Journal of Clinical Images and Case Reports

Commentary

A SCITECHNOL JOURNAL

Sonographic Spectrum of Rheumatoid Arthritis

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Introduction

In recent years, we've seen new improvements in ultrasound (USG) technology, which has resulted in new advancements in musculoskeletal system diagnostics. Ultrasound can currently analyses structures as small as peripheral nerves with a diameter of 1 mm thanks to the introduction of high-frequency (up to 18 MHz) linear probes, sensitive Doppler, and harmonic imaging, Labra and capsular ligamentous complexes. We can now detect the early phases of rheumatoid arthritis, which occur before irreparable joint damage develops. Finally, we're looking for a role for elastography in musculotendinous structure evaluation. Ultrasound and magnetic resonance imaging are used in the early phases of peripheral arthritis diagnostic workup (MRI). Ultrasound is especially suggested because of its wide availability, ease of testing, cheaper cost, ability to do dynamic tests, and the fact that it does not require the patient to remain immobile during the procedure [1].

The use of ultrasound does not reveal specific rheumatic illnesses. It only assists in determining the type of abnormality, its progression, and its location. Among the anomalies are:

Pathologies of the synovial membrane, such as thickening, hypervascularization, fibrosis of the synovium, synovial sheaths, and bursae.

Exudate is a common symptom of synovial disease.

Tendon changes, such as tenosynovitis with tendon inflammation, can lead to tendon injury, such as partial or full tendon rupture.

Cartilage degradation, cysts, inflammation, and other osteochondral alterations

The synovial membrane is not detectable on ultrasonography in healthy people. The first indication of rheumatoid arthritis is a varying degree of thickening of the synovial membrane in the joint capsule, tendon sheath, or bursa, caused by growth (hyperplasia) of the synovial membrane's intimal layer and swelling of the subintimal layer induced by inflammation. The echogenicity of an inflamed synovial membrane is similar to that of exudate, which is commonly associated with synovial disease. By exerting pressure with the transducer, exudate may be distinguished from a thick, inflammatory synovial membrane. Exudate and synovial membrane thickening can also be distinguished using power Doppler ultrasonography (PDUS). Neoangiogenesis and active inflammation are both evidenced by aberrant vascularization within the thickened synovium (synovitis). Ectopic lymphoid tissue arises in the subintimal layer of synovium at a given point of development. It secretes a variety of enzymes, cytokines, and growth factors, which cause the surrounding tissue to degrade. When this synovium, known as the pannus, is positioned near the bone, it causes bone erosion and cartilage degradation. Erosions begin at the joint capsule-articular cartilage interface, where the synovial lining is inflammatory. Subchondral erosion is the result of cartilage deterioration and the creation of flaws in the subchondral bone of the joint surface.

Adipocytes and infiltrating inflammatory cells synthesize proinflammatory cytokines and growth factors that affect the metabolism of cartilage and synovial membrane, and maintain the inflammatory response. They appear as cortical defects of various sizes filled with synovial membrane that is either avascular or vascularized (so-called active erosions) [2]. Infiltrating inflammatory cells generate cytokines, which activate osteoclasts, which then destroy the trabecular. Subchondral inflammatory cysts (geodes) and erosions result as a result of this. The epiphysis suffers erosive degradation as the disease advances, increasing the likelihood of joint subluxation and dislocation. An ultrasound, like an x-ray, does not evaluate all of the articular surfaces. An MRI is the preferred procedure. Tenosynovitis is an inflammatory disease in the tendon sheath that mimics inflammation in the joint capsule - synovial thickening, increased vascularization, and exudation. Persistent inflammation can affect the tendon (tenosynovitis with tendinitis), causing the thickness of the tendon to rise on gray-scale ultrasonography, resulting in an oval structure becomes round cross-section.

Tendons that have been weakened by inflammation may be injured. Anechoic patches of delamination will emerge if a heterogeneous and hypo echoic tendon is partially ruptured. Prior to elective reconstruction in the case of a complete rupture, the level of injury should be examined, and the distance between the stumps and the length of damage in both tendon stumps should be measured.

References

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Citation: *Tremblay W (2021) Sonographic Spectrum of Rheumatoid Arthritis. J Clin Image Case Rep 5:7.*

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Received: July 07, 2021 **Accepted:** July 21, 2021 **Published:** July 28, 2021

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