leads to increased mortality in rheumatoid arthritis (RA). In addition to traditional, Framingham risk factors, several immuno-inflammatory cells, mediators and molecules may link atherosclerosis to arthritis. Among immune cells, primarily TH1 cells, as well as endothelial cells play a crucial role in synovial and vascular inflammation. Various cell surface molecules, such as adhesion receptors, CD40-CD40 ligand or members of the RANK-RANK ligand-osteoprotegerin system, as well as soluble pro-inflammatory cytokines, chemokines, autoantibodies and proteases have been implicated in RA and vascular damage. The early assessment of atherosclerosis and early intervention would decrease cardiovascular risk in RA.

Disclosure of Interest: None declared

DOI: 10.1136/annrheumdis-2017-eular.7197

SP0127 TAPERING BIOLOGICS INDUCES A PROTHROMBOTIC STATE IN RHEUMATOID ARTHRITIS?

M.T. Nurmohamed on behalf of M. Heslinga, J. Meijers & M. Nurmohamed. *Rheumatology, Amsterdam Rheumatology immunology Center | VUmc & Reade, Amsterdam, Netherlands*

In addition to the "traditional" risk factors for venous trombo-embolism (VTE), like age, trauma and immobilisation, inflammation could also be regarded a risk factor for VTE. For example, patients with acute inflammatory conditions (sepsis), but also patients with chronic inflammation, like inflammatory bowel disease (IBD) and rheumatoid arthritis (RA), have an increased risk of thrombosis.

Inflammation can lead to activation of coagulation, and vice-versa, coagulation also has considerable effects on overall inflammatory activity. First, the inflammatory cytokine network induces several pro-thrombotic conditions including insulin resistance, dyslipidaemia, endothelial dysfunction and alteration of coagulation and fibrinolysis. Second, activation of the extrinsic coagulation system and impairment of the fibrinolytic pathway may contribute to amplify and perpetuate the inflammatory response. Previous studies have reported several blood parameters that reflect a prothrombotic state in RA. These include increased levels of thrombin-antithrombin complex, prothrombin fragment F1+2, von Willebrand factor, plasmin-alpha2-antiplasmin complex and D-dimer, as well as an increased platelet count. Impaired fibrinolysis combined with increased antithrombin levels have also been reported in RA. An important mediator in the inflammatory pathway is tumor necrosis factor-α (TNF-α). In the general population, TNF-α induces a disbalance between clotting and fibrinolysis, resulting in a hypercoagulable state. Since TNF-α is the key player in RA, RA is an ideal "human model" to study the interplay between inflammation and coagulation. Hence, RA can be considered as a pro-thrombotic state, which explains partly why patients with RA are at increased risk of thrombo-embolic cardiovascular events.(1)

Only one small study suggested that TNF-inhibitors (TNFi) is accompanied with normalization of thrombotic biomarkers: an improvement of clinical and laboratory parameters as well as a reduction in the activation of coagulation and endothelial dysfunction was found in RA patients treated with a TNFi. In addition, we previously demonstrated that combination therapy with corticosteroids improves the procoagulant state that exists in early RA. (2)

Nowadays, tapering of biological therapies is becoming more and more standard of care. However, the effects on the coagulation status in RA are unknown. In light of the growing evidence of an increased cardiovascular morbidity and mortality in RA, mostly independent of traditional risk factors, treatment strategies in RA should not only aim at relieving symptoms and inhibiting joint destruction but should have a beneficial effect on the vasculature and haemostasis to reduce cardiovascular events. Although modest, there is evidence suggesting a beneficial effect of TNFi on the haemostatic status in RA. Unfavourable changes in haemostatic markers, such as TAT, F1+2, vWF, PAP, D-dimer and thrombin generation, which indicate a pro-thrombotic state, may therefore (re)occur when RA patients stop with TNFi treatment. We first assessed arterial wall inflammation with 18F-FDG PET scans in RA patients in remission under TNFi therapy or DMARD therapy versus controls. The FDG uptake in the aorta in DMARD remission patients was similar to the controls, whereas the uptake in RA patients in remission under antiTNF was significantly higher than in controls either when looking at the overall aortic uptake or the most diseased segment. Theoretically, stopping TNF blockade in these patients might lead to increased inflammation and thus coagulation activation. Therefore, we are presently investigating it and to what extent tapering/stopping TNFi therapy induces a pro-thrombotic state in RA patients.

References:

[1] Van den Oever et al. Ann Rheum Dis. 2014;73:954–7.
[2] Van Den Oever et al Ann Rheum Dis 2012;71(Suppl3):348.
[3] Bernelot Moens et al Arthritis Res Therapy 2016;18: 115.

Disclosure of Interest: None declared

DOI: 10.1136/annrheumdis-2017-eular.7143

FRIDAY, 16 JUNE 2017

MRI I & II

SP0128 MRI OF ENTHESITIS – BY CONVENTIONAL AND WHOLE-BODY MRI - INCLUDING QUIZ CASES

I. Eshed[1,2]. *[1]Diagnostic Imaging, Tel Aviv University, Tel Aviv; [2]Diagnostic Imaging, Sheba Medical Center, Ramat Gan, Israel*

Entheses are sites of attachment of tendons, ligaments, fascia, or capsule into bone, providing a mechanism for reducing stress at the bony interface. Entheses dissipate biomechanical stress and, in doing so, are thought to be subjected to repeated micro traumas.

Inflammation of the entheses, enthesitis, is a well-known hallmark of spondyloarthritis (SpA), playing a central role in disease pathogenesis. It can also be associated with degenerative, endocrinologic, metabolic and traumatic conditions. Magnetic resonance imaging (MRI) is a sensitive tool for the detection of early signs of enthesitis in patients with SpA. The MRI features of enthesitis are well described, and include thickened enthesis with altered signal intensity and perientheseal soft tissue edema. Bone marrow edema and erosions at the adjacent bone appear mainly in SpA-associated enthesitis. Contrast material administration improves the reliability, sensitivity and specificity of detecting enthesitis on an MRI.

Whole-body (WB) MRI allows assessment of all peripheral and axial joints and entheses from "head-to-toe" in one examination. The promising role of WBMRI in the evaluation of enthesitis in SpA and other rheumatic diseases was evaluated in several cross sectional and prospective studies. Indeed WB MRI was shown to be sensitive in the detection of inflammatory lesions, including enthesitis, on multiple sites, potentially serving as a one stop shop for the estimate of active disease load.

In the current presentation, the typical imaging properties of enthesitis on conventional and WB-MRI will be presented along with several challenging quiz cases.

Disclosure of Interest: None declared

DOI: 10.1136/annrheumdis-2017-eular.1911

SP0129 CLASSIFICATION, DIAGNOSIS AND DIFFERENTIAL DIAGNOSIS OF AXIAL SPONDYLOARTHRITIS BY MRI - INCLUDING QUIZ CASES

M. Østergaard. *Copenhagen Center for Arthritis Research, Copenhagen University Hospital Rigshospitalet, Glostrup, Denmark*

Magnetic resonance imaging (MRI) is essential in the process of diagnosis of axial spondyloarthritis (axSpA) in clinical practice, as described in recent EULAR recommendations (1). However, several important differential diagnoses need to be considered. MRI is also key in the Assessment in SpondyloArthritis International Society (ASAS) classification criteria for axSpA (2), for which a consensus definition of a positive MRI was made in 2009 (3). The ASAS MRI working group has recently provided an updated definition of what is needed to fulfill the MRI-criterion in the ASAS criteria (4), based on a consensus excercise. This talk will describe the evidence behind the use of MRI for diagnosis of axSpA, describe the current ASAS consensus on how to use MRI for classification of axSpA, and examples of the most important differential diagnoses will be shown. The presentation will include patient cases for audience review.

References:

[1] Mandl P, Navarro-Compan V, Terslev L et al. EULAR recommendations for the use of imaging in the diagnosis and management of spondyloarthritis in clinical practice. Ann Rheum Dis (Published online) 2015.
[2] Rudwaleit M, Landewe R, van der Heijde D et al. The development of Assessment of SpondyloArthritis international Society classification criteria for axial spondyloarthritis (part I): classification of paper patients by expert opinion including uncertainty appraisal. Ann Rheum Dis 2009; 68(6):770–776.
[3] Rudwaleit M, Jurik AG, Hermann KG, et al. Defining active sacroiliitis on magnetic resonance imaging (MRI) for classification of axial spondyloarthritis: a consensual approach by the ASAS/OMERACT MRI group. Ann Rheum Dis 2009; 68(10):1520–1527.
[4] Lambert RG, Bakker PA, van der Heijde D et al. Defining active sacroiliitis on MRI for classification of axial spondyloarthritis: update by the ASAS MRI working group. Ann Rheum Dis (Published online) 2016.

Disclosure of Interest: None declared

DOI: 10.1136/annrheumdis-2017-eular.7169

FRIDAY, 16 JUNE 2017

WIN & HOT session

SP0130 WIN SESSION: OSTEOPOROSIS

P. Geusens. *Rheumatology, MaastrichtUMC and UHasselt, Genk, Belgium*

The clinical pathway for fracture prevention consists of a 5-step approach: case finding, risk evaluation, differential diagnosis, treatment and follow up. For each of these steps new insights have emerged during the last year.