INTRODUCTION: THE IMPORTANCE OF IMAGING

Rheumatoid arthritis (RA) is a chronic progressive systemic inflammatory disease of synovial tissue. Its aetiology is poorly understood, although autoimmune and biomechanical factors are implicated. The overall incidence in the general population is in the region of 1% (Symmons et al. 2002, Lawrence et al. 1998). Females are affected approximately three times more commonly than males (Symmons et al. 2002).

Approximately 90% of patients have involvement of the foot or ankle at some point during their disease and 20% of patients present with foot problems (Resnick 2002). Symptoms of the foot usually precede the hands and are affected more severely. Therefore, not only are the feet frequently affected by RA, but also the symptoms often predate other areas.

Plain film radiography is the mainstay of imaging of the ankle and foot. In recent years the use of other imaging modalities such as ultrasound (U/S) and magnetic resonance imaging (MRI) have become more important and provide an improved abnormality detection rate due to their ability to image in multiple planes rather than the 2D representation seen with plain film (McGonagle et al. 2001). They are important in not only the diagnosis, but also the follow-up and evaluation of RA (Ostergaard and Szkudlarek 2003). Imaging also plays a pivotal role in diagnosis confirmation in patients with confusing presentations or negative serology.

Because bone and cartilage destruction is largely irreversible, a definite radiological diagnosis can drastically alter the patient’s prognosis. The goal of the rheumatologist is to arrest the disease before this stage.
Clinical examination is often negative in the early stage of RA. MRI and U/S can both detect subclinical disease (Wakefield et al. 2004). Twenty per cent of patients presenting with sub-clinical disease can be shown to have synovitis at presentation, allowing earlier treatment options (Wakefield et al. 2004). Synovitis and erosive change are used to arbitrate diagnosis and treatment protocols. They are the hallmarks of disease progression and severity.

Present-day treatment with pharmacological agents such as biologics is dependent on a firm diagnosis of erosive disease or synovitis. There is a trend to treat rheumatoid disease earlier and more aggressively, often with expensive pharmacological agents, in order to improve outcome levels and reduce morbidity. Disease-modifying drugs can then be initiated early in a preventative and potentially curative manner. Imaging can help to confirm the diagnosis at the sub-clinical stage (Ostergaard et al. 1996). The most appropriate modality depends on the stage of disease and clinical question to be addressed.

Periarticular soft-tissue disease is a consequence of the joint inflammatory process; MRI and U/S are also of value in the assessment of the extrarticular pathology. This chapter describes the common pathological changes and the techniques used to assess rheumatoid disease of the foot and ankle.

**HOW TO LOOK FOR RHEUMATOID DISEASE OF THE FOOT AND ANKLE**

**Intraarticular disease**

**How to detect erosions**

Bone erosions are cortical breaks that are the cornerstone of the diagnosis of inflammatory arthropathy and one of the most important prognostic indicators (Winalski 1996). Erosions are, however, only identified in 40% of new-onset RA and so cannot be relied upon to give a definitive diagnosis (Conaghan 2003).

Erosions on radiographs are characterized by cortical discontinuity with diagnosis reliant on a tangential orientation of the X-ray beam to the eroded cortex (Fig. 5.1). Where erosion is not projected tangentially, a clear cortical break is not demonstrated. These en face erosions are seen as focal areas of reduced bone density (Fig. 5.2). Erosion activity can be determined by assessing the erosion margin. Inactive and active erosions are distinguished by sclerotic and non-sclerotic margins (Fig. 5.1). Plain radiography is, however, an insensitive tool in the detection of erosions, which are only detected when a substantial amount of bone is destroyed. It may take months or years for the plain film to detect the osseus changes of the disease.

This lack of sensitivity results in a significant temporal delay between presentation and radiological diagnosis, a delay that is potentially detrimental to patient morbidity. Poor peripheral bone density or overexposure of the radiograph can result in the artefactual impression of cortical disruption.

MRI is, however, considered the gold standard in the detection of erosive disease (Tehranzadeh et al. 2004). MRI detects approximately three times as many erosions than the plain radiograph and at an earlier stage. The multiplanar imaging utilized in MRI is, in part, responsible for the improved erosion detection rate (McQueen 2000). It is only on rare occasions that plain film detects erosion not visualized with MRI.

It is apparent that only one in four erosions detected by MRI actually progress to erosions seen on the plain radiograph. However, if a large amount of erosion is visualized on MRI then the likelihood of later plain film erosive detection is higher (McQueen et al. 2001, Ory, 2003).

MRI produces more false positives due to the multiple planes. Contrast enhancement should routinely be employed to increase diagnostic accuracy.

Bone-marrow oedema is often seen as a precursor of future erosion and can be used to predict change (McQueen et al. 2003). Erosions appear as areas of cortical discontinuity on MRI. They are of low signal on T1 weighted in the marrow adjacent to the bony surface or under the cartilage. The contents are fluid or synovium and are bright on T2. Erosions are seen to enhance with gadolinium contrast. Longitudinal follow-up of erosions can, however, be difficult due to repositioning error (Fig. 5.3) (Winalsiki 1996).

Ultrasound is also of use in the detection of erosions. It has the advantage of being a multiplanar modality and is imaged in real time. It is best utilized in site-specific joints, when only one or two joints require to be imaged and as an adjunct to plain radiography (Fig. 5.4).

It can detect 6.5 times as many erosions in 7.5 times as many patients in early RA compared with 3.4 times as many erosions in 2.7 times as many patients in late RA than plain film (Wakefield et al. 2000). In addition, it has been shown to be useful in site specific asymptomatic joints (e.g. MTP), which frequently reveal erosive change (Lopez-Ben et al. 2004).

**How to detect synovitis**

The histo-pathological hallmark of RA has been described in Chapter 1. Synovial inflammation is the primary pathological process. Synovitis predates bone damage and the amount of synovitis predicts future bone damage.
Figure 5.1  AP forefoot radiographs. This demonstrates typical rheumatoid changes in the forefoot mainly affecting the fourth and fifth MTP joints. (A) Shows a relatively inactive disease with well-defined somewhat sclerotic margins to the erosions. (B) Shows inactive disease characterized by decreased peri-articular bone density with ill-defined erosion.

Figure 5.2  AP radiograph of the 5th MTP joint showing the appearances of an en-face erosion. The base-line (A) shows the erosion as a focal lucency within the metatarsal head that enlarges on the follow-up film 6 months later (B).
Figure 5.3  MR demonstration of early erosion. (A) T2 fat suppressed coronal scan with synovitis and effusion within the joint associated with bone-marrow oedema. (B) T1 weighted coronal MR scanning shows loss of cortical definition with the area of bone marrow oedema in keeping with an associated erosion.

Figure 5.4  Sagittal power Doppler sonogram. There is disruption of the bone cortex in keeping with erosion. Hypoechoic synovitis is present adjacent to and within the erosion. This synovial tissue is vascularized as demonstrated by power Doppler.
Imaging of the synovium allows direct assessment of the therapeutically targeted abnormality. In addition to its ability to detect erosive change, MRI is considered the gold standard at detecting synovitis. It is able to detect the abnormally inflamed synovium before the irreparable cartilage destruction. It is significantly more sensitive than clinical examination and will show synovial pathology before a plain film (Goupille et al. 2001).

Normal synovium is difficult to differentiate from fluid. It does, however, have a very slightly lower signal on T2 and differentiation is possible on a heavily weighted T2 study. Synovium is of intermediate signal on T1.

Synovium enhances after intravenous injection of gadolinium. The degree of enhancement is proportional to its blood supply, the extracellular fluid volume and capillary permeability (Fig. 5.5). Optimal synovial enhancement occurs within 5 min. After this window, the effusion within a joint will start to enhance, which may overestimate the volume of synovium present. Synovial fluid enhancement occurs later than the synovium, which then plateaus and persists for about 1 h (Ostergaard and Klarlund 2001). Data from the joint, therefore, requires to be collected relatively early in order to be able to differentiate synovial tissue from synovial fluid. MRI can help characterize the nature of the synovitis with fibrous pannous enhancing more slowly on T1 weighted contrast enhanced dynamic sequences than acutely inflamed synovium.

The degree of enhancement of the synovium is a secondary indicator of synovial inflammation. Dynamic MRI allows evaluation of the degree and rate of synovial enhancement to be assessed. Early enhancement and high rate of enhancement of the synovium correlates well with inflammatory markers and with the histological assessment of synovitis. (Ostergaard et al. 1998).

Quantitative measurements can also be used to measure the volume of synovium within the capsule (Ostergaard et al. 1999). The volume can be measured by manual outline or computer processing (Ostergaard 1997). Both techniques are of use in the early detection, predictor of disease severity and follow-up to assess joint response and remission. The quantification of the synovitis correlates well with the clinical signs of inflammation, the histopathology assessed by biopsy and the rate of progression of the disease (Ostergaard et al. 1997, Ostergaard et al. 1999).

These qualitative and quantitative measures of synovial disease are, however, at present experimental techniques that are not yet utilized in routine clinical practice.

MRI scoring is a method of semi-quantifying disease severity on the basis of erosive disease, synovitis and bone oedema. Outcome determined by OMERACT is the recommended scoring system for synovitis and bone damage (Ostergaard et al. 2003). The scores correlate well with the clinical state and have become the gold standard in monitoring disease activity. These scoring systems have been developed for the hand, but are easily transferable to the assessment of metatarsophalangeal joint disease.

Ultrasound can also detect synovitis. Synovium is not seen at ultrasound unless it is abnormal. Synovial tissue, however, usually appears hypoechoic and is defined by OMERACT as 'hypoechogenic thickened intra-articular tissue that is non-displaceable and poorly compressible which may exhibit Doppler signal.' It can be differentiated from joint effusion by pressure that will disperse an effusion, but distort synovium (Fig. 5.6) (O’Connor 2002). Differentiating synovitis from fluid may, however, be difficult, although colour or power Doppler can be used to visualize the increased vascularity in hyperaemic synovitis (Rubin 1999).

The volume of synovium within a joint can also be assessed with ultrasound. It is limited, however, by technical factors and reproducibility. Doppler U/S, however, appears to provide a potential method of semi-quantitative assessment of synovial tissue, though the data are preliminary and sometimes conflicting (Szkudlarek et al. 2003, Szkudlarek et al. 2001, Newman et al. 1996).

U/S allows direct assessment of the synovium and can visualize erosions. It images in real time with high
resolution and has advantages over MRI in cost savings, multisite assessment and patient compliance, which make a compelling argument for the use of U/S in the assessment of RA (O'Connor 2002). U/S scoring systems are currently being evaluated by the OMERACT group.

**How to detect a joint effusion**

U/S and MRI can both detect joint effusions with high sensitivity. U/S, however, is deemed the gold standard in the accessible joint. Joint effusions are anechoic with distal acoustic enhancement. Joint motion is particularly important in the demonstration of small joint effusions. Active or passive joint movement whilst scanning causes redistribution of any fluid present and can push fluid into ultrasonically visible areas (O'Connor 2002).

Simple fluid is anechoic with no internal echoes. The fluid is compressible and can be moved with probe pressure and demonstrates distal acoustic enhancement with no demonstrable vascularity.

The presence of effusion is a sensitive predictor of joint disease, but is unfortunately completely non-specific (O'Connor 2002). The main therapeutic impact is in the exclusion of intra-articular fluid, making articular disease much less likely. This is especially important in the setting of infection where the absence of joint effusion effectively excludes septic arthritis. Ultrasound has a role in this setting allowing guided aspiration or wash-out of the joint to be performed, helping differentiate synovitis, complex fluid and infection.

**Extraarticular disease**

**How to detect tendon and ligament pathology**

Tendon pathology and tenosynovitis are commonly due to the direct effects of synovial inflammation. Eroded bone may also fray tendons by a process of attrition and periarticular inflammation, thus weakening ligaments and tendons. Tendon rupture is a serious consequence of tenosynovitis and may further exacerbate an already unstable joint.

U/S is an excellent tool in the assessment of the tendons particularly around the ankle (Martinoli et al. 2002, Rawool and Nazarian 2000). Full- and partial-thickness tears are readily diagnosed. Tendons should be examined routinely in both the transverse and longitudinal planes.

Tendons have an organized fibrillar appearance. Tendinopathy manifests itself as loss of the normal organized tendon structure with increased tendon thickness. Increasing amounts of glycoprotein matrix produce an increase in the water content of the tendon, fibroblast and tenocyte proliferation and neovascularization. The tendon becomes more hypoechoic due to the increased water content and heterogenous, tissue (O'Connor 2002). Neovascularity during the reparative process in tendinopathy can also be demonstrated with power or colour Doppler; excess pressure may, however, obliterate subtle blood flow. Neovascularization is related to an unfavourable outcome.

Tenosynovitis may be evident with fluid or synovial thickening within the synovial sheath. Artefacts are, however, common in musculoskeletal ultrasound. Anisotropy and beam edge artefacts are important causes of potential error. Linear array ultrasound probes are, however, particularly prone to anisotropy. Anisotropy results from tissues that contain multiple parallel linear sound interfaces such as tendons or muscles that cause preferential reflection of the beam in one direction. If the probe is not perpendicular to the fibre axis, then it results in a dramatic reduction in echogenicity of the tissue. Angulation of the probe or beam steering can overcome the artefact (Connolly et al. 2001). Scrupulous technique must be adopted to ensure the ligament or tendon is imaged parallel to the face of the ultrasound transducer (O'Connor 2002).

Beam edge artefact results in a characteristic appearance at the edge of particularly large tendons, such as the Achilles, with loss of signal and distal acoustic shadowing that can mimic or obscure fluid or inflammation in the paratenon.

Both anisotropy and beam edge artefact may mimic disease to the unwary ultrasonologist and represents a pitfall in the ultrasound assessment of tendons and ligaments.

Ligaments are also vulnerable to anisotropy. Dynamic stressing of ligaments, such as the lateral collateral ligament of the ankle, can be used to assess its integrity and joint stability with U/S.

MRI is also used in the assessment of ligaments and tendons in the foot and ankle. It is, however, a static...
examination and stressing of the structure is not possible. It provides exquisite images of the anatomy of both tendons and ligaments, and is highly sensitive at exploring the full spectrum of tendon pathologies.

Tendons are homogenously of low signal on all MRI sequences. T1, T2* and STIR sequences are often used in several planes to optimize imaging. Tendinopathy results in intermediate signal, thickening and sheath fluid often with surrounding oedema (Tuitt, 2002). Contrast is not routinely administered to diagnose tendinopathy or ligamentous rupture. Focal enhancement of tendon sheaths is a marker of tenosynovitis.

In summary, MRI is better at staging large area anatomy (e.g. tendon retraction) and inflammatory change. U/S best demonstrates architectural disruption and movement-related pathology. These modalities must be considered complementary and should be used in combination for the assessment of tendon pathology.

How to detect cysts, ganglia, bursa and peri-articular masses

Fluid collections around the foot and ankle can be detected with both U/S and MRI, and differentiated from solid masses. As with joint effusions, simple encysted fluid is anechoic with acoustic enhancement deep to the fluid. Fluid structures are high signal on MRI T2 and STIR sequences and low signal on T1.

Large effusions lead to distension and decompression into synovial cysts. Ganglion cysts are often seen adjacent to joints and are particularly common around the ankle. They are mucin filled and frequently septated (O’Connor 2002). On U/S and MRI they appear as well-defined cystic lesions and may communicate with the joint or adjacent tendon sheath.

Bursae are pouches of fluid that facilitate movement between adjacent structures by reducing friction. Two types of bursae are recognized, those with a synovial lining and adventitial bursa that have no synovial lining. An adventitial bursa is acquired as the result of friction between two structures leading to the collection of fluid within the intervening soft tissue. Synovial inflammation may cause bursitis in those lined with synovium. The bursa becomes distended, the thickened synovium may be evident on both U/S and MRI, and mimics the characteristics of joint synovitis (O’Connor 2002). Inter-metatarsal bursitis can be one of the first features of RA mimicking forefoot joint disease. Rheumatoid nodules have also been described within synovial bursae (Fig. 5.7).

Necrotic cellular debris may sometimes develop within the bursa forming multiple intraarticular fibrinous deposits, known as rice body bursitis. The nodules can be visualized within the bursa on U/S and appear as low signal on both T1 and T2 weighted MRI (Spence et al. 1998).

The imaging characteristics of bursal or cystic fluid may differ if infected or haemorrhagic. Abscess collections may appear as cystic masses with the fluid containing debris. The abscess cavity will appear avascular with Doppler imaging and low signal without enhancement on T1 weighted MRI imaging.

However, the wall of the abscess, which may be thick and irregular, may appear hypervascular owing to increased perfusion about the collection. This will enhance avidly with gadolinium. U/S is particularly useful in guiding aspiration and drainage of cystic collections, especially when infection is suspected.

Rheumatoid nodules occur in periarticular extensor surfaces prone to mechanical irritation. They appear as non-cystic masses within the subcutaneous tissues, especially over the heel. Their appearance on MRI is variable, but is usually a low signal on T1 and T2 with solid or ring enhancement post contrast (Fig. 5.8) (Scutellari and Orzincolo 1998, Starok et al. 1998, el-Noueam et al. 1997). The nodules may also undergo cystic degeneration.

How to image the complications of rheumatoid arthritis.

MRI is often the optimal examination when the focus of the pathology cannot be firmly defined. Inflammation is inseparable from oedema and this appears as high signal on T2 and STIR examination. This often localizes the site of pathology when the symptoms and signs are non-specific (Narvaez et al. 2002).

Infection should be suspected in a disproportionately inflamed joint. A large joint effusion, bone destruction, marrow oedema and periarticular oedema are signs of
septic arthritis, but differentiation from active RA can be difficult. In these circumstances U/S-guided aspiration or biopsy may be of use (O'Connor 2002). The degree of marrow enhancement and amount of marrow oedema are the best indicators of extent of infection. Differentiation may, however, be difficult from non-infected joints and clinical correlation is necessary (Jelinek et al. 1996). Often U/S-guided aspiration for microbiological examination of the effusion is required.

Insufficiency fractures occur due to osteoporosis and altered ankle and foot mechanics (Narvaez et al. 2002). These may be visualized on plain film, but are notoriously difficult to see. On MRI, the marrow at the site of fracture if usually oedematous and the fracture reveals itself as a band like low signal on T1.

Avascular necrosis is a complication of the vascular compromise and steroid administration in rheumatoid patients (van Vugt et al. 1996). MRI changes predate the plain film. MRI shows a subchondral low signal line on T1 with a surrounding ring or double halo on T2. Bone infarcts may also occur and appear as serpiginous medullary areas of low signal on T1.

**GENERAL FEATURES OF THE RHEUMATOID JOINT OF THE FOOT AND ANKLE**

All the joints and the surrounding soft tissues of the foot and ankle may be affected by RA (Weishaupt et al. 1999, Resnick 2002, Vidigal et al. 1975).

On the plain radiograph, periarticular soft-tissue swelling is often one of the first signs of disease and predate the bony changes. The swelling is usually fusiform and symmetrical. It is due to joint effusion, synovial hypertrophy, oedema and tenosynovitis.

Thickening of the synovium, joint effusion and oedema of the cartilage may cause an initial and transient increase in joint space seen on the plain film before progressive cartilaginous destruction leads to joint space loss.

Juxta-articular osteoporosis is characteristic of RA, but not specific. With chronicity and steroid administration generalized osteoporosis may occur, but is frequently not visualized unless profound.

Synovial inflammation predate any bony change (Conaghan et al. 2003). As synovial proliferation turns to pannus, punched out para-articular, central or peripheral erosions develop. Subchondral sclerosis is minimal around the erosions. Erosions actually take years to develop and are indicative of irreparability.

Joint space narrowing indicates irreversible cartilage destruction. Osteophytes are lacking in the rheumatoid joint, unless there is secondary osteoarthritis.

With chronic inflammation, structural deformities occur owing to capsular contraction and ligamentous laxity. Tendon pathology and tenosynovitis is commonly due to direct destruction and may lead to rupture further exacerbating joint instability. This causes abnormal mechanics and secondary degenerative changes.

Ancillary features such as neuropathy, vasculitis, stress fractures, bursitis, infection, oedema, and rheumatoid nodules may also occur in the foot and ankle. Excess synovial fluid within the joint may decompress into synovial cysts.

**DISEASE OF SPECIFIC JOINTS**

**Disease of the forefoot**

**Metatarsal joint disease**

The foot is more frequently affected than the ankle. Forefoot pain is the presenting feature in 10–20% (Resnick, 2002).

The lateral metatarsophalangeal joints are affected earliest in the disease with the 5th metatarsophalangeal joint most commonly affected. The metatarsal head abnormalities generally antedate the phalanges. Erosions tend to occur on the medial aspect of the metatarsophalangeal joints with the exception of the 5th; this is more commonly eroded on its lateral side. The 1st metatarsal phalangeal joint is the least commonly affected in RA (Resnick 2002).

Erosions tend to be para-articular at the cartilage-synovial interface in the bare area, named due to a lack
of cartilaginous cover (Maini 1979). There may also be localized porosis, extreme thinning or a translucent zone within the cortex, which is the precursor of erosion.

Migration of pannus below the articular surface results in subchondral erosion and articular collapse resulting in surface irregularities.

The sesamoids of the 1st MTP joint may be displaced by synovial mass and also become eroded and inflamed (Resnick et al. 1977).

Chronic synovial inflammation disrupts and weakens the ligamentous structural integrity of both the ankle, but particularly, the foot. This together with bone destruction and tendinous attrition results in joint malalignment. Disruption of the transverse ligaments between adjacent metatarsals results in widening and splaying of the forefoot. The phalanges become laterally deviated and subluxed, and may eventually dislocate dorsally in a valgus position (Fig. 5.9) (Kerschbaumer et al. 1996).

The plantar capsule and plate becomes destroyed by synovial disease and the metatarsal heads herniate downwards particularly those of the 2nd and 3rd metatarsal heads. The fat pad covering the metatarsal heads becomes atrophic and fails to cushion the soft tissues from bone resulting in painful callosities.

Hallux valgus is common and worsens with the duration of the disease (Haas et al. 1999). The extensor hallucis longus tendon becomes displaced into the first web space and the altered forces during contraction acts as an adductor increasing the valgus deformity.

Stress fractures most commonly affect the second metatarsal neck and shaft, due to weakening of the bone with chronic steroid administration (Elkayam et al. 2000). Plain film may fail to show the fracture for several weeks. MRI is the investigation of choice in the equivocal cases. Oedema is visualized in the bone marrow on STIR sequences and a low signal line on both T1 and STIR at the fracture site.

**Interphalangeal joints**

The interphalangeal joint of the 1st toe is the most commonly affected. The other proximal interphalangeal joints are commonly affected with joint space loss and erosion. The distal interphalangeal joints are usually spared (Halla et al. 1986).

Subluxation of both the metatarsophalangeal joints and interphalangeal joints results in an imbalance of the intrinsic and extrinsic muscles of the toes, this in combination with contraction of the extensor muscles results in mallet, hammer and claw toes. Hyperextension often occurs at the interphalangeal joints. Painful callosities may also occur on the dorsal aspect of cock up toes due to pressure.

**Disease of the midfoot and hindfoot**

**Talonavicular, calcaneocuboid and subtalar joints**

Midfoot involvement in RA is common. Synovitis is, however, a less conspicuous feature than in the forefoot. Joint space narrowing is prominent with sclerosis and secondary osteophytosis more common (Resnick 2002).

In the midfoot, RA has a predilection for the talocalcaneonavicular joint. Plain films often fail, however, to demonstrate the degree of early cartilage loss or erosive disease. MRI is of use to visualize the synovitis and erosive change in the midtarsal joints. Erosions are infrequently visualized and are generally small. Complete bony ankylosis of the talocalcaneonavicular joint may occur with chronicity.

The calcaneonavicular, intercuneiform, cuneocuboid and cuboideonavicular tend to be less severely, but similarly affected, as the joints are communicative. Weakness of the musculature and ligaments results in pes planus and pes planovalgus as the arch of the foot becomes unsupported.

The tendon of tibialis posterior may become weakened by the intensity and duration of activity placed upon it in an attempt to support the longitudinal arch. This is the most common tendon affected by RA (Coakley et al. 1994). A full-thickness tear results in acquired unilateral flatfoot, valgus hindfoot, forefoot abduction and talonavicular subluxation.

Synovial proliferation may involve the subtalar joint producing a subtalar mass in the posterior recess. Subtalar disease tends to occur prior to the ankle disease. Progressive deformity occurs due to slow destruction on the surrounding supporting tissues.
Sinus tarsi syndrome may also result from inflamed synovial tissue in the subtalar and talo-calcaneonavicular joints. This gives rise to local pain and reflects true subtalar joint involvement.

Synovial cysts or synovitis within bursa may extrude from the subtalar region causing large posterior masses, which can be visualized with both U/S and MRI.

**Ankle**
The tibiotalar joint is less frequently involved than the subtalar and midtarsal joints (Abdo & Iorio 1994). Osseous changes occur infrequently and late (Resnick 2002).

Valgus deformity, pronation and eversion of the hindfoot result from exaggerated forces on the inflamed subtalar joint. Subluxation, osteoporosis and stress fractures occur late in disease.

**Disease of the surrounding soft tissues**

**Achilles tendinopathy**
The surrounding soft-tissue structures of the heel are frequently diseased in RA. Heel pain is often due to Achilles tendinopathy and frequently chronic. Thickening and fusiform swelling of the tendon is frequently present (Jarvinen et al. 2001). Tendon thickening can be related to tendinopathy, paratendonitis or focal rheumatoid nodules. U/S is the most effective way of differentiating these entities in the rheumatoid patient. Tendon degeneration tends to occur in the middle third of the Achilles tendon. The swelling converts the normal ellipsoid cross-sectional area of the tendon into a circular configuration in the transverse plane. The tendon becomes heterogenous and darker in echotexture due to increased fluid content within the tendon (Fig. 5.10).

![Figure 5.10](image)

(A) Sagittal power Doppler sonogram of the Achilles tendon. There is diffuse tendinopathic change present with thickening and hypoechogenicity of the tendon associated with neovascularity. (B) Sagittal grey scale Doppler sonogram of the Achilles tendon. Rheumatoid nodule in the paratendon simulating tendinopathy.
Weakness of the Achilles occurs 2–6 cm proximal to the insertion, at the enthesis and at the muscle/tendon interface. Rupture most frequently occurs in the most hypovascular region 2–6 cm from the insertion. Suspected clinical tears of the Achilles can be readily diagnosed with U/S and MRI (Stiskal et al. 1997).

Steroid injection of the tendon under U/S guidance is of questionable value and may predispose the tendon to rupture. New therapies using dry needling, autologous blood injection and lithotripsy in the treatment of tendonopathy are under clinical assessment.

Achilles bursitis

Retrocalcaneal and subcutaneous bursitis is more common than Achilles tendinosis (Stiskal et al. 1997).

A small amount of fluid may be visualized in the retrocalcaneal bursa in Kager’s triangle deep to the distal third of the Achilles. Synovial hypertrophy and effusion may lead to expansion of the joint capsule and expansion of the retrocalcaneal bursa, which extends out of Kager’s triangle in a teardrop fashion (Fig. 5.11). This produces a painful mass on the posterosuperior aspect of the heel, which causes impingement on the peroneal tendons. Occasionally, the superficial calcaneal bursa becomes distended.

Figure 5.11  Sagittal power Doppler sonogram (A) and lateral calcaneal radiography (B) demonstrating retrocalcaneal bursitis. The ultrasound shows hypoechoic thickening of the retrocalcaneal bursa associated with hyperaemia. The radiograph shows the plain film changes of retrocalcaneal bursitis with erosion of the posterior corner of the calcaneus.
Impingement can be assessed with MRI. A synovial mass or bursa may be visualized on the lateral or posterior aspect of the ankle with fluid identified in the peroneal tendon sheaths.

**Peroneal tendons**

Lateral pain is the hallmark of peroneal tendon pathology with eventual loss of eversion and cavovarvalgus deformity. Problems arising within the peroneus longus include tenosynovitis, tendinopathy and tendinous disruption. Longitudinal tears of the tendon are commonly seen in peroneus brevis.

Longitudinal tears and ruptures of the peroneal tendons can be readily identified on MRI and U/S (Rasmussen 2000, Diaz et al. 1998, Tuite 2002). Imaging features include a chevron-shaped tendon, increased signal on T1 and T2, flattened peroneal groove, abnormalities of the lateral ligament complex, and fibular spurring.

The peroneal tendons are most vulnerable from adjacent synovial inflammation in three specific tunnels, the calcaneotrochlear process, in the region of the inferior peroneal retinaculum and the cuboid notch where the tendon sharply changes direction passing the plantar surface of the foot.

Subluxation of the peroneal tendons may occur following an acute traumatic episode or chronically after disruption of the inferior peroneal retinaculum. Dynamic U/S can visualize the tendon flicking over the fibular into its aberrant position (Neustadter et al. 2004).

Plain radiography fails to directly visualize the tendons. Tenosynovitis may be identified on U/S and MRI with fluid identified within the tendon sheath (Bare & Haddad 2001). Fluid may be visualized in the normal sheath, but if the tendon is completely surrounded, this denotes pathology.

**Plantar fascia**

Plantar fasciitis is a common cause of subcalcaneal heel pain, which may be related to the biomechanical alterations of the foot and is seen particularly in pes planus and pes cavus.

It is, however, a misnomer and is, in fact, a tendinitis of the common tendinous aponeurosis of the superficial intrinsic muscles of the foot. On U/S examination, there is thickening of the plantar ‘ascia’ to greater than 4 mm (normally 3 mm) measured in the sagittal plane at the point of it crossing the inferior calcaneal border. The contralateral side is often also thickened, but asymptomatic; however, a discrepancy of greater than 1 mm is abnormal. The echogenicity of the ‘ascia’ also decreases and becomes heterogeneous like other tendinopathies. Occasionally, fluid is seen deep or superficial to the fascia (Gibbon & Long 1999). U/S-guided steroid injection is useful in treatment.

MRI can also be used to delineate the pathology with oedema and intermediate signal change identified of the fascia (Theodorou et al. 2001).

**SUMMARY**

Imaging allows the demonstration of the hallmarks of rheumatoid disease, notably erosions and synovitis. It also allows the complications of the disease on the foot and ankle to be assessed and, potentially, treated, even before clinically evident.

The role of the radiologist is integral to the diagnosis, management and prognosis of the patient with rheumatoid arthritis. Plain film radiography was previously the sole technique available. With the advent of MRI and U/S the role of the imager has taken on a new relevance. Diagnosis can now be confirmed earlier and treatment protocols initiated to try to control the disease prior to the irreparable damage to bone, cartilage and joint, with the antecedent morbidity.

MRI and U/S should be routinely employed by the rheumatologist in order to optimally treat the foot and ankle in rheumatoid arthritis.

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