OSTEOARTHRITIS

Osteoarthritis (OA) was previously thought to be a degenerative joint disease, a consequence of ageing, which led to its being described as a 'wear and tear' disease. It is now realised that OA results from a combination of factors that include genetic predisposition, joint integrity, local mechanical forces and inflammatory responses.

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OA has been divided into primary or secondary type. The joint involvement in the primary form often affects the hands, feet, knee, hip and spine. Secondary OA often follows on an insult to a joint, e.g. trauma, inflammation from rheumatoid arthritis, joint sepsis or crystal deposition.

PATHOGENESIS

The primary pathology in OA is articular cartilage failure. Dysfunction of the chondrocyte is thought to be responsible for the development of the osteoarthritic process.

The initiating mechanism is damage to normal cartilage by physical forces, which can be either single events (macrotrauma) or repeated microtrauma.[1] Cartilage loss is caused by chondrocytes reacting to these insults, both by releasing degradative enzymes (proteases, collagenases and inflammatory cytokines) and by exhibiting an inadequate repair response.[2]

Risk factors for OA therefore include the following:

- Age. The National Health and Nutrition Examination Survey found the prevalence of OA to be less than 0.1% in those aged 25 - 34 years compared with 80% in people over the age of 55.[3]
- Female gender. The reason for the increased risk in women has not been fully explained, but hormonal effects have been proposed.
- Obesity. This is an independent risk factor for knee, hip and hand OA.[4] The increased risk of OA caused by obesity can be decreased by weight loss.
- Muscle weakness. Weakness of the quadriceps is associated with the initiation/progression of knee OA.[5]
- Exercise and proprioceptive defects. Bilateral abnormalities of joint proprioception have been demonstrated in patients with unilateral knee OA.[6] Proprioceptive deficits slow protective periarticular muscular reflexes and therefore increase the risk of cartilage damage imposed by strenuous loads.
- Calcium crystal pyrophosphate deposition disease (CCPD). There is a relationship between the radiographic severity of OA and the incidence of calcium crystals in the synovial fluid.

DIAGNOSIS

The diagnosis of OA remains a clinical one, based on patient history, examination and radiographic findings. There is a lack of diagnostic physical or laboratory findings. Radiographic changes and patient symptoms cannot be correlated to any of these.

Clinical features

- Age – predominantly after the age of 40 years.
- Symptoms – joint pain, early morning stiffness of less than 30 minutes, loss of mobility.

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The aims of management are to control pain, improve quality of life and prevent progression of the disease.

- Examination – joint tenderness to palpation, bony enlargement, crepitus, decreased range of movement, joint deformity.
- Radiographic – joint space narrowing, osteophytes, subchondral cysts, subchondral sclerosis (Figs 1 and 2).
- Synovial fluid analysis – non-inflammatory, clear, no crystals, normal viscosity – white cell count < 2 000/µl.
- Joints affected – hand distal interphalangeal (DIP) joints and proximal interphalageal (PIP) joints, first metacarpophalangeal (MP) joint of the foot, cervical and lumbar spine, hip, knee, ankle subtalar joint.
- Joints uncommonly affected – wrist, elbow, shoulder, hand MP joints.

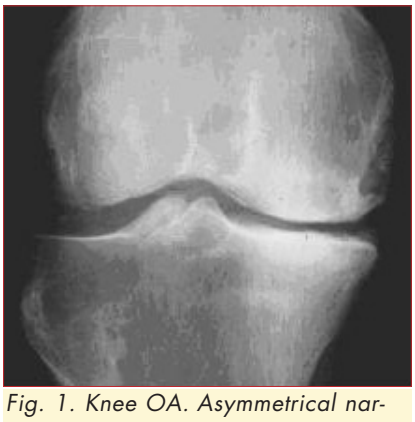

Fig. 1. Knee OA. Asymmetrical narrowing of the joint space with dense sclerosis of the subchondral bone and osteophyte formation. (Grassi W, Department of Rheumatology, University of Ancona, Italy.)

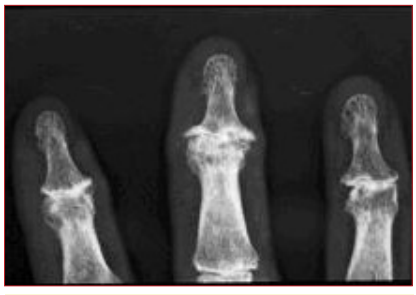

Fig. 2. Erosive OA of the distal interphalangeal joints. (Trotta F, Department of Rheumatology, University of Ferrara, Italy.)

Differential diagnosis

- CCPD.
- Rheumatoid arthritis (RA) – warm, swollen joints that feel soft. Hand DIP joints are not involved, early morning stiffness lasts for hours, presence of raised inflammatory markers and rheumatoid factor.
- Exclude sepsis when a single OA joint presents as an acutely painful synovitis.

TREATMENT

The aims of management are to control pain, improve quality of life and prevent progression of the disease. These aims should be explained to the patient. Treatment success depends on patient education and combining non-pharmacological and pharmacological therapies. Surgery may be required in some patients. It is reserved for patients who suffer intractable pain failing treatment with all other modalities as well as for those who suffer loss of function. Psychosocial support has been shown to improve pain score and functional status.[7]

Non-pharmacological therapy

- Obesity. A relationship exists between obesity and OA. For every 5 kg increase in weight the odds ratio for radiographic knee OA are increased by 1.36.[8] Obesity is associated with a relative risk of 3 for the development of hand OA, suggesting that it is causally related, rather than there being a concomitant increase in joint loading. Obesity is the most easily modifiable risk factor associated with the development of OA; patients should be counselled to lose weight and even modest weight loss is beneficial.
- Rest. Pain after prolonged joint activity improves with rest. Rest is however only recommended for short periods (12 - 24 hours), after which exercise should resume. Rest exceeding 24 hours increases muscle weakness and decreases joint mobility.
- Exercise. The relationship between exercise and OA is controversial. Low-impact, appropriate exercise, e.g. walking, cycling and swimming, is advised to decrease joint loading. Improving muscle strength around the joint involved has been shown to decrease pain and increase functional outcome.

Pharmacological therapy

The principal reason for using pharmacological agents is pain relief in patients who do not respond to non-pharmacological therapy.

Medication available includes the following:

- Paracetamol, at doses of up to 4 g/day, is the drug of choice for pain. A 2004 meta-analysis of 10 randomised trials found evidence that paracetamol was superior to placebo.[9] Side-effects of paracetamol are mild. Hepatotoxicity may occur in patients who suffer from chronic alcohol abuse, particularly if the transaminases are elevated.
- Opioid analgesics, including codeine, should be avoided for long-term use.
- Tramadol, alone or in combination with paracetamol, may be useful.
- Non-steroidal anti-inflammatory drugs (NSAIDs) are indicated in patients who fail to respond to the above pharmacological and non-pharmacological interventions. The use of these drugs is often limited by their toxicity, which includes peptic ulcer disease, renal and hepatic dysfunction and CNS disturbances in the elderly. The selective COX-2 inhibitors display equal efficacy to traditional NSAIDs but are associat-

ed with lower gastrointestinal tract toxicity. Recent evidence has brought into question the long-term cardio/cerebrovascular safety of these agents when used in high doses.

- Intra-articular glucocorticoids. A recent meta-analysis found that patients who received glucocorticoid knee injections were twice as likely as those who received placebo to have short-term improvement.[10] The use of aseptic techniques in performing this procedure is essential.
- Disease-modifying OA drugs (DMOADs). These potential disease-modifying drugs which either prevent cartilage breakdown or facilitate repair have been described and include tetracyclines, glucosamine sulphate (1 500 mg/day) and chondroitin sulfate. All show modest effects, but the trials to date have been flawed. Studies are currently underway to determine their efficacy.

Drug therapy for symptomatic relief of pain includes the use of analgesics such as paracetamol or NSAIDs, topical NSAIDs and intra-articular cortisone injections. NSAID use is often limited by toxicity, which includes peptic ulcer disease, renal or hepatic impairment and CNS dysfunction in the elderly.

References available on request.

IN A NUTSHELL

OA results from a combination of factors that include genetic predisposition, joint integrity, local mechanical forces and inflammatory responses.

Traditionally, OA has been divided into primary or secondary type.

The joint involvement in the primary form often affects the hands, feet, knee, hip and spine.

Secondary OA often follows on an insult to a joint, e.g. trauma, inflammation from rheumatoid arthritis, joint sepsis or crystal deposition.

The primary pathology in OA is articular cartilage failure.

Risk factors are age, female gender, obesity, muscle weakness, a combination of exercise with proprioceptive defects, and calcium crystal pyrophosphate deposition disease.

The diagnosis of OA remains a clinical one, based on patient history, examination and radiographic findings.

The aims of management are to control pain, improve quality of life and prevent progression of the disease.

Non-pharmacological therapy includes weight management, short-term rest and low-impact exercise.

Pharmacological treatment should start with paracetamol. Tramadol may be added and NSAIDs are sometimes used, but must be administered with caution, particularly in the elderly.

Intra-articular glucocorticoid therapy can be useful, as can disease-modifying drugs.

Surgery is reserved for patients with intractable pain in whom non-pharmacological and pharmacological management have failed, or who suffer loss of function.