showed that joint inflammation is common in ochronotic patients, associated in some cases with peripheral enthesis involvement confirming previously published data (1). The prevalence and the characteristics of the inflammatory manifestations should be further studied in larger cohorts of patients as they could play an important role in the joint damage process in these patients and provide a rationale for the use of new drugs.

References:
[1] Filippou G, Frediani B, Selvi E et al, Tendon involvement in patients with ochronosis: an ultrasonographic study. Ann Rheum Dis 2008 Dec;67(12): 1785–6.

Disclosure of Interest: None declared
DOI: 10.1136/annrheumdis-2017-eular.6678

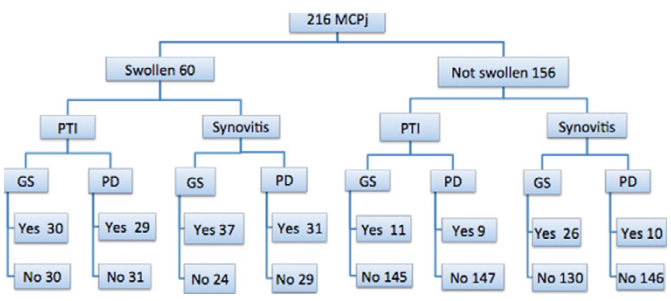
SAT0626 METACARPOPHALANGEAL JOINT SWELLING IN PSORIATIC ARTHRITIS: WHAT DOES IT MEAN?

C. Macía[1], S. Falcao[2], M. Gutierrez[3], J. Medina[4], H.B. Hammer[5], E. De Miguel[6]. *[1]Rheumatology, Hospital Universitario Severo Ochoa, Madrid, Spain; [2]Rheumatology, Chronic Diseases Study Center (CEDOC), NOVA Medical School, UNL, Lisboa, Portugal; [3]Rheumatology, Division of Musculoskeletal and Rheumatic Disorders. Instituto Nacional de Rehabilitación, Mexico City, Mexico; [4]Rheumatology, Hospital Clínico Universitario, Valladolid, Spain; [5]Rheumatology, Diakonhjemmet Hospital, Oslo, Norway; [6]Rheumatology, Hospital Universitario la Paz, Madrid, Spain*

Background: Clinical metacarpophalangeal joint (MCPj) swelling is a frequent finding in psoriatic arthritis (PsA). It is assumed to be caused by intra-articular synovitis (IAS). However, ultrasound (US) have also demonstrated in PsA peritenon inflammation of the extensor digitorum tendon (PTI).To date the data about the significance of this two lesions (IAS and PTI) in MCPj swelling are sparse.
Objectives: Our objective was to explore PTI and IAS as the cause of clinical MCPj swelling in PsA patients.
Methods: 27 consecutive non selected PsA patients, fulfilling CASPAR criteria, with clinical involvement of at least one 2nd to 5th MCPj were included. A MyLab 70 XVG machine, Esaote, Genova, Italy, with a greyscale (GS) 13 MHz probe, and a 7.1 MHz power Doppler (PD) frequency (PRF 750 Hz, Gain 60) was used. Videos (3–5 sec) of each MCPj 2nd to 5th of both hands in transversal and longitudinal views were obtained for central reader analysis, scoring US as presence or absence in GS and PD of: 1) PTI (defined as an hypoechoic swelling of the soft tissue surrounding the extensor tendon at MCPj level with or without PD) and 2) IAS (OMERACT definition). US pathology for each joint and lesion was defined as at least three of five central readers having the same score. SPSS analysis was performed for frequencies, percentage of agreement and Cohen's Kappa test.
Results: 27 PsA patients with a mean (SD) age of 56 (11) years and disease duration 109 (101) months were included. Isolated peripheral involvement was present in 21 patients (78%) and 6 (22%) had both axial and peripheral affection. Mean (SD) CRP level was 8.3 (8.2) mg/l and ESR 21.9 (19.3) mm. The mean DAS28 ESR was 3.88 (1.23). Psoriasis involvement included skin and nails in 15 (55.5%) of the patients.
A total of 216 MCPj were examined, with 60 (27,7%) being clinically swollen. The figure illustrates the agreement between clinical and US assessments, and the table shows the kappa values for the agreements. PTI in at least one MCPj was found in 19/27 patients (70%) with a total of 41/216 locations (19%) in GS. For IAS, there was GS in at least one MCPj in 23/27 patients (85%) with a total of 63/216 locations (29%). 28 of 41 (68.3%) joints had both PTI and IAS.

Table 1. US findings versus Clinical joint swelling

	Agreement n (%)	Kappa
Any US lesion (vs. clinical swelling)	167/216 (77.3%)	0.471
Any grey scale lesion (vs. clinical swelling)	166/216 (76.8%)	0.426
Any power Doppler lesion (vs. clinical swelling)	169/216 (78.2%)	0.550
Grey scale IAS (vs. clinical swelling)	166/216 (76.8%)	0.426
Power Doppler IAS (vs. clinical swelling)	177/216 (81.9%)	0.499
Grey scale PTI (vs. clinical swelling)	175/216 (81.0%)	0.474
Power Doppler PTI (vs. clinical swelling)	176/216 (81.5%)	0.478



Conclusions: Our study identifies two different US lesions (IAS and PTI) causing clinical joint swelling. PTI is near as frequent as IAS as a cause of MCPj swelling, and future studies are necessary to explore the added value of assessing PTI for prognosis or treatment.

Disclosure of Interest: None declared
DOI: 10.1136/annrheumdis-2017-eular.1437

SAT0627 MUSCLE BIOPSY: MASTER ROLE IN DIFFERENTIAL DIAGNOSIS IN PATIENTS WITH SUSPECTED MYOPATHY

D. Santos-Faria, J. Sousa-Neves, M. Cerqueira, J.L. Silva, J.R. Rodrigues, D. Peixoto, J. Tavares-Costa, S. Alcino, C. Afonso, F. Teixeira. *Rheumatoloy, Unidade Local de Saúde do Alto Minho, Ponte de Lima, Portugal*

Background: The muscle biopsy may be a fundamental technique in the suspicion of myopathy, with high specificity to distinguish between normal or abnormal muscle tissue. In association with clinical and laboratory findings, the muscle biopsy has an important role to a more accurate diagnosis.
Objectives: To evaluate the usefulness and safety of muscle biopsies performed in a Rheumatology Unit in patients with suspected myopathy.
Methods: Retrospective analysis of the clinical charts of patients submitted to muscle biopsy between January 2010 and December 2016 at our Rheumatology Unit. Demographic, clinical, laboratory, electromyographic and histological data were collected. The histological study was performed in a Neuropathology Specialized Unit.
Results: A total of 46 patients, 19 men and 27 women, with a mean age of 53.3±17.1 years, were evaluated. Clinical manifestations included muscle weakness, myalgia and decreased muscle strength. Most patients also had increased muscle enzymes, particularly creatine kinase, but in a patient with generalized muscle atrophy, muscle enzymes were overall diminished. Of the 46 biopsies, 12 (26.1%) did not show alterations, 8 (17.4%) showed nonspecific alterations and only 1 biopsy was not conclusive because the sample was inadequate. In 4 patients, the histological features did not present specific characteristics of a myopathy, but revealed a preferential atrophy of type 2 fibbers, usually associated with prolonged corticosteroid therapy. Among the others, 9 (19.6%) were compatible with inflammatory myopathies, namely polymyositis (6), dermatomyositis (1), inclusion body myopathy (1), and localized nodular myositis (1). In the latter case, the patient had a different clinical presentation, with intermittent episodes of pain, oedema and flushing of different muscle groups. In addition, 5 metabolic myopathies (2 McArdle's diseases and 3 non-specific metabolic disorders), 2 muscular dystrophies (1 Becker's muscular dystrophy and 1 dystrophinopathy), 1 suspected case of myotonic dystrophy and 1 myopathy associated with statins use were diagnosed. In a patient with muscle weakness and prior diagnosis of systemic vasculitis, the histology showed a chronic inflammatory process with no specific alterations. In the patient with overall decrease in muscle enzymes, the biopsy revealed neurogenic atrophy, without inflammatory infiltrates. Overall, the results of electromyography (EMG) did not correlate with the histological findings, because EMG identified alterations both in cases with histologically compatible inflammatory myopathy and in cases without histological pathology. On the other hand, EMG did not reveal any changes in some of the metabolic myopathies. Muscle biopsies were performed mainly in the deltoid muscle. There were no relevant immediate or late complications with this technique.
Conclusions: Although muscle biopsy is an invasive technique, it is a safe technique and allows the differential diagnosis between the various myopathies, which is fundamental to an appropriate treatment.

References:
[1] Michelle EH, Mammen AL. Myositis Mimics. Curr Rheumatol Rep. 2015; 17(63):1–8.
[2] Barsotti S, Terenzi R et al. One year in review 2015: idiopathic inflammatory myopathies. Clin Exp Rheumatol. 2015;33(5):593–601.

Disclosure of Interest: None declared
DOI: 10.1136/annrheumdis-2017-eular.5069

SAT0628 INCREASE OF CORTICAL MICRO-CHANNELS (COMICS) AS A NEW FEATURE OF STRUCTURAL DAMAGE IN PAIENTS WITH RHEUMATOID ARTHRITIS

D. Werner[1,2], D. Simon[1], M. Englbrecht[1], F. Stemmler[1], A. Berlin[1], J. Haschka[3], N. Renner[4], T. Buder[5], J. Rech[1], K. Engelke[6], A.J. Hueber[1], G. Schett[1], A. Kleyer[1]. *[1]Department of Internal Medicine 3 – Rheumatology and Immunology, Universitätsklinikum Erlangen, Friedrich-Alexander-University Erlangen-Nürnberg (FAU), Erlangen; [2]Rheumatologie & Nephrologie, Städtisches Klinikum Dresden, Dresden, Germany; [3]St.Vincent Hospital, Medical Department, Academic Teaching Hospital of Medical University of Vienna, Vienna, Austria; [4]Department of Trauma and Orthopedic Surgery, Friedrich-Alexander-Unviersity Erlangen-Nuremberg; [5]Institute of Anatomy, Friedrich-Alexander-University Erlangen-Nürnberg (FAU); [6]Institute of Medical Physics, Friedrich-Alexander-University Erlangen-Nuremberg Erlangen, Erlangen, Germany*

Background: Bone damage in rheumatoid arthritis (RA) typically emerges at certain anatomical hotspots corresponding to the so-called "bare area", an intra-articular region between the cartilage and the insertion site of the joint capsule (1,2). We hypothesized that this region exhibits certain micro-anatomical properties, which facilitates the emergence of bone erosions.