Please may we have a new design?

Figure 1: The development of osteoarthritis. Reproduced from Nuki [with permission from Arthritis Research UK

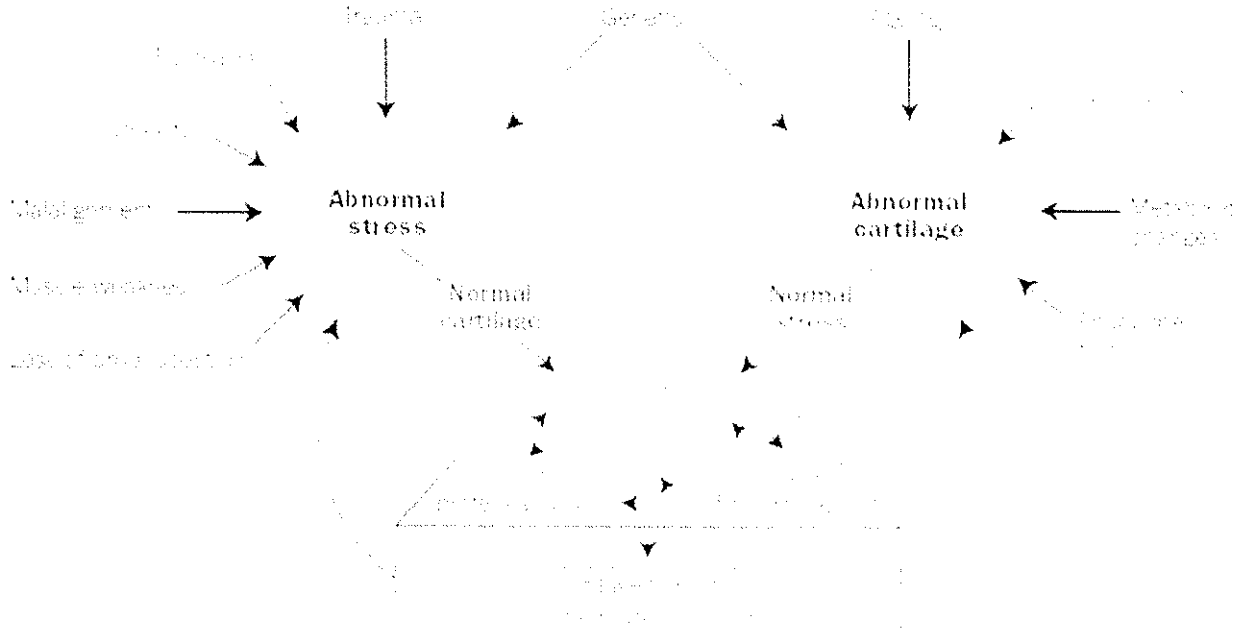
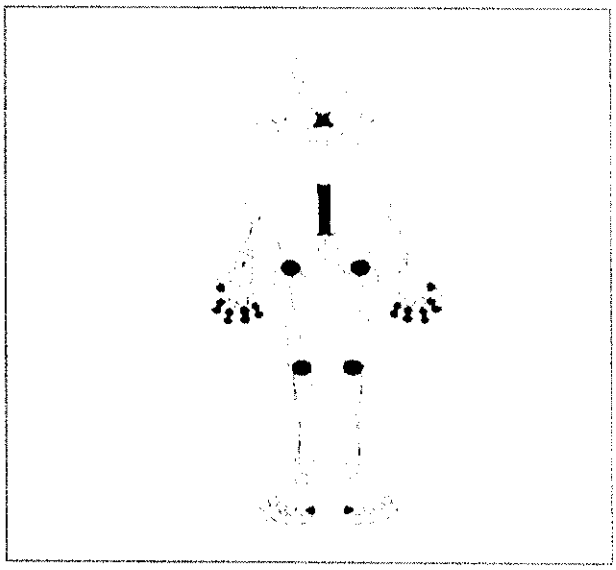


Figure 2: Joints commonly involved in osteoarthritis



KEY POINTS

- The prevalence of OA is increasing
- OA accounts for significant disability
- Multiple risk factors are associated with OA
- The whole joint organ is involved in OA
- Weight-bearing joints are commonly affected
- Secondary causes of OA should be excluded

of various biomechanical, biochemical, inflammatory and immunologic factors.[17] The pathological process involves fragmentation and thinning of articular cartilage, thickening of subchondral bone and cyst formation, development of osteophytes, variable degrees of inflammation, ligamentous laxity and muscle weakness. The interplay between various local and systemic factors resulting in the development of OA is depicted in Figure 1.[18]

OA commonly affects weight-bearing and stressed joints (hips, knees and 1st metatarsophalangeal joints), small hand joints and the cervical and lumbar spine (Figure 2). The spine will not be discussed for the purposes of this article. The recognised subsets of hand OA now include nodal (presence of Heberden's and Bouchard's nodes), generalised (hand OA, with OA at other sites), thumb-base, and an erosive variant characterised by subchondral erosions and an inflammatory component.[19]

Most OA can be categorised into primary and secondary variants. Primary (idiopathic) OA occurs in previously undamaged joints and can be further classified as localised (1 or 2 sites) or generalised (≥ 3 sites). Ageing and genetic factors are the key players in the development of primary OA. Secondary OA is associated with well-recognised causes such as trauma, anatomic abnormalities, rheumatoid or other inflammatory arthritides, and metabolic or endocrine disorders (Table I).[20]

(e.g. Vimovo® (naproxen/esomeprazole)

function.[36] Additional concerns about paracetamol's narrow therapeutic margin for liver toxicity, has prompted the FDA to recently recommend that daily doses should be less than 4 g/day. Despite these dose-related concerns, paracetamol still remains the safest first line option.

Opioids. The addition of weak opioids (e.g. tramadol, codeine) should be considered if paracetamol or non-steroidal anti-inflammatory drugs (NSAIDS) are ineffective, poorly tolerated or contraindicated. Stronger opioids (e.g. fentanyl) should only be reserved for severe refractory pain in exceptional circumstances.[25,26,29] Narcotic analgesics should be used with caution in the elderly, as they may cause side effects like constipation, confusion and dizziness.

Non-steroidal anti-inflammatory drug. Oral NSAIDs, including selective and non-selective COX-2 inhibitors (COXIBs), can be added or substituted in patients who respond inadequately to paracetamol.[25-31] All NSAIDs/COXIBs are about equally effective for pain relief in OA, however patient responses may vary considerably to specific agents. The choice of drug should be carefully based on the patient's age, comorbidities, side effect profile and cost. All oral NSAIDs should be used at the lowest effective dose for the shortest possible time.

Major concerns include their potential to cause serious GIT, renal and cardiovascular complications. The EULAR[25-27] and OARSI[29] guidelines recommend that patients at risk of GIT toxicity are to use either a COX-2 selective inhibitor or a non-selective NSAID coprescribed with a proton pump inhibitor (PPI) or misoprostol for gastroprotection. Of note is that many elderly patients are in the at-risk GIT category and have cardiovascular disease necessitating aspirin use. The protective benefits of the COXIBs are, unfortunately, negated if the patient takes aspirin. For this reason, and based on a cost-effective analysis, the NICE guidelines suggest routine addition of a PPI to both non-selective NSAIDs and COX-2 inhibitors.[31] Overall, it is preferable to avoid all NSAIDs and consider other analgesics in any patient with a significant history of peptic ulceration or bleeding.

The risk of atherothrombotic events is common to both COXIBs and the traditional NSAID. It appears that the risk may be lower with naproxen.[37] The safe recommendation is that all NSAIDs should be avoided in patients with ischaemic heart disease or stroke. In patients with significant risk factors for heart disease (e.g. hyperlipidaemia, hypertension, diabetes, smoking), these agents should be used with caution.

NSAIDs are contraindicated in patients with renal dysfunction. Elderly patients often have multiple comorbidities and drug interactions and renal function should be carefully monitored if NSAIDs are prescribed. Any patient with a history of a bleeding diathesis or on anticoagulation therapy should avoid NSAIDs.

(e.g Arthrotec® (diclofenac/misoprostol)

Tricyclic compounds and antidepressants. Chronic pain in OA is often accompanied by a vicious cycle of poor sleep, anxiety and depression. The use of amitryptyline at a dose of 10-25 mg at night may be of benefit. Antidepressants (e.g. selective serotonin reuptake inhibitors) may be added in selected patients.

Topical treatments. Topical NSAIDs or capsaicin are effective as adjunctive treatments for hand and knee OA.[27,29,31]

Intra-articular injections. Intra-articular (IA) injections of long-acting corticosteroids may be considered for patients experiencing acute flares with knee effusions, hip OA (given under ultrasound or X-ray guidance) or CMC joint OA not responding to analgesics and NSAIDs.[25-27,29] IA steroids give rapid pain relief and the effect may last for four to 12 weeks. Proper aseptic technique should be employed.

Hyaluronic acid is a glycosaminoglycan found in synovial joint fluid that allows viscous lubrication. IA hyaluronan gives a delayed onset but more prolonged duration of pain relief compared to IA corticosteroids. They are very costly and repeated injections are often required for symptomatic relief. Most guidelines give guarded recommendations for their use in knee or hip OA, however NICE does not support this use.

Surgery. Referral for joint replacement surgery should be considered in patients who experience persistent pain and reduced function that are refractory to non-surgical therapies, and which impact markedly on their quality of life.[25,28,31] Hip and knee arthroplasties should not be delayed until there is prolonged functional limitation and generalised deconditioning. Unicompartmental knee replacement may be considered for patients with involvement of one tibiofemoral compartment. In younger, more mobile, symptomatic patients, corrective osteotomies and joint-preserving procedures (e.g. hip resurfacing) may delay the need for total joint replacement.[29] Current evidence does not support arthroscopic lavage and/or debridement as part of unselected knee OA treatment.[30,38] Several surgical options are available for severe thumb base OA when conservative therapies have failed.[2]

KEY POINTS

- Treatment should be individualised
- Consult allied health professionals to deliver effective symptomatic therapy
- Patient education, exercise and weight loss are core treatments
- Paracetamol is recommended as first-line analgesic
- Topical preparations are useful adjuncts for hand and knee OA
- Prescribe NSAIDs with caution, based on patient risk factors
- NSAIDs should be given at the lowest effective dose for the shortest time
- Weak opioids can be considered as alternative analgesics
- IA corticosteroids can be used for flares and provide rapid pain relief
- Refractory pain and loss of function are indications for surgical referral