Osteoarthritis and the inflammatory arthritides

Robert D Sandler
Lisa Dunkley

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 in detail 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; spondyloarthritis; 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 most common 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 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 genetics 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), stiffness and deformity. Spinal OA may cause neurological symptoms such from 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. Groups investigating the role of inflammation in the pathogenesis of OA have identified some novel therapeutic targets but current trial data show limited efficacy. 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, where the crystals can precipitate at cooler temperatures. Classically this will manifest in the great toe, but also commonly in the knee and ankle, wrist, hand and any joints affected by OA. The crystals trigger acute episodes of cytokine release and consequent neutrophilic inflammation. Patients frequently report 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 by-product 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, concurrent drug therapy, particularly aspirin and thiazide diuretics and alcohol intake. Hyperuricaemia is probably underrecognized 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, as urate crystals precipitate in the extravascular space)

Robert D Sandler *BMedSci (Hons) MBChB MRCP is a Core Medical Trainee 2 within the South Yorkshire Foundation School, UK. Conflicts of interest: none.*

Lisa Dunkley *PGcertMedEd FHEA FRCP is a Consultant Rheumatologist at the Royal Hallamshire Hospital, Sheffield, UK. Conflicts of interest: none.*

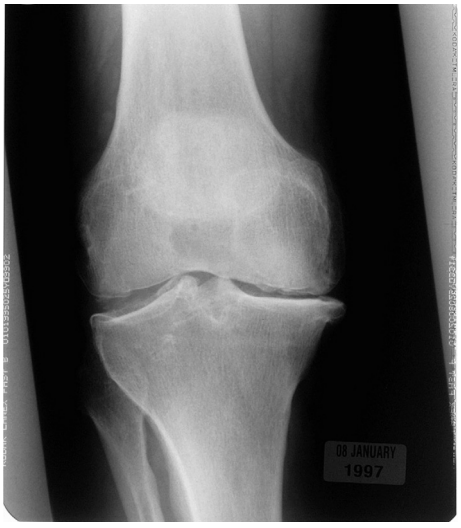

Figure 1 X-ray of right knee showing severe joint space narrowing with osteophytes and subchondral cysts.

and by demonstration of negatively birefringent, needle-shaped urate crystals in joint aspirate under polarized light microscopy. A key differential for an acute monoarthritis is septic arthritis, which would be confirmed on synovial fluid microscopy and culture, highlighting the importance of joint aspiration.

Treatment of gout involves lifestyle modification – to avoid high purine foods e.g. 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 comorbidity and NSAID or colchicine are contraindicated). Long-term treatment of hyperuricaemia is with xanthine oxidase inhibitors such as allopurinol or febuxostat, which act to reduce serum urate levels. Lesinurad, a uric acid transport inhibitor, has just been licensed as an add-on therapy for patients whose urate levels are unsatisfactory despite first-line therapy, though it is not yet recommended by NICE. Recent work has demonstrated involvement of interleukin-1 (IL-1) pathways in gout and treatment with anakinra (IL-1 receptor antagonist) shows promising results. It is worth noting that, on initiation, these treatments may paradoxically trigger a flare of gout and must always be started alongside prophylactic treatment such as NSAID or low-dose colchicine for around 3 months.

Pseudogout or calcium pyrophosphate dehydrogenase (CPPD) 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 the wrist (Figure 2). Diagnosis is based on clinical presentation, typical XR appearances and the demonstration of positively birefringent, rhomboid crystals from joint aspirate under polarized light microscopy. The aetiology is unknown but CPPD can be associated with haemochromatosis and acromegaly, and in younger patients these conditions should be actively excluded.

Hydroxyapatite is the third type of crystal arthropathy, most common in older women and recognized as a cause of destructive shoulder arthritis, classically described as the 'Milwaukee shoulder syndrome'. In this syndrome there is often significant

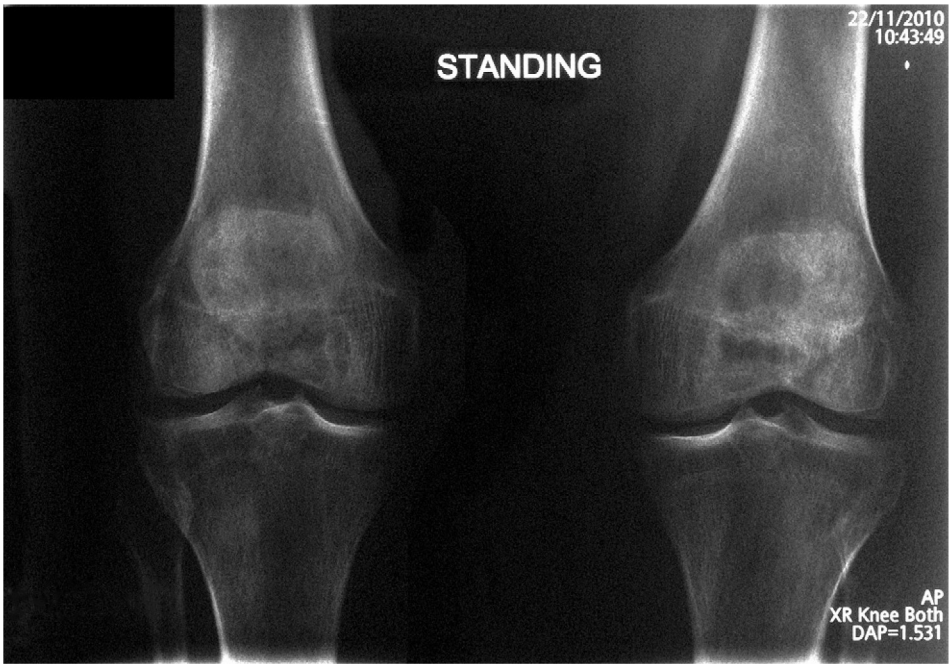

Figure 2 X-ray of both knees with typical chondrocalcinosis in menisci, spiking of tibial spines and preservation of joint spaces.

