The role of ultrasonography in the diagnosis of rheumatoid arthritis and peripheral spondyloarthropathies

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Summary

In recent years a dynamic development of ultrasound technology has been observed. Consequently, ultrasound is increasingly being utilized in rheumatology. With the introduction of high-frequency (up to 18 MHz) linear probes, sensitive Doppler and harmonic imaging, ultrasound can now assess structures as small as peripheral nerves with a diameter of 1 mm [1–4], capsular ligamentous complexes and labra [5–7]. We are now able to detect the early stages of rheumatoid arthritis, which precede the development of irreversible joint damage [8,9]. Finally, we are looking for a place for elastography in the evaluation of musculotendinous structures [10].

The diagnostic workup of early stages of peripheral arthritis utilizes ultrasound and magnetic resonance imaging (MRI). The use of ultrasound is especially recommended due to its wide availability, ease of testing, lower cost, the ability to perform dynamic test and no need to remain motionless during the test [9,11,12].

In clinical practice, an ultrasound is performed to:
• diagnose rheumatic disease lesions,
• monitor treatment,
• confirm remission, in cases where clinical evidence is not clear.

Ultrasound examination does not identify specific rheumatic diseases. It only helps determine the type of abnormalities, their progress and location. These abnormalities include [11,12]:

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1. Synovial membrane pathologies, including: thickening, hipervascularisation, fibrosis of the synovium, synovial sheaths and bursae,
2. Exudate, which usually accompanies synovial pathology,
3. Changes in tendons, i.e. tenosynovitis with inflammation of the tendon, leading to tendon damage, i.e. a partial or complete tendon rupture,
4. Osteochondral changes, including: cartilage damage, cysts, inflammation, erosions,
5. Enthesopathies, that is tendon and ligaments' attachments pathologies.

In rheumatoid patients, USG is performed in the diagnostic workup for peripheral arthritis and enthesopathy.

**Peripheral Joints**

At the present, we know that rheumatic joint disease may take place within the synovium, in the subchondral bone and in the articular or extra-articular adipose tissue [8,9]. Inflamed synovial membrane has low echogenicity, similar to that of exudate, which usually accompanies synovial pathology. Exudate can be differentiated from a thick and inflamed synovial membrane by applying pressure with the transducer. Doing so will lead to displacement or compression of a low- or medium-pressure exudate [12]. A high-pressure effusion that does not displace is an indication for decompression of the joint, sheath or bursa, ideally under ultrasound guidance. Power Doppler ultrasound (PDUS) can also differentiate between exudate and synovial membrane thickening. Features of abnormal vascularization within the thickened synovium is an evidence of neoangiogenesis and a proof of active inflammation (synovitis) [12,13] (Figure 1). The intensity of synovial hipervascularisation correlates with the severity of inflammation. Lack of vascularization within a hyperechoic synovial membrane indicates fibrosis due to effective pharmacological treatment or radionuclide synovectomy (Figure 2).

In healthy individuals, the synovial membrane is not visible on an ultrasound. The first sign of rheumatic disease is a variable degree of thickening of the synovial membrane in the joint capsule, tendon sheath or bursa, due to growth (hyperplasia) of intimal layer of the synovial membrane and the swelling of subintimal layer caused by inflammation [8,9]. Inflamed synovium of the radiocarpal and midcarpal joint cavities featuring intense vascularization on PDUS, active inflammatory process.

At a certain stage of development, ectopic lymphoid tissue forms in the subintimal layer of synovium. It secretes a number of enzymes, cytokines and growth factors, leading to a degradation of the surrounding tissue [8,9]. Such synovium, called the pannus, leads to bone erosion and cartilage destruction when located near the bone. Erosions initially form at the boundary between articular cartilage and joint capsule with inflamed synovial lining. This is followed by damage to cartilage and the formation of defects in the subchondral bone of the joint surface, a so-called subchondral erosion. They are seen as cortical defects of various size filled with synovial membrane that is either avascular or vascularised (so-called active erosions) [12,14] (Figure 3A, 3B).

An identical process takes place in the articular or extra-articular adipose tissue and the bone marrow [8,9,15]. Adipocytes and the infiltrating inflammatory cells synthetize proinflammatory cytokines and growth factors that affect the metabolism of cartilage and synovial membrane, and maintain the inflammatory response. On ultrasound, inflamed adipose tissue appears hyperechogenic (swollen) and exhibits the features of vascularization (Figure 4). Inflamed adipose tissue appears hyperechogenic (swollen) and exhibits the features of vascularization (Figure 4).
cytokines that activate osteoclasts which in turn destroy the trabeculae. This leads to the formation of subchondral inflammatory cysts (geodes) and erosions [8,9].

As the disease progresses, the epiphysis undergoes erosive destruction, increasing the risk of joint subluxation and dislocation. As is the case with x-ray, an ultrasound does not assess all of all the articular surfaces. The method of choice is an MRI.

Inflammatory process in the tendon sheath (tenosynovitis) presents similarly an inflammation in the joint capsule – synovial thickening, increased vascularization, frequently accompanied by exudation [11] (Figure 5). Persistent inflammation may include the tendon (tenosynovitis with tendinitis), in case of which tendon thickness increases on gray-scale ultrasonography in such a way that the cross-section of an oval structure becomes round. While a PDUS shows vessels infiltrating the tendon from within the inflamed synovium (Figure 6A, 6B).

Tendon weakened by inflammation may become damaged [11]. In case of a partial rupture of a heterogeneous and hypochoic tendon, anechoic areas of delamination will appear. In case of a complete rupture, the level of injury should be assessed and the distance between the stumps and the length of damage in both tendon stumps should be measured prior to elective reconstruction. Dynamic ultrasound may show separation of the stumps. Dynamic ultrasound may reveal contractures of interphalangeal joint capsular structures, caused by a lack of rehabilitation, as well as abnormal tendon displacement (e.g. extensor carpi ulnaris m.) due to stretching or torn retinaculum.

Inflammation of flexor muscle tendon sheath may lead to Carpal Tunnel Syndrome by narrowing the osteo-fibrous carpal tunnel [11].
Enthesopathies

Tendon, ligament and joint capsule insertion pathology (enthesopathy) is a characteristic feature of peripheral spondyloarthropathy. On ultrasound, the affected entheses appear thick (swollen) due to structural damage (i.e. delamination) and scarring following injury. Within them, inflammatory changes can be observed. The cortex may have uneven outline, showing bone erosions at various stages of activity [12] (Figure 7). In the subchondral layer, inflammatory cysts (geodes) may be seen. It should be noted that a similar clinical picture can be observed in the course of chronic damage and degeneration. Currently, there are no morphological characteristics that would differentiate the etiology of that is tendons and ligaments’ insertions pathologies.

Conclusions

Ultrasonography is widely used in rheumatology. It allows for visualization of early inflammatory changes in the joints, corresponding to the pathological processes taking place in the synovium and adipose tissue along with an assessment of their progress. This is done in order to monitor treatment in cases of doubt, to confirm or exclude remission.

Early detection of lesions allows for the implementation of treatment that may prevent irreversible damage to the joints. In addition, visualization of a pathologically-modified synovial joint or sheath is important in terms of qualification for surgical or isotopic synovectomy. Prior
to surgery, determination of the location of pathologically modified synovial membrane and the degree of vascularization, will be crucial.

Staging of lesions on ultrasound is generally a qualitative process (pathology is either present or not). Semi-quantitative scales are used mainly for the purposes of treatment monitoring. The most frequently used classifications take into account the results of PDUS, e.g. 0 – no flow, 1 – one or two vessels visible in the synovium, 2 – numerous vessels occupying approximately 50% of the thickened synovium, 3 – vessels occupy more than 50% by volume of synovium [12]. There is much hope associated with the quantitative methods of measurement.

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