Osteoarthritis and the inflammatory arthritides

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Abstract

This article aims to provide surgeons with a practical, clinical overview of different forms of 'arthritis' — a term encompassing most of the joint pathology causing joint symptoms or dysfunction. Conventionally, arthritis can be non-inflammatory (osteoarthritis) or inflammatory (crystal and autoimmune arthropathies). Septic arthritis is an important differential diagnosis when patients present with tender, swollen joints but is not covered here. Common symptoms and signs in patients with different types of arthritis are reviewed, as well as aetiology and pathogenesis. Non-surgical treatment is described, with particular reference to the inflammatory arthropathies since the new, effective biologic treatments are particularly important where surgery is planned or patients present with suspected sepsis. Diagnosis of inflammatory arthritis (particularly in children) may be delayed and in an era of effective treatment it is important that all clinicians involved in musculoskeletal medicine and surgery are aware of potential differential diagnoses for joint pain and deformity. Good communication between rheumatologists and surgeons in managing different forms of arthritis is especially important.

Keywords Ankylosing spondylitis; arthritis; gout; juvenile idiopathic arthritis; osteoarthritis; psoriatic arthritis; rheumatoid arthritis; spondyloarthropathy; systemic lupus erythematosus

Osteoarthritis

Osteoarthritis (OA) is not a single disease or process; rather it is the outcome of the range of processes leading to pathological, structural and eventually symptomatic failure of one or more synovial joints. Recently a paradigm shift has moved the conception of OA from an exclusively degenerative condition of old or worn joints to an emphasis on OA as a dynamic, remodelling and regenerative condition. OA is the commonest type of arthritis and the leading cause of disability in those over the age of 65 years in developed countries. It may be classified as primary (idiopathic) or secondary to other processes such as trauma, congenital/developmental, mechanical or local factors (for example obesity or hypermobility) or as a sequelae of other inflammatory arthritides.

OA involves all tissues in the joint — initially there is loss of proteoglycan from the matrix of articular cartilage resulting in fibrillation, fissuring and degeneration. In more advanced disease, cartilage loss is such that the articulating surface is subchondral bone (eburnation). There is increased bone remodelling with subchondral osteosclerosis and cyst formation, articular surface deformity and osteophyte formation. Varying degrees of synovial inflammation and ligament degeneration may also occur and OA is accompanied by peri-articular muscle wasting and biomechanical changes. These pathological processes lead to the characteristic X-ray (XR) features (Figure 1). There is often poor correlation between XR changes and symptoms. OA has multiple aetiological factors with gender, age and genetic factors increasing susceptibility and more local factors such as joint biomechanics, obesity, trauma and muscle weakness determining the site and severity of the disease.

OA can affect any synovial joint and typical presenting features are mechanical pain in joints (worse with activity or weight bearing), joint stiffness and deformity. Spinal OA may cause neurological symptoms such as radiculopathy and spinal stenosis.

Treatment is currently symptomatic — medical therapies aim to control pain and physiotherapy maintains mobility, muscle strength and biomechanical integrity of the joint. In advanced disease with joint failure (disabling joint pain — particularly nocturnal — and loss of joint function and deformity) management is surgical. With better understanding of collagen matrix factors it may be that future disease-modifying medical therapies targeted at underlying pathological processes will become available. There is increasing evidence that exercise has a crucial role to play in symptomatic management and explaining the importance of reducing obesity and promoting exercise to patients may help prevent OA progression.

Crystal arthropathies

Arthritis, both acute and chronic, can be caused by crystal deposition in joints. Gout is characterized by hyperuricaemia and monosodium urate crystal deposition; typically this is in peripheral joints (especially the great toe, but commonly also in the knee and ankle, wrist and hand) and in those joints affected by OA. The crystals trigger acute episodes of cytokine release and consequent neutrophilic inflammation. The patient experiences

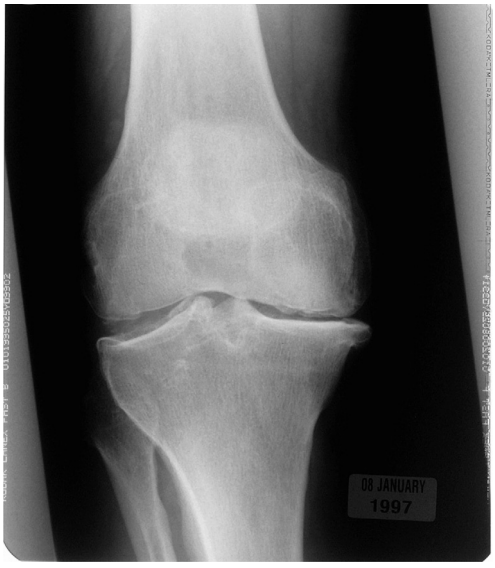

Figure 1 X-ray of right knee showing severe joint space narrowing with osteophytes and subchondral cysts.

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excruciating joint pain which typically starts in the early hours of the morning. The affected joints are often swollen and erythematous with patients unable to weight bear on them. Where hyperuricaemia and gout are chronic, urate may be present as macroscopic deposits (tophi) around joints or in cartilaginous structures such as the ear. Urate may also accumulate in the kidney causing at micro-level, urate nephropathy and at macro-level, urate calculi.

Urate is a byproduct of purine metabolism and normally excreted through the kidneys. A minority of people with gout produce excess urate but much more commonly gout results from the under-excretion of urate. This may be due to renal failure, intercurrent drug therapy, particularly aspirin and thiazide diuretics and alcohol intake. Hyperuricaemia is probably under-recognised and not everyone with hyperuricaemia has gout. Diagnosis of acute gout is on typical history and presentation, high serum urate (although this may paradoxically lower in acute attacks) and by demonstration of the typical negatively birefringent needle-shaped urate crystals in joint aspirate under polarised light microspcopy. Treatment involves lifestyle factor modification to avoid high-purine foods, for example meat and shellfish, reduce alcohol and fructose intake (fructose increases serum uric acid by increased purine breakdown and increased purine synthesis) and avoid obesity. Precipitating medicines such as aspirin and diuretics should be avoided but this is often not possible. Acute gout is treated symptomatically by aspiration and injection of affected joints with corticosteroids and systemic administration of non-steroidal anti-inflammatory drugs (NSAID), low-dose colchicine or low-dose oral prednisolone (particularly where patients have many co-morbidities and non-steroidal anti-inflammatory drugs (NSAIDs) or colchicines are contraindicated). Long-term treatment of hyperuricaemia is with xanthine oxidase inhibitors such as allopurinol or febuxostat which act to reduce serum urate levels. On initiation such treatment may paradoxically flare gout and must always be started alongside prophylactic treatment such as NSAID or low-dose colchicine.

Calcium pyrophosphate (CPP) arthropathy is the result of calcium pyrophosphate deposition in joint tissues which triggers inflammation in a similar way to gout. This acute arthritis is commoner in older people and often poly-arthritic. It is associated with chondrocalcinosis on XR particularly in the knee and at the wrist (Figure 2) and diagnosis is on clinical presentation, typical XR appearances and the demonstration of positively birefringent, rhomboid crystals under polarized light. Aetiology is unknown but CPP disease or pseudogout may be associated with haemochromatosis and acromegaly and in younger patients these conditions should be actively excluded. Hydroxyapatite is the third type of crystal arthropathy, commonest in older women and recognised as a cause of destructive shoulder arthritis classically described as the 'Milwaukee shoulder syndrome'. In this syndrome there often is significant humeral head destruction, CPP deposits and rotator cuff tears. Treatment of both is symptomatic with NSAIDs, intra-articular steroid injections, physiotherapy and surgery where joint failure supervenes.

Rheumatoid arthritis

Rheumatoid arthritis (RA) is a destructive, auto-immune arthritis that affects 1% of the adult population; women three times more commonly than men. It can occur at any age, but most often between the ages of 40–50. Risk factors include presence of the 'shared epitope' (a sequence of five amino acids on the HLA-DRB1 chain — chromosome 6), a personal or family history of auto-immune disease, and smoking.

RA principally affects synovial joints, but is a systemic inflammatory disorder affecting structures beyond the joints including for example, skin (rheumatoid nodules, pyoderma

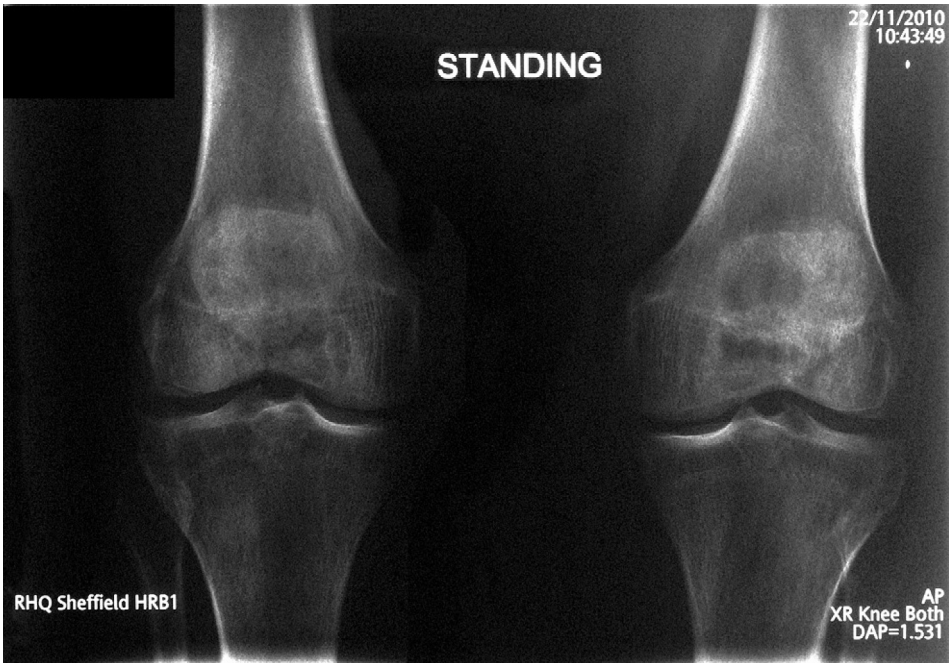

Figure 2 X-ray of both knees with typical chondrocalcinosis in menisci, spiking of tibial spines and preservation of joint spaces.