MRI DIFFICULT CASES OF THE AXIAL SKELETON

Lennart Jans, Ghent University Hospital, Radiology, Gent, Belgium

MRI has revolutionized the assessment of axial spondyloarthritides (SpA) in clinical practice. MRI of the sacroiliac joint is a cornerstone for diagnosis and classification, MRI of the spine may help with difficult cases as spinal changes may anticipate sacroiliac changes and indicate disease burden. In daily practice, however, the interpretation of axial MRI is challenging. Experience has tempered the initial enthusiasm as the limitations of the ASAS criteria in daily practice become evident.

Firstly, the ASAS criteria are intended to classify patients with ‘back pain’ of more than 3 months’ duration and with onset before 45 years of age as having axial SpA. In other patient groups, however, sacroiliitis on MRI as defined by ASAS has a lower sensitivity and specificity.

Secondly, the definition of a ‘positive’ MRI for sacrolitits is validated to limited extend only. MRI of the sacroiliac joint requires inflammatory changes to meet the criteria, without a clear quantitative requirement. Bone marrow edema lacks sensitivity and specificity as MRI findings suggestive of sacroilitis may be produced by non-inflammatory disorders, a point that remains poorly investigated.

Thirdly, structural changes in the sacroiliac joints are not taken into account in the ASAS criteria. The diagnostic performance of MRI of the sacroiliac joint could be improved by including structural lesions, but to date this is not the case.

Fourthly, spinal imaging is not included in the ASAS criteria. MRI of the spine is considered ‘positive’ when at least 3 inflammatory or several structural lesions are present, with a sensitivity and specificity similar to those of the sacroiliac joints. The difficulty here is the lack of data. It has not been reliably shown that inflammation proceeds syndesmophyte formation and MRI seems incapable of accurately evaluating treatment response. Only about 4% of patients with ‘negative’ sacroiliac MRI are reclassified based on positive spinal MRI findings.

Clinicians should be aware of unreasonable expectations on MRI. If MRI findings are considered in isolation the findings are not reliable. The diagnosis of SpA can only be made by an expert if patient’s history, clinical examination, laboratory findings, and imaging studies converge. When solving difficult cases, collaboration is key: clear communication between rheumatologist and radiologist is mandatory. Radiologists should withstand the pressure to call if a patient has SpA or not based on MRI alone.

REFERENCE:

Disclosure of Interests: None declared


Thursday, 13 June 2019
15:00 – 17:00
Ultrasound advanced I
muscle cells undergo continuous cycles of death and regeneration as a consequence of the pressure of the immune response targeting antigens preferentially expressed with regenerating fibers (1, 2).

Objectives: I will review recent evidence on the relative role of unconventional cellular and molecular pathways activated in the tissues of subjects with IIM and in relevant experimental models of skeletal muscle regeneration and autoimmunity, focusing on the action of antigen-specific and regulatory T cells and immune cells populations of innate cells.

Methods: A specialized population of regulatory T (Treg) cells, has been recently characterized in the inflamed and regenerating muscle, which influence both the immune response, by promoting the M1- to M2 switch, and the activation of precursor/stem muscle cells. Treg cells control the efficiency of muscle repair and might be involved in preventing the systemic effects of the autoimmune response generated as a consequence of the disruption of the immunologically protected environment of the skeletal muscle (6, 7).

Conclusion: Identification of the pathways that are physiologically involved in restricting the onset and accelerating the termination of autoimmune responses targeting the skeletal muscle might be valuable for the identification of novel targets for molecular intervention in patients with IIM.

REFERENCES:

Disclosure of Interests: None declared

SP0106
REGULATION OF AUTOANTIBODY ACTIVITY BY T CELL SUBSETS
Gerhard Köröke, University Hospital Erlangen, Internal Medicine 3, Erlangen, Germany

Our recent data show that the IL-23–T(H)17 axis controls the intrinsic inflammatory activity of autoantibodies and thereby triggers the clinical onset of autoimmune arthritis in mice and humans suffering from rheumatoid arthritis. TH17 cells regulate expression of sialyltransferases in newly differentiating antibody-producing cells and determine the glycosylation profile and activity of immunoglobulin G (IgG) that is produced by the consecutively emerging plasma cells.

Disclosure of Interests: Gerhard Köröke Grant/research support from: Lilly, Pfizer, Speakers bureau: Novartis

FRIDAY, 14 JUNE 2019
08:15:00 – 09:45:00
Seeing is believing: Nanotechnologies in tissue imaging

SP0104
THE JANUS-FACED GLADIATOR: NEUTROPHILS IN STERILE INFLAMMATION AND AUTOIMMUNITY
Markus Hoffmann, University of Maryland, School of Medicine, United States of America

Background: Neutrophils, and particularly neutrophil extracellular traps (NETs), have been connected with inflammatory and autoimmune diseases, such as RA, SLE, gout and many others. However, the outcome of a deficiency of NET formation can be observed in chronic granulomatous disease and Papillon-Lefèvre syndrome and is characterized by non-resolving inflammation and frequent autoimmunity.

Objectives: I will give an overview over pro- and anti-inflammatory effects of neutrophils and NETs, respectively, focusing on our results in gout and SLE.

Methods: We investigated the impact of NET-deficiency and/or neutrophil-depletion on animal models of SLE (pristane-induced lupus) and gout (MSU crystal-induced arthritis). Mechanistic studies were performed with isolated neutrophils from mice and humans.

Results: In experimental lupus and gouty arthritis neutrophil depletion or neutropenia worsens disease. Also mice strains with normal neutrophil numbers but defects in NET formation (NOX2 or PAD4-deficient mice) have exacerbated and unresolved inflammation. Release of inflammatory mediators by stimulated neutrophils was highest at intermediate cell densities (20–40 x 10^6 cells/cm^3). Above such densities, mediator release by normal neutrophils was outweighed by degradative degradation via proteases expressed on aggregated NETs. Additionally, binding to aggregated NETs conferred protection of the serine protease neutrophil elastase (NE) against inactivation by α-antitrypsin (AAT).

Conclusion: Although neutrophils are often regarded as archetypical pro-inflammatory cells, evidence is increasing that they can also exert anti-inflammatory and immunomodulatory function. Formation and aggregation of NETs and degradation of inflammatory mediators by serine proteases are important neutrophil tools to resolve gouty arthritis and other forms of localized inflammatory conditions. On the downside, NETs come at the price of collateral tissue damage and may cause occlusion of ductal structures.

REFERENCES:

Disclosure of Interests: None declared