Name of journal: *World Journal of Orthopedics*

ESPS Manuscript NO: 8498

Columns: TOPIC HIGHLIGHT

WJO 5th Anniversary Special Issues (2): Ankle

**Power Doppler ultrasonographic assessment of the ankle in patients with inflammatory rheumatic diseases**

Suzuki T. Power Doppler ultrasonography for rheumatic ankles

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**Author contributions:** Suzuki T was involved in the conception of this topic highlight article, the literature review process, the acquisition of ultrasound images and preparing the manuscript.

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**Received:** December 28, 2013 **Revised:** April 7, 2014

**Accepted:** May 29, 2014

**Published online:**

**Abstract**

Ankle involvement is frequent in patients with inflammatory rheumatic diseases, but accurate evaluation by physical examination is often difficult because of the complex anatomical structures of the ankle. Over the last decade, ultrasound (US) has become a practical imaging tool for the assessment of articular and periarticular pathologies, including joint synovitis, tenosynovitis, and enthesitis in rheumatic diseases. Progress in power Doppler (PD) technology has enabled evaluation of the strength of ongoing inflammation. PDUS is very useful for identifying the location and kind of pathologies in rheumatic ankles as well as for distinguishing between inflammatory processes and degenerative changes or between active inflammation and residual damage. The aim of this paper is to illustrate the US assessment of ankle lesions in patients with inflammatory rheumatic diseases, including rheumatoid arthritis, spondyloarthritis, and systemic lupus erythematosus, focusing on the utility of PDUS.

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**Key words:** Ankle; Power Doppler; Ultrasound; Rheumatoid arthritis; Psoriatic arthritis; Spondyloarthritis; Tenosynovitis; Enthesitis

**Core tip:** Over the last decade, ultrasound (US) has become a practical imaging tool for the assessment of articular and periarticular pathologies in rheumatic diseases. Progress in power Doppler (PD) technology has enabled evaluation of the strength of ongoing inflammation. PDUS is useful not only for identifying the pathologies in rheumatic ankles, but also for distinguishing between inflammatory processes and degenerative changes or between active inflammation and residual damage. The aim of this paper is to illustrate the US assessment of ankle lesions in patients with inflammatory rheumatic diseases, including rheumatoid arthritis and spondyloarthritis, focusing on the utility of PDUS.

Suzuki T. Power Doppler ultrasonographic assessment of the ankle in patients with inflammatory rheumatic diseases. *World J Orthop* 2014; In press

**Available from:**

**DOI:**

INTRODUCTION

Many kinds of systemic arthritic diseases affect the ankle. Among them, there are also diseases for which ankle involvement is a hallmark. As the ankle is one of the major weight-bearing joints, it is prone to being affected by injury and/or by degenerative disorders. In the ankles of patients with inflammatory rheumatic diseases, this extrinsic damage commonly coexists, and it is not always easy to distinguish between them.

Anatomical structures in the ankle lie close together and are sometimes overlying, making differentiation between adjacent structures problematic[1]. Inflamed synovia are relatively small in size and located relatively deep beneath the skin. Obesity, peripheral edema, and predisposing degenerative conditions such as osteophytes, especially in elderly patients, also impede the detection of synovitis.

In this context, a diagnostic imaging technique is important for the evaluation of ankle lesions. However, plain radiography has limited value for the detection of soft tissue changes, and it lacks tissue specificity. Although magnetic resonance (MR) imaging is a high resolution imaging method that can disclose both bone and soft tissue abnormalities, it involves problems such as MR-incompatible metallic implants, patient claustrophobia, accessibility issues, and high cost, all of which prevent it from being widely used for the examination of ankles in rheumatic diseases.

In contrast, ultrasound (US) is a versatile, accurate, safe, and relatively cheap modality that is increasingly used in daily rheumatologic practice. It offers multiplanar evaluation and allows parallel dynamic assessment of multiple target structures, including joints, tendons, ligaments, and bony cortex[2].

In addition to the advantages of grey-scale ultrasound (GSUS), power Doppler (PD) technology has allowed the visualization of the hyperemia of soft tissues in inflammatory articular diseases [3]. A comparison of PDUS findings with histopathologic findings of synovial membrane vascularity in rheumatoid arthritis (RA) patients who have undergone knee arthroplasty has shown that PD signals represent abnormal hypervascularity in RA synovial tissue[4]. Many reports indicate that the intensity of PD signals in the synovium correlates with the intensity of articular and tenosynovial inflammation in RA patients[5,6]. Moreover, PD techniques seem to help differentiate inflammatory enthesis diseases[7].

The aim of this paper is to illustrate the US assessment of ankle lesions in patients with inflammatory rheumatic diseases, focusing on the utility of PDUS.

US SCANNING TECHNIQUE FOR THE ANKLE REGION

The important pathologies addressed here are synovial inflammations, including articular synovitis and tenosynovitis, and enthesitis of tendon insertion, including subtendinous bursitis. Structural damage such as bone erosion and tendon degeneration are addressed collaterally and ligament lesions are not mentioned here.

The Outcome Measures in Rheumatology Clinical Trials (OMERACT) US definitions for the most common pathological findings occurring in inflammatory rheumatic diseases are presented in Table 1[8]. For detailed information regarding the US machine settings and fundamental scanning techniques, please refer to the relevant published papers[9].

Data regarding the positioning of the patient and anatomical structures to be scanned in the ankle region are presented in Table 2[10]. An encompassing scanning technique makes it possible for the examiners not to overlook pathologies, regardless of whether they were symptomatic or asymptomatic[11,12]. Utilizing these techniques, we have shown the frequencies of various pathologies in symptomatic ankles with either early or established RA in a previous study (Table 3)[13]. This study revealed that ankle tenosynovitis is observed more frequently than ankle synovitis among early RA patients.

US EVALUATION OF PATHOLOGIES IN ANKLES WITH RHEUMATIC DISEASES

The major inflammatory rheumatic diseases addressed in this paper are RA, psoriatic arthritis (PsA), reactive arthritis (ReA), undifferentiated spondyloarthritis (uSpA), and systemic lupus erythematosus (SLE). US assessment of crystal-associated diseases and degenerative disorders related to aging or trauma will be addressed elsewhere. Multiplanar scanning using both GSUS and PDUS should be carried out. Synovial fluid detected by GSUS and/or hyperemia detected in or adjacent to the synovial membrane by PDUS represents synovitis[14]. Although the degree of hyperemia is indicative of the extent of inflammation, hyperemia itself is not specific to a certain disease[15].

Synovial hypertrophy detected by GSUS represents cellular infiltration into synovial tissues with or without synovial cell proliferation. Fine and slow blood flow detected in thickened synovium by PDUS represents proliferative synovitis associated with vascularization, which is a hallmark of the active synovitis observed in RA, PsA, and sometimes SLE[16].

The respective features of the pathologies in inflammatory rheumatic disease depicted in each anatomical structure are addressed below.

Tibiotalar joint (talocrural joint)

To visualize the anterior recess of the tibiotalar joint, place the transducer in the sagittal plane distal to the tibia. In the normal joint, a hyperechoic anterior fat pad can be seen to fill the space between the tibia and the talus anteriorly with a layer of anechoic hyaline cartilage on the surface of the talar dome[17].

A small simple effusion in mild acute synovitis of the tibiotalar joint in a patient with reactive arthritis is depicted as an anechoic area (Figure 1). Severe proliferative synovitis of the tibiotalar joint in rheumatoid arthritis is depicted as a hypoechoic area over the talar dome (Figure 2). Large effusion and/or proliferative synovium lead to the displacement of the fat pad covering the talus neck and creating capsule distension.

Complex joint fluid can be seen as a hypoechoic area. To distinguish synovium from complex joint fluid, blood flow detected by PD sonography is helpful[18]. However, PD sensitivity is relatively low at the median part of the anterior recess because the synovium is located deep beneath the tendons and the fat body. Strong blood flow in the dorsalis pedis artery also hinders the identification of the fine synovial vessels. In order to increase the sensitivity of the PD detection, scanning both lateral and medial sides of the anterior recess must be performed (Figure 3).

Posterior subtalar joint

The subtalar or talocalcaneal joint is composed of three facets: a broad posterior facet representing the primary articulating surface, a medially located middle facet in which the sustentaculum tali articulates with the medial process of the talus, and an anterior facet. There are limited sonographic windows for imaging the subtalar joint (STJ), and the posterior subtalar joint (PSTJ) is usually the target. Synovitis of the PSTJ is best evaluated by scanning its posterior recess. The joint space of the PSTJ is easy to determine in the sagittal view crossing the posterior talar process. In order to fully visualize the posterior recess, which tends to distend laterally, the transducer should be placed just lateral to the Achilles tendon in an anatomic parasagittal plane by medially angling the transducer face (Figure 4). Because synovium is depicted as relatively shallow through this posterolateral window, better detection of the PD signal and a safer route for aspiration are provided[19].

The medial facet of the PSTJ can be partly observed in the coronal view crossing the medial malleolus. In the normal joint, the joint space between the talus and the sustentaculum tali can be depicted beneath the tibiocalcaneal ligament. In the case of synovitis, effusion and/or proliferated synovium stretch cranially along the tibiocalcaneal ligament (Figure 5).

Talonavicular joint

The talonavicular joint (TNJ) is best evaluated by the longitudinal dorsal view of the joint. In the case of TNJ synovitis in RA patients, proliferated synovium bulges out of the joint space dorsally and the PD signal can be easily detected (Figure 6).

It has been reported that TNJ synovitis is frequently detected by US in both symptomatic and asymptomatic ankles in RA patients[13]. Similarly, proliferative synovitis of the TNJ can arise in PsA patients (Figure 7).

It has also been reported that TNJ synovitis is often detected by US in the ankles of healthy control subjects[20]. The ankle is one of the sites of predilection for osteoarthritis. In long-standing RA patients, degenerative changes in the TNJ are frequently encountered (Figure 8). PDUS can add to the information regarding the activity of ongoing inflammation in the joint.

Extensor tendons

The three tendons in the anterior aspect of the ankle from medial to lateral are the tibialis anterior (TA), extensor hallucis longus (EHL), and extensor digitorum longus (EDL)[17]. These extensor tendons pass beneath retinacula and have sheaths to protect them. The main pathology that occurs in inflammatory rheumatic diseases in this area is tenosynovitis. Tenosynovitis of the EDL is most frequently observed, followed by that of the TA. Tenosynovitis of the EHL seems to be less frequent while the myotendinous junction should not be confounded with dilated tenosynovium. These extensor tendons tend to present with serous tenosynovitis rather than proliferative tenosynovitis, unlike the medial flexors and lateral peroneal tendons, which frequently present with proliferative tenosynovitis (Figure 9)[21].

Medial flexors

By transverse scanning of the medial ankle tendons, the tibialis posterior (TP) tendon, flexor digitorum longus (FDL) tendon, and flexor hallucis longus (FHL) tendon can be visualized from the anterior to the posterior side[22].

As the TP and FDL hook around the medial malleolus and the FHL runs in a groove along the calcaneus, all tendons have individual sheaths. Tenosynovitis frequently occurs in this area in inflammatory rheumatic diseases. Tenosynovitis of TP is most frequently observed, and is sometimes accompanied by FDL tenosynovitis (Figure 10). Pathology usually occurs at or distal to the medial malleolus. Fluid collection within the synovial sheath is predominantly observed near the distal end of the sheath, while small effusion often exists at the same site in the ankles of healthy subjects. Proliferative tenosynovitis of TP is frequently observed in RA patients[21]. Thickening and hyperemia of the sheath with or without fluid in it are the typical features. It has been reported that tenosynovitis is observed more frequently than joint synovitis in symptomatic ankles with early RA[13].

The second site of pathology on the TP is where it inserts into the navicular bone. A longitudinal scan is more informative in proximity to its insertion. An important active inflammatory pathology in rheumatic diseases at tendon insertion is enthesitis. Enthesitis frequently occurs in patients with SpA (Figure 11A). The distal end of a normal TP tendon splays at the insertion and may appear hypoechoic due to anisotrophy. Therefore, the presence of intratendinous hyperemia permits a more confident diagnosis of enthesitis.

Although enthesitis is the hallmark of PsA, ankle tenosynovitis is also often observed in PsA patients. In some cases, the implication of enthesitis in a functional enthesis formed by fibrocartilage on the surface of bone and tendon is suggested (Figure 11B)[23].

Periarticular involvement is an important characteristic of musculoskeletal manifestation in SLE patients[24]. In particular, tendinopathies including tendonitis, tenosynovitis and tendon rupture are thought to be common. Although there have only been a few reports about the sonographic feature of tenosynovitis only in hands or wrists with SLE, ankle tendinopathies do not seem to be rare (Figure 12). The appearance of tenosynovitis or enthesitis, however, seems to be unspecific.

Peroneal tendons

The lateral ankle tendons are the peroneus longus and brevis. They hook behind the retromalleolar sulcus of the fibula and are stabilized by the superior peroneal retinaculum. At the level of the lateral malleolus, the peroneus longus and brevis share a common sheath. Distal to the lateral malleolus, the peroneals diverge[22].

Pathology usually occurs at or distal to the lateral malleolus (Figure 13). Tenosynovitis of peroneus longus and brevis can occur either independently or concurrently. Proliferative tenosynovitis of peroneal tendons and TP is frequently observed in RA patients[21]. Thickening and hyperemia of the sheath with or without fluid in it are sonographic findings suggestive of active inflammation.

In addition to TP, enthesitis of PB at its insertion into the base of the fifth metatarsal is frequently observed in PsA patients (Figure 14A). Tenosynovitis of peroneal tendons is also commonly observed in PsA patients (Figure 14B).

Achilles tendon

The Achilles tendon is a typical tendon with a linear fibrillar pattern. It inserts into a relatively small footprint on the posterosuperior aspect of the calcaneus. Between the calcaneus and the Achilles tendon there is a retrocalcaneal bursa. A normal bursa is thin walled and contains only minimal fluid[25].

Achilles tendon enthesopathy is a common finding in SpA, and can be present in both symptomatic and asymptomatic patients[7]. In the OMERACT definition (Table 1), acute and chronic inflammatory aspects in gray-scale US (*i.e.,* loss of normal echo structure, increased thickness, or focal calcific deposits) and Doppler US are combined with findings of structural damage (*i.e*., enthesophytes and bony erosions). This combination may be helpful for diagnostic purposes (*i.e*., presence or absence of enthesis involvement) but probably not for responsiveness or for the differential diagnosis of inflammatory diseases (*i.e.*, SpA versus RA versus mechanical or metabolic entheseal involvement). Enthesitis rather than enthesopathy is important to evaluate in inflammatory rheumatic diseases. For defining enthesitis, a Doppler signal should be detected at the cortical enthesis insertion and should be differentiated from a reflecting surface artifact or nutrition vessel signal[7]. A Doppler signal may be detected even in the absence of either bone changes (cortical irregularities, erosions, or enthesophytes) or tendon degeneration (tendinosis, tendon erosions and tears). For example, ReA patients with subacute enthesitis together with enthesial and intratendinous hyperemia and a small amount of fluid in retrocalcaneal bursa with minimal structural damage (Figure 15).

It has been suggested that inflammation at the bursal part of the fibrocartilage is important for the pathogenesis of enthesitis in SpA patients[26]. Bone erosion that is cranially adjacent to the insertion is characteristic of enthesitis in SpA. In early undifferentiated SpA patients, a Doppler signal at the enthesis insertion that is associated with cortical erosion can be observed with minimal tendinosis (Figure 16). In chronic active enthesitis, increased thickness, hypoechogenicity, and loss of fibrillar pattern of the Achilles tendon are associated with abnormal vascularization at the enthesis insertion with bone changes (Figure 17). Enthesitis is also often observed among RA patients. It has been suggested that retrocalcaneal bursa is primarily involved in the rheumatoid enthesitis symptom (Figure 18)[7].

The Achilles tendon does not have a tendon sheath. Instead, it has a U-shaped paratenon wrapping around the tendon from its dorsal aspect, which is normally barely visible. Achilles paratenonitis is inflammation in the paratenon and can occur in isolation or in association with tendinopathy. The paratenon becomes thickened, hypoechoic and hyperemic in the case of paratenonitis (Figure 19). Achilles paratenonitis is sometimes observed among RA patients, especially in the early stage of disease[13].

CONCLUSION

US is useful for identifying the location and kind of pathologies in rheumatic ankles. Furthermore, PDUS is useful for distinguishing between inflammatory processes and degenerative changes or between active inflammation and residual damage. PDUS is of vital use in the assessment of ankles in patients with inflammatory rheumatic diseases in daily practice.

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**P-Reviewers:** Azzoni R **S-Editor:** Ji FF **L-Editor: E-Editor:**

**Figure 1 Mild tibiotalar joint synovitis in reactive arthritis.** Sagittal grey-scale sonogram of the anterior recess of the tibiotalar joint shows localized anechoic synovial effusion (arrow). Tib: Tibia; Tal: Talus; F: Fat pad; A: Dorsalis pedis artery.

**Figure 2 Severe tibiotalar joint synovitis in rheumatoid arthritis.** Sagittal grey-scale sonogram of the anterior recess of the tibiotalar joint shows that a normal hyperechoic anterior fat pad (F) is displaced anteriorly by hypoechoic synovium (arrow). Tib: Tibia; Tal: Talus; F: Fat pad; E: Extensor hallucis longus.

**Figure 3 Synovial proliferation in rheumatoid arthritis.** Sagittal power Doppler sonogram of the lateral side of the anterior recess of the tibiotalar joint shows synovial thickening (arrow) with peripheral vasculalization. Tib: Tibia; Tal: Talus.

**Figure 4 Synovitis of the posterior subtalar joint in rheumatoid arthritis.** A: Sagittal grey-scale sonogram crossing the posterior talar process shows the edge of the distended posterior recess (arrow) of the PSTJ. B: Posterolateral view of the power Doppler sonogram fully depicting the distended posterior recess (arrow) of the PSTJ. Note the close relation of this recess with the superior margin of the calcaneus (Cal). Tib: Tibia; Tal: Talus; KF: Kager’s fat pad; AT: Achilles tendon, arrowhead (joint space); PSTJ: posterior subtalar joint; PP: Posterior talar process.

**Figure 5 Synovitis of the posterior subtalar joint in rheumatoid arthritis.** Coronal grey-scale (A) and power Doppler (B) sonogram of the medial facet (arrowhead) of the PSTJ. PD-signal-positive proliferated synovium (arrow) of the PSTJ stretches cranially along the tibiocalcaneal ligament (L). Tal: Talus; Cal: Calcaneus; ST: Sustentaculum tali; PSTJ: posterior subtalar joint.

**Figure 6 Synovitis of the talonavicular joint in rheumatoid arthritis.** Longitudinal grey-scale (A) and power Doppler (B) sonogram of the dorsal aspect of the TNJ shows marked synovial thickening (arrow) with peripheral vasculalization. Tal: Talus; Nav: Navicular; TNJ: Talonavicular joint.

**Figure 7 Synovitis of the talonavicular joint in psoriatic arthritis.** Longitudinal power Doppler sonogram of the dorsal aspect of the TNJ shows synovial effusion and marked synovial thickening (arrow) with peripheral vasculalization. TNJ: Talonavicular joint; Tal: Talus; Nav: navicular.

**Figure 8 Deformity of the talonavicular joint in rheumatoid arthritis.** Longitudinal power Doppler sonogram of the dorsal aspect of the TNJ shows joint space narrowing (arrow) and osteophyte formation (arrowhead) with minimum flow signal adjacent to an osteophyte. TNJ: Talonavicular joint; Tib: Tibia; Tal: Talus; Nav: Navicular.

**Figure 9 Serous tenosynovitis of the extensor digitorum longus in rheumatoid arthritis.** Transverse (A) and longitudinal (B) grey-scale sonogram of the dorsal aspect of the EDL shows marked expansion of the tendon sheath with anechoic synovial effusion (arrow). Tal: Talus; Nav: Navicular; Cun: Cuneiform; EDL: Extensor digitorum longus.

**Figure 10 Proliferative tenosynovitis of the tibialis posterior in rheumatoid arthritis.** A: Transverse grey-scale sonogram through the medial ankle at the level of the medial malleolus (MM). Hypoechoic thickened tenosynovium of TP with anechoic effusion (arrow) within the sheath is depicted; B: Power Doppler sonogram shows hyperemia of the tenosynovium surrounding TP and the flexor digitorum longus. Tib: Tibia; TP: Tibialis posterior; MM: Medial malleolus; FDL: Flexor digitorum longus.

**Figure 11 Figures.** A: Enthesitis of the tibialis posterior at the navicular insertion in psoriatic arthritis. Longitudinal power Doppler sonogram of TP shows intratendinous hypoechoic change and loss of the fibrillar echoes with hyperemia adjacent to the insertion into the navicular bone (Nav). Cortical irregularities of the navicular bone at the insertion are also depicted; B: Tenosynovitis of the contralateral TP in the same patient. Transverse power Doppler sonogram at the level of the tip of the medial malleolus shows thickening and hyperemia of both the tendon sheath and the flexor retinaculum. Cortical irregularities of the medial malleolus are also depicted. Nav: Navicular bone; MM: Medial malleolus.

**Figure 12 Figures.** A: Tenosynovitis of the TP in systemic lupus erythematosus. Transverse power Doppler sonogram over the inframalleolar area shows thickening and hyperemia of the tendon sheath with small synovial effusion; B: Enthesitis of the contralateral TP in the same patient. Longitudinal power Doppler sonogram of the TP shows intratendinous hyperemia adjacent to the insertion into the navicular bone. Tal: Talus; Cal: Calcaneus; TP: Tibialis posterior; Nav: Navicular bone.

**Figure 13 Proliferative tenosynovitis of the peroneus longus and brevis in rheumatoid arthritis.** Transverse power Doppler sonogram through the lateral ankle at the level of the lateral malleolus shows thickening and marked hyperemia of the tenosynovium surrounding the peroneus longus and peroneus brevis. PL: Peroneus longus; PB: Peroneus brevis; LM: Lateral malleolus.

**Figure 14 Figures.** A: Enthesitis of the PB in psoriatic arthritis. Longitudinal power Doppler sonogram of the PB shows intratendinous hypoechoic change and loss of the fibrillar echoes with hyperemia adjacent to the insertion into the base of MT5. Cortical irregularities of the bone at the insertion are also depicted; B: Tenosynovitis of the contralateral PB in the same patient. Transverse power Doppler sonogram at the level of the peroneal tubercle of the Cal shows thickening and hyperemia of the PB tendon sheath. PL: Peroneus longus; PB: Peroneus brevis; MT5: The fifth metatarsal bone; Cal: Calcaneus.

**Figure 15 Achilles tendon enthesitis in reactive arthritis.** Longitudinal power Doppler sonogram of the AT shows enthesial and intratendinous hyperemia and a small amount of fluid in retrocalcaneal bursa (arrow) with minimal cortical irregularities and tendinosis. Cal: Calcaneus; AT: Achilles tendon.

**Figure 16 Achilles tendon enthesitis in early undifferentiated spondyloarthritis.** Longitudinal power Doppler sonogram of AT shows abnormal vascularization at the enthesis insertion into the calcaneal bonewith minimal tendinosis. Cal: Calcaneus; AT: Achilles tendon.

**Figure 17 Achilles tendon enthesitis in chronic active psoriatic arthritis.** Longitudinal power Doppler sonogram of AT shows increased thickness, hypoechogenicity, and loss of fibrillar pattern of AT with intratendinous hyperemia adjacent to the enthesis insertion into the calcaneal bone. Calcification (arrowhead) is also depicted. Cal: Calcaneus; AT: Achilles tendon.

**Figure 18 Retrocalcaneal bursitis in rheumatoid arthritis.** Longitudinal power Doppler sonogram of the AT shows distended retrocalcaneal bursa (arrow) associated with peribursal hyperemia. Increased thickness of the AT with intratendinous hyperemia adjacent to the bursa is also depicted. Cal: Calcaneus; AT: Achilles tendon.

**Figure 19 Achilles paratenonitis in early rheumatoid arthritis.** Transverse (A) and longitudinal (B) power Doppler sonogram of the AT shows thickened, hypoechoic, and hyperemic paratenon (arrow) at the level of the KF with minimal tendinopathy. Hyperemia in the KF is also depicted. AT: Achilles tendon; KF: Kager’s fat pad.

**Table 1 The Outcome Measures in Rheumatology Clinical Trials definitions of ultrasound pathological findings**

|  |  |
| --- | --- |
| RA Bone Erosion | An intra-articular discontinuity of the bone surface that is visible in two perpendicular planes |
| Synovial Fluid | Abnormal hypoechoic or anechoic (relative to subdermal fat, but sometimes may be isoechoic or hyperechoic) intra-articular material that is displaceable and compressible, but does not exhibit a Doppler signal |
| Synovial Hypertrophy | Abnormal hypoechoic (relative to subdermal fat, but sometimes may be isoechoic or hyperechoic) intra-articular tissue that is nondisplaceable and poorly compressible and which may exhibit a Doppler signal |
| Tenosynovitis | Hypoechoic or anechoic thickened tissue with or with- out fluid within the tendon sheath, which is seen in two perpendicular planes and which may exhibit a Doppler signal |
| Enthesopathy | Abnormally hypoechoic (loss of normal fibrillar architecture) and/or thickened tendon or ligament at its bony attachment (may occasionally contain hyperechoic foci consistent with calcification), seen in two perpendicular planes that may exhibit a Doppler signal and/or bony changes, including enthesophytes, erosions, or irregularity |

**Table 2 Patient positions for ultrasound examination of ankle structures**

1 Supine, with flexed knee, foot on the examination bed

Tibiotalar joint: anterior recess

Talonavicular joint

Subtalar joint: lateral and medial recess

Tendon compartments

Anterior: Tibialis anterior tendon

Extensor hallux longus tendon

Extensor digitorum longus tendon

Lateral: Peroneus brevis tendon

Peroneus longus tendon

Medial (frog position):

Tibialis posterior tendon

Flexor digitorum longus tendon

Flexor hallux longus tendon

2 Prone, with the foot hanging over the examination bed

Achilles tendon

Superficial and retrocalcaneal bursae

Subtalar joint: posterior recess

**Table 3 The frequencies of various pathologies in the symptomatic ankles with either early or established rheumatoid arthritis**

|  |  |  |  |  |
| --- | --- | --- | --- | --- |
|  |  | Early RA | Established RA | Overall |
| Number of ankles | | 62 | 38 | 100 |
| Joint synovitis | |  |  |  |
|  | Talocrural joint synovitis (%) | 32.2 | 39.5 | 35.0 |
|  | Subtalar joint synovitis (%) | 30.7 | 36.8 | 33.0 |
|  | Talonavicular joint synovitis (%) | 27.4 | 26.3 | 27.0 |
|  | Overall (%) | 48.4 | 68.4 | 56.0 |
| Tenosynovitis | |  |  |  |
|  | Ankle flexors (TP, FDL, FHL) (%) | 54.8 | 31.6 | 46.0 |
|  | Peroneal tendons (PB, PL) (%) | 33.9 | 21.1 | 29.0 |
|  | Ankle extensors (TA, EHL, EDL) (%) | 12.9 | 5.3 | 10.0 |
|  | Overall | 69.4 | 47.4 | 61.0 |
| Achilles tendon involvement | |  |  |  |
|  | Retrocalcaneal bursitis (%) | 35.5 | 13.2 | 27.0 |
|  | AT enthesitis (%) | 19.4 | 26.3 | 22.0 |
|  | AT tendonitis (%) | 12.9 | 13.2 | 13.0 |
|  | AT paratenonitis (%) | 8.1 | 2.6 | 4.0 |
|  | Overall (%) | 38.7 | 39.5 | 39.0 |
| TP: Tibialis posterior; FDL: Flexor digitorum longus; FHL: Flexor hallucis longus; PB: Peroneus brevis; PL: Peroneus longus; TA: Tibialis anterior; EDL: Extensor digitorum longus; EHL: Extensor hallucis longus; AT: Achilles tendon. | | | | |