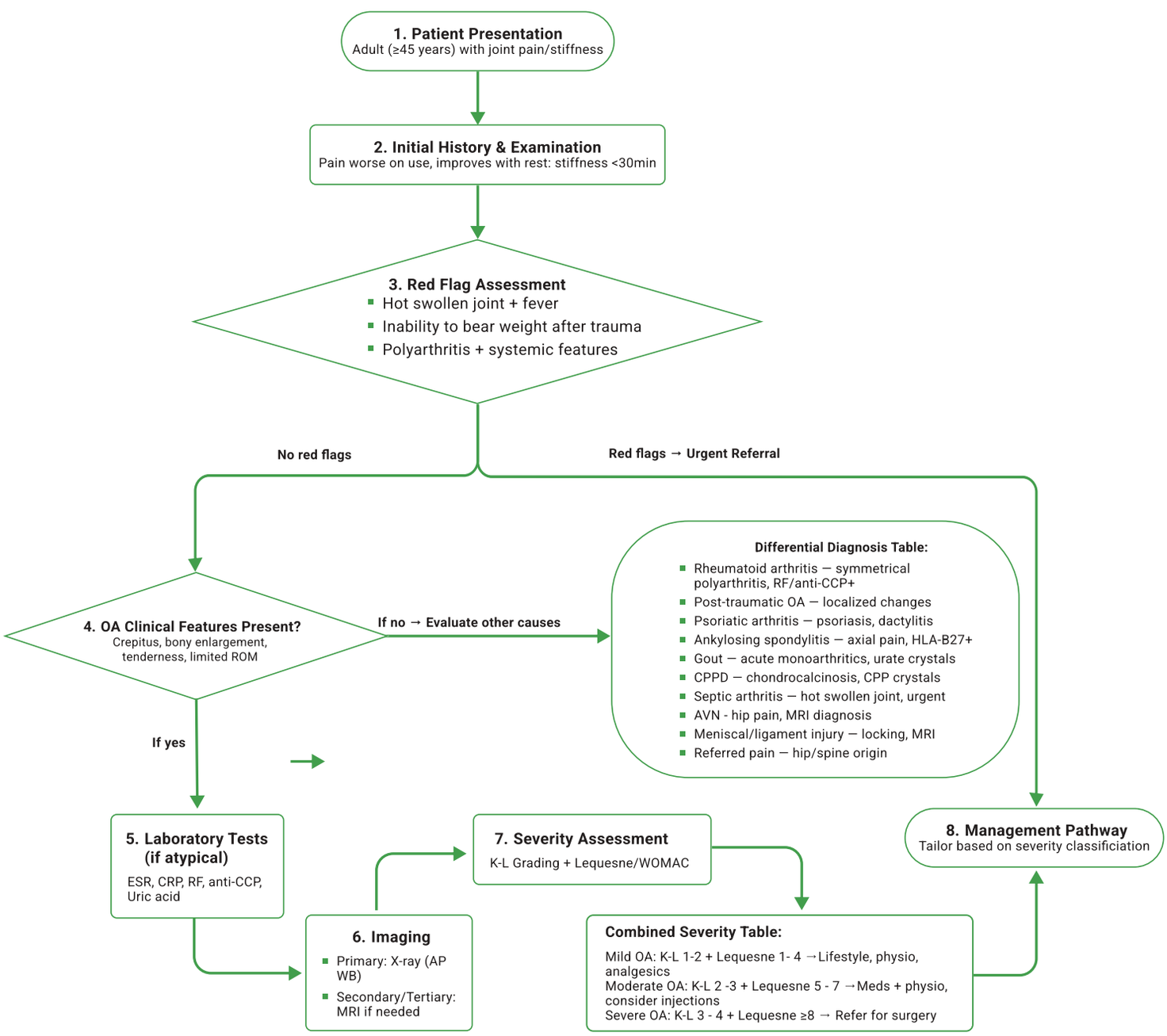
An integrated classification combining radiographic findings (K-L grade) with functional scores (WOMAC or Lequesne) provides a more comprehensive picture:

Category	Radiographic Severity (Kellgren-Lawrence Grade)	Functional Severity (Lequesne Index / WOMAC)	Clinical Relevance
Mild OA	Grade 1-2: Definite osteophytes, possible joint space narrowing (mild OA)	**Lequesne**: 1-4 (mild disability) **WOMAC**: low score, minimal limitation	Emphasize lifestyle modification, weight management, physiotherapy, activity pacing, and simple analgesics as needed.
Moderate OA	Grade 2-3: Multiple osteophytes, definite joint space narrowing, some sclerosis, possible deformity (moderate OA)	**Lequesne**: 5-7 (moderate disability) **WOMAC**: moderate score, regular interference with Activities of daily living (ADLs)	Combination of pharmacologic (NSAIDs, topical agents) and non, pharmacologic measures; consider Intra-articular corticosteroid injections; structured rehabilitation program.
Severe OA	Grade 3-4: Large osteophytes, marked joint space narrowing, severe sclerosis, definite deformity (severe OA)	**Lequesne**: 8-10 (severe), 11-13 (very severe), ≥14 (extremely severe disability) **WOMAC**: high score, significant limitation	Refractory to conservative measures; requires orthopedic evaluation for surgical intervention; optimize pain control and pre, surgical conditioning.

Key Points in Severity Assessment:

- Radiographic severity does not always correlate with symptom severity; some patients with advanced OA on X-ray may report minimal symptoms, while others with early OA may have significant pain and disability.
- Regular reassessment using both imaging and functional scales is important to monitor progression and treatment response.
- Severity classification should guide stepwise management starting with conservative measures in early disease and escalating to invasive interventions only when necessary.

DIAGNOSTIC FLOWCHART



DIFFERENTIAL DIAGNOSIS

Condition	Key Distinguishing Features	Investigations	Differentiating Points from OA
Rheumatoid Arthritis (RA)	Symmetrical polyarthritis, morning stiffness >60 min, joint swelling, systemic symptoms (fatigue, low, grade fever)	Raised ESR/CRP, RF +/-, anti, CCP +, X-ray shows erosions without osteophytes	OA stiffness <30 min, osteophytes present, usually asymmetrical
Psoriatic Arthritis	Asymmetric arthritis, dactylitis ("sausage digits"), skin/nail psoriasis	Raised ESR/CRP, RF negative X-ray: pencil, in, cup deformity	OA lacks skin/nail changes and erosive deformities
Ankylosing Spondylitis	Axial skeleton involvement, sacroiliitis, morning stiffness improving with activity, younger onset	Raised ESR/CRP, HLA, B27 positive, X-ray/MRI sacroiliac joints	OA usually affects peripheral joints, older onset
Gout	Sudden severe monoarthritic (1st MTP common), redness, swelling	Raised Serum uric acid, MSU crystals in synovial fluid	OA pain gradual, non, inflammatory; gout has acute flares
CPPD (Pseudogout)	Acute/chronic arthritis, knees/wrists common, possible low, grade inflammation	Chondrocalcinosis on X-ray, CPP crystals in synovial fluid	OA lacks chondrocalcinosis and acute inflammatory episodes
Septic Arthritis	Acute hot swollen joint, fever, inability to move joint	Joint aspiration (Gram stain, culture), Raised ESR/CRP & WBC	OA is non-infective; septic arthritis needs urgent antibiotics
Avascular Necrosis (AVN)	Hip pain may progress rapidly, history of steroid use/alcoholism	MRI hip (early changes), X-ray (late collapse)	OA changes are chronic; AVN may be sudden in progression
Post, traumatic Arthritis	Localized to previously injured joint, history of fracture/ligament injury	X-ray shows localized changes	OA may be generalized without trauma history
Meniscal/ Ligament Injury	Locking, instability, pain with twisting, positive special tests	MRI joint	OA pain is activity, related but without locking/ instability
Referred Pain (hip/spine)	Pain referred to knee or hip, neurological signs possible	X-ray spine/hip, neurological exam	OA pain localized to affected joint without nerve involvement

Key Clinical Pointers for Differentiation:

- Morning stiffness > 60 min → inflammatory arthritis likely.
- Acute monoarthritis with swelling/redness → consider septic arthritis or gout.
- Bilateral symmetrical small joint involvement → think RA.
- Abrupt episodic pain with swelling → think crystal arthropathy.
- Mechanical pain without systemic signs → more consistent with OA.

MANAGEMENT GOALS

Goals: relieve pain and stiffness; improve function and mobility; slow structural progression; preserve quality of life; delay or avoid surgery; build long-term self, management through education and lifestyle change.

MANAGEMENT PRINCIPLES

The management of OA is guided by its chronic, progressive, and non-curable nature. Management should be individualized, considering disease severity, the patient's functional status, comorbidities, preferences, and available healthcare resources.

- **Stepwise Approach**
  - Mild OA: Education, lifestyle modification, exercise, weight management, occasional analgesics.
  - Moderate OA: Add NSAIDs or topical agents, physiotherapy, and Intra-articular corticosteroids if needed.
  - Severe OA: Optimize conservative care, introduce assistive devices, and consider referral for surgical evaluation.
- **Multimodal Care**: Combine non, pharmacological measures (education, exercise, physiotherapy, weight loss, activity modification, supportive devices) with pharmacological options (analgesics, NSAIDs, Intra-articular therapies) tailored to safety and tolerance.
- **Rational use**: avoid excessive NSAIDs; don't prolong immobilization; avoid unproven/ unsafe therapies; time surgery to substantial functional compromise after conservative measures fail.
- **Monitoring**: reassess pain, function, and adverse effects regularly; step up or taper treatment to maintain control with minimal harm; trigger referral for red flags, progressive deformity/instability, or refractory symptoms.

NON-PHARMACOLOGICAL INTERVENTIONS

Non, pharmacological strategies form the cornerstone of OA management and should be implemented for all patients, regardless of disease stage or pharmacological treatment.

Do's	Don'ts	Less, Evidenced / Adjunctive Options
Maintain regular low, impact physical activity (walking, swimming, cycling, yoga, tai chi).	Avoid high-impact activities (running, jumping, contact sports).	Acupuncture - some patients report relief, but evidence is inconsistent.
Practice weight management to reduce load on weight, bearing joints.	Do not prolong rest or immobilization - this worsens stiffness and muscle wasting.	TENS (Transcutaneous Electrical Nerve Stimulation) - temporary pain relief, limited long-term benefit.
Do strengthening exercises for quadriceps, hip, and core.	Avoid unsuitable footwear such as high heels or unstable shoes, thin soled slippers.	Nutritional supplements (glucosamine, chondroitin, fish oil, turmeric) - mixed evidence.
Use heat for stiffness and cold packs for swelling.	Avoid self, medicating with unproven remedies (copper bracelets, magnets).	Alternative therapies - yoga, mindfulness, Ayurveda, chiropractic may support well, being.
Use assistive devices (canes, braces, walkers) appropriately.	Don't over, rely on braces/canes without rehab, as this worsens muscle weakness.	PRP/Stem Cell therapy - marketed but still experimental, not standard care.
Opt for patient education: pacing, gait training, joint protection techniques, ergonomic adjustments.	Avoid depending only on passive therapies (massage, ultrasound) without exercise.	Hydrotherapy - buoyancy helps in obese/ advanced OA, but not widely available.

PHARMACOLOGICAL THERAPY

Pharmacological therapy is supportive and symptom, focused. It does not reverse cartilage damage or halt disease progression, but it plays a key role in reducing pain, improving function, and enabling patients to participate in non, pharmacological interventions such as exercise and physiotherapy.

Short courses of NSAIDs can control acute symptom exacerbations triggered by overuse or inflammation, preventing prolonged disability. In advanced disease, medications may provide temporary relief while patients await surgical intervention or when surgery is not possible due to comorbidities.

Tramadol or weak opioids may be considered in refractory cases, but only for short-term or targeted use due to side effect risks. Long-term use, particularly of NSAIDs or opioids, carries significant risks (GI bleeding, renal impairment, cardiovascular events, dependence), so regular review is essential.

Note: Use the lowest effective dose for the shortest duration, review regularly, and always combine with non-drug measures.

Not recommended / uncertain: Glucosamine/chondroitin (not recommended by several guidelines); platelet, rich plasma and stem, cell injections lack consistent evidence for routine use. (NICE)

Pharmacological therapy

Indication	Drug	Dose	Route	Duration	Cautions / Monitoring
First-line analgesia	Paracetamol (Acetaminophen)	500-1000 mg every 6-8 hours (max 3 g/day)	Oral	Intermittent or short-term regular use	Avoid in chronic liver disease; caution with alcohol use
Mild-moderate pain	Topical NSAIDs (e.g., Diclofenac 1% gel)	2-4 g applied to affected joint 3-4 times/day	Topical	As needed	Local skin irritation; avoid on broken skin
Moderate-severe pain	Oral NSAIDs (e.g., Ibuprofen 200-400 mg TID; Naproxen 250-500 mg BID)	Variable	Oral	Shortest effective duration	GI bleeding risk, renal impairment, cardiovascular risk; *Consider gastroprotection (PPI) in High-risk patients
Severe pain unresponsive to NSAIDs/ steroids (short-term use)	Weak opioids (e.g., Tramadol 25-50 mg q6-8h)	Oral	Shortest duration possible	Sedation, constipation, dependence; avoid in elderly unless no alternatives	Avoid opioid use; impractical in resource, limited settings.
Adjunct in neuropathic pain component (selected cases)	Pregabalin: 75 mg at night (or 50 mg twice daily); titrate to 150-300 mg/day as needed (max 600 mg/day). Adjust for renal impairment. If unsuitable, amitriptyline (10-25 mg at night, titrate) or gabapentin (100-300 mg three times daily, titrate	Oral	Trial for 4-6 weeks	Nausea, dry mouth, fatigue; avoid in severe liver disease	Avoid opioid use; impractical in resource, limited settings.

***PPIs with long-term NSAIDs**: Give a PPI whenever an oral NSAID is used for OA, especially in older patients, anyone with prior ulcer/bleed, high NSAID dose, or concomitant aspirin/anticoagulant/antiplatelet/steroid use. Omeprazole 20 mg daily or pantoprazole 40 mg daily, or equivalent, for as long as the NSAID is needed; deprescribe once the NSAID stops. Test-and-treat Helicobacter pylori in patients with ulcer history or high risk.

Note: PPIs reduce upper-GI ulcers/bleeds but don't prevent lower-GI injury. Monitor renal/ CV risks from NSAIDs and PPI adverse effects if therapy is prolonged.

Intra-articular injections

Intra-articular injections are used as an adjunct in OA management when symptoms persist despite optimal non, pharmacological measures and oral/topical pharmacologic therapy particularly when inflammation is suspected (joint effusion, synovitis). They provide localized symptom relief, especially for knee and hip OA, and can help bridge patients to more definitive treatments.

- Corticosteroid Injections: Triamcinolone acetonide (20-40 mg), Or methylprednisolone acetate (40 mg). Provides pain relief for 4-12 weeks in many patients; effect diminishes with repeated injections. Not more than 3-4 injections per joint per year to minimize cartilage damage. **Cautions:** Use aseptic technique to prevent infection; transient flare in pain may occur; can cause hyperglycemia in diabetic patients; avoid in suspected or confirmed joint infection.
  Hyaluronic Acid in case of symptomatic knee OA when other options are unsuitable or ineffective. Evidence for benefit is mixed; some patients experience moderate pain relief for several months. Limitations are high cost, need for repeated injections, limited benefit in advanced OA.(NICE)

Surgical Interventions

Surgery is considered for patients with advanced OA who have persistent pain, functional limitation, or joint deformity despite maximal conservative therapy. The choice of procedure depends on the joint involved, severity of disease, patient age, activity level, and comorbidities.

a. Arthroscopic Debridement / Lavage in selected patients with mechanical symptoms due to loose bodies or meniscal tears. Not recommended for routine OA management, no proven long-term benefit in degenerative disease without mechanical locking.

b. Osteotomy in younger, active patients with Uni-compartmental knee OA and malalignment (varus or valgus). Goal is to redistribute weight, bearing forces to preserve joint function and delay need for joint replacement.

c. Uni-compartmental Knee Arthroplasty (UKA) is limited to one compartment of the knee, with intact ligaments and good range of motion. Advantages are smaller incision, quicker recovery, retains more native bone but is not suitable for widespread OA.

d. Total Joint Arthroplasty (TJA) in case of severe, multi, compartment OA with pain and disability unresponsive to conservative care. Common sites include Total knee replacement (TKR), total hip replacement (THR). High success rates in pain relief and function restoration; lifespan of prosthesis is 15-20 years. Risks involve Infection, thromboembolism, loosening prosthesis or failure, need for revision surgery.

e. Joint Fusion (Arthrodesis) in selected cases with severe pain and instability where arthroplasty is not feasible (e.g., small joints of the hand/wrist, failed prior surgery). However, it eliminates joint movement, potentially affecting function.

ASSESSMENT OF RESPONSE

Regular monitoring is essential to evaluate treatment effectiveness, detect adverse effects early, and decide when to step, up or step, down therapy. The frequency and depth of follow, up depend on disease severity, treatment type, and comorbidities.

Category	Timing / Criteria	What to assess	Action / Next step
Timing of review	Initial follow, up	4-6 weeks after starting or changing therapy	Reassess pain, function, tolerance
	Stable patients	Every 3-6 months	Maintain plan; reinforce exercise/adherence
	High-risk (uncontrolled pain, long-term NSAIDs, major comorbidities)	More frequent visits	Closer monitoring; risk mitigation
	Post, injection	2-4 weeks after Intra-articular steroid	Check response, adverse effects, flare plan
Domains to assess	Symptom control	Pain (VAS/NRS), stiffness, fatigue; scores: WOMAC, Lequesne	Track trends; set targets
	Function	ADLs: walking, stairs, self, care; ROM, mobility	Align rehabilitation goals; adjust physio
	Adverse effects-NSAIDs	GI, renal, cardiovascular	Gastroprotection, dose adjust/switch/stop
	Adverse effects-Steroid injections	Local infection, post, injection flare, hyperglycemia	Asepsis, glucose check in diabetes, limit frequency
	Adverse effects-Opioids	Sedation, constipation, dependence	Avoid/initiate taper; bowel regimen if needed
	Adherence	Exercise, weight management, meds	Simplify regimen; coaching; barriers removal
	Patient satisfaction	Willingness to continue plan	Shared decisions; adjust to preferences
Treatment adjustment	Inadequate response	Persistent symptoms or poor function	Optimize/switch NSAID; add non, drug therapies; consider joint injection; treat comorbidities (obesity, DM, HTN)
	Adverse effects	Drug intolerance/toxicity	Taper, switch, or use safer alternatives
Step-Up	Any of the following	Moderate-severe pain despite optimal non, drug + first-line drugs; decline in function; radiographic progression with symptoms	Escalate therapy; consider injections; refer for surgical evaluation if refractory

Step-Down	All suitable	Stable ≥3-6 months; less need for analgesics; patient preference to minimize meds	Taper to lowest effective regimen; maintain rehabilitation/lifestyle
Referral to higher care	Indications	Rapid deterioration or major functional loss; poor response to conservative care; recurrent effusions, deformity, or surgical consideration	Refer to specialist/tertiary center; pre, op optimization if surgery likely

PROGNOSIS AND PROGRESSION

OA is a chronic, slowly progressive disorder of synovial joints. Although it cannot be cured, timely diagnosis, consistent management, and targeted lifestyle changes can substantially reduce symptoms, slow structural deterioration, and maintain function for many years. The course of OA varies widely between individuals, some experience minimal progression over decades, while others show steady worsening of pain, stiffness, and functional limitation. Importantly, radiographic severity does not always align with symptom burden; some patients with advanced imaging changes report little discomfort, whereas others with early disease may have significant pain and disability. Worse prognosis is seen with older age, obesity, malalignment, prior injury, high-impact loading, and higher radiographic grade. Better outcomes: early diagnosis, exercise and weight control, good pain plans, prompt flare management. With consistent non-surgical care and surgery when needed many people maintain function for years.

Referral to Specialist / Tertiary Care

Referral level	When to refer (triggers)	Indications/examples	What to send with referral
Primary to Secondary care	Diagnosis uncertain or atypical features	Suspect inflammatory arthritis, crystal arthropathy, avascular necrosis	Working diagnosis and differentials
	Symptoms persist/worsen despite 3-6 months of lifestyle, physiotherapy, and first-line meds	Ongoing pain, stiffness, flares limiting ADLs	Treatments tried (drugs, doses, duration), response/side effects
	Progressive functional limitation	Impact on work, mobility, self, care	Functional status (WOMAC/ Lequesne/VAS/NRS), ROM findings
	Recurrent/persistent effusions or concern for infection/inflammation	Hot joint, aspiration considered	Relevant labs (ESR/CRP, RF/anti, CCP, urate), aspiration results if any
	Imaging needed but unavailable in primary care	X-ray for staging/ confirmation	Imaging requests or any existing reports

	Injection indicated but not feasible safely in primary care	Corticosteroid or hyaluronic acid	Reason injection needed; contraindications checked
Secondary to Tertiary/ Orthopedics	Severe OA refractory to maximal conservative therapy (incl. injections)	KL grade 3-4; Lequesne ≥8 with significant disability	Full conservative care record, injection details, timelines
	Structural deformity/instability/ severe malalignment	Varus/valgus, instability affecting function	Latest weight, bearing X-rays; any MRI/US if done
	Candidate for surgery after counseling	TKA/UKA, osteotomy, arthrodesis considered	Comorbidity list, meds (anticoagulants, steroids), anesthesia risks
	Complex comorbidities need multidisciplinary optimization	Cardio, renal, metabolic risk, frailty, diabetes control	Recent labs (renal, HbA1c), cardiology/other clearances if available
	Diagnostic uncertainty persists after secondary review	Overlap pathology	Summary of prior specialist opinions and pending questions

COMPLICATIONS

OA can lead to a range of complications that affect physical function, overall health, and quality of life. These complications may result from the disease process itself, secondary effects of reduced mobility, or adverse outcomes of treatment.

Category	Key Complications
Joint, Related Complications	■ Progressive cartilage loss- deformity (varus/valgus knees), instability ■ Reduced ROM and stiffness-risk of contractures ■ Recurrent effusions- swelling, pain, mobility loss ■ Joint instability- ligament laxity, fall risk
Functional & Musculoskeletal	■ Muscle weakness/atrophy (quadriceps in knee OA) ■ Gait abnormalities → compensatory strain on spine/other joints ■ Secondary OA in unaffected joints
Systemic & Secondary Health Effects	■ Physical inactivity leads to weight gain, insulin resistance, metabolic syndrome ■ ↑ Cardiovascular risk (obesity, inflammation, sedentary lifestyle) ■ Falls & fractures from weakness, imbalance, instability
Pharmacological Complications	■ NSAIDs- GI bleeding, ulcers, renal/cardiovascular events ■ Intra-articular steroids- flare, rare infection, hyperglycemia, cartilage damage with repeated use ■ Opioids- dependence, sedation, constipation, fall risk

Psychosocial Complications	■ Depression & anxiety from pain/disability ■ Social isolation due to reduced mobility ■ Work disability- unemployment/early retirement

PREVENTION AND HEALTH PROMOTION

OA prevention focuses on reducing modifiable risk factors and promoting lifelong joint health, while health promotion aims to maintain mobility, independence, and quality of life in those with or at risk of OA.

Primary prevention (before OA): keep weight in check; prevent joint injuries (proper lifting, sports gear, workplace ergonomics); do low, impact aerobic plus strength exercise; correct malalignment/flat feet early with physio/orthotics.

Secondary prevention (early OA): diagnose early; start exercise, weight control, and appropriate meds; modify activities (avoid high, impact, use aquatic/low, load options); manage comorbidities like diabetes and metabolic syndrome.

Tertiary prevention (established OA): structured rehabilitation (physiotherapy/ Occupational Therapy) to maintain function; use canes/braces/orthotics as needed; address mood and social support; prevent falls with balance training and home safety.

Community/policy: run awareness drives on risk and early symptoms; workplace ergonomics programs; embed OA prevention in national non, communicable disease policies.

PATIENT EDUCATION

Patient education is a central component of OA management, aimed at empowering individuals to actively participate in their care and make informed decisions that improve long-term outcomes. Key objectives are:

- Understand the disease: OA is chronic and not curable, but symptoms are controllable.
- Set goals: less pain, better mobility, independence; aim to slow progression.
- Lifestyle: weight control, low, impact exercise, pace activities, protection of joints.
- Adherence: continue physio, exercises, and medicines even when you feel better.

- Red flags: new or sudden swelling, redness, warmth, severe pain, or fever → seek urgent care.
- Safe medicines: take as prescribed; don't double doses; avoid unsupervised NSAIDs.
- Emotional health: watch mood, stay socially connected, ask for support.
- Self, management: track symptoms, use diaries and support groups, make shared decisions.

Instructions to Patient / Caregiver

Do's	Don'ts
Take medications exactly as prescribed. Use NSAIDs for the shortest duration and always with food. Report side effects early.	Don't self, adjust doses or combine painkillers without medical advice. Don't overuse NSAIDs or opioids.
Stay active with joint, friendly exercise (walking, swimming, cycling, stretching, and muscle strengthening 2-3 times/week).	Avoid long inactivity, prolonged sitting, or high-impact exercises like running, jumping, or deep squats.
Maintain a healthy weight; even small reductions reduce joint load. Combine balanced diet with exercise.	Don't rely on "quick fixes" like crash diets or unproven supplements as substitutes for lifestyle change.
Protect joints with proper lifting techniques, activity pacing, and alternating tasks.	Don't overstrain joints with repetitive heavy lifting or kneeling.
Use assistive devices (cane, brace, walker) as instructed. Choose cushioned, supportive footwear.	Don't use ill, fitting devices or continue using broken/ unsuitable supports.
Apply heat before activity to ease stiffness and cold after activity to reduce swelling or flares.	Don't apply heat on acutely swollen joints or for prolonged periods that may burn skin.
Keep a record of symptoms, pain levels, and medication side effects. Report sudden changes promptly.	Don't ignore sudden swelling, redness, warmth, severe pain, or inability to move a joint.
Maintain social activities, hobbies, and seek counseling/support groups for coping.	Don't isolate yourself or neglect emotional health.

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