Her other background includes beta-thalassaemia trait and excision of calcific fibrotic tissue on bilateral anterolateral orbits in 2015.
In 2018 she reported an 18-month history of non-tender, non-fluctuant, slow growing left thigh mass with USS revealing a well demarcated subcutaneous complex cystic lesion of ~2x4x7cm. There was no preceding trauma or skin infection. Histology from a needle biopsy revealed diffuse histiocytosis with positive immunohistochemistry (ICH) for S100, CD68 and CD31, it was negative for CD1a, consistent with **Extra-nodal Rosai-Dorfman disease (RDD)**.
She developed constitutional symptoms after reporting months of gradual weight loss with gradual ESR, CRP rise and leucocytosis. Her SLE symptoms were stable and given lack of SLE-specific symptoms; PET-CT was used to identify systemic RDD; the thigh mass showed strong FDG avidity along with a small focus of uptake in the small bowel, thought to be RDD related with no other areas of uptake.
She had ongoing ooze from the enlarging thigh lesion (5 x 26 x 15 cm), this was sent for MCS and AAFB; which isolated **Mycobacterium avium**. She was treated with rifampicin, ethambutol and clarithromycin resulting in improved thigh lesion, constitutional symptoms and inflammatory markers.

Objectives:

[1] To describe a rare associated complication of severe SLE and to educate and inform clinicians regarding possible masquerades of disease
[2] To education and inform about the approach to diagnosis of mycobacterium infection.

Methods: Case report and literature review.

Results: Mycobacterium infections rarely complicate RDD; to date, only one case report is published involving an HIV infected patient with RDD confirmed on LN biopsy presenting with splenomegaly and treated with oral corticosteroids (OCS) complicated by *Mycobacterium avium complex* and *Salmonella enterica* confirmed on bone marrow biopsy/culture, similar to our patient, he presented with constitutional symptoms and weight loss[(2)].
Mycobacterium can also mimic RDD, a case report has described a 74 year old with tender lymphadenopathy diagnosed with RDD on LN biopsy. She was treated with IV and OCS, but was unresponsive. A repeat LN biopsy and CT imaging revealed the presence of *mycobacterium kansasii;* her biopsy was positive for CD68/S100 throughout. Of note, she had high levels of anti-interferon autoantibodies and was diagnosed with adult-onset immunodeficiency syndrome[(3)].

Conclusion: This case illustrates the need for a MDT approach for multi-system diseases such as SLE and RDD, and the need to consider atypical infections when blood tests are incongruent with clinical state.

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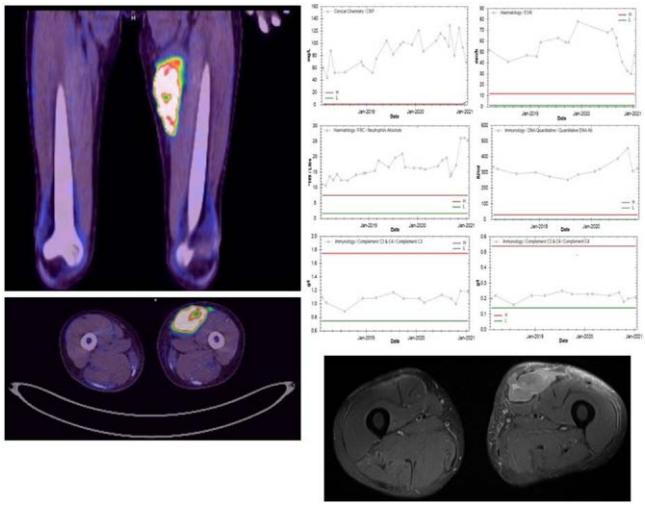

Image 1.

Table 1.

SLE Clinical History
Diagnosed 2006 ('97 ACR Classification Criteria)
Clinical
-Polyarthritis
-Glandular (lacrimal swelling)
-Pericardial effusion/Pleural Effusion
-Myositis
Serological
-Anti-nuclear antibody (ANA)
-Anti-dsDNA
-Anti-U1-RNP
-Anti- SS-A/Ro
-Lupus Anticoagulant
Previous SLE Treatment
-Hydroxychloroquine (HCQ)
-Methotrexate (MTX)
-Azathioprine (AZA)
-Rituximab (RTX)

Disclosure of Interests: None declared
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Challenges for Patients and Patients' Organisations in Times of the Pandemic

OP0082-PARE THE EFFECT COVID-19 HAS ON THE MENTAL HEALTH OF PEOPLE LIVING WITH RHEUMATIC DISEASES. FROM DATA TO INTERVENTIONS.

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Background: Covid-19 has had an important impact on the mental health conditions of over 5 million Italians suffering from one of the over 150 rheumatic diseases. In order to understand the psychological impact of the Covid-19 emergency and the restrictions imposed to counter it, the Italian National Association of People with Rheumatic and Rare Diseases – APMARR APS launched the research "Living with a rheumatic pathology".

Objectives: Gather data directly from Italian patients about the impact of the COVID-19 and consequent restrictions on their mental health and feelings; evaluate the most effective intervention to be implemented to face the pandemic by Patients organization.

Methods: A qualitative-quantitative survey was carried out through a questionnaire administered throughout the national territory to a sample of N = 1,001 people. The people invited to complete the questionnaire were women (55,9%) and men (44,1%), aged 18-85 years (age 18-41=26,7%; age 42-65=64%; age >65=9,3%) with at least one rheumatic pathology. The questionnaire was made up of 39 questions, of which 29 were closed and 10 were open. For the administration of the questionnaires, the CAWI (Computer Aided Web Interview) methodology of on-line survey was used. The 1,001 interviews were carried out from 7 to 14 August 2020.

Results: More than 4 out of 10 people (total sample 44.2%; male 60%, female 35,7%; age 18-41=39,1%; age 42-65=45,9%; age >65 = 50%) declared that the emergency period has somehow caused a worsening of their health condition. People declared that the deterioration of their health is due to the emergency period for the following reasons: 1) Psychological: such as stress and anxiety: "Too much stress and anxiety made the symptoms worse."; "The stress of the quarantine affected my problem"; "Insomnia. Nervousness. General ailments. Depression. Strong stress" 2) Inability to perform physiotherapy and motor activities due to the lockdown 3) Postponement of examinations, visits and checks 4) remote working, in some cases described as harmful for people's mental and physical health: "Due to Covid19 I had to do remote working and I worked even 12 hours a day including holidays to the detriment of my family life".
Furthermore, from January 31, 2020 a significant increase emerged in communication problems with rheumatology specialist compared to the period before the emergency due to Covid-19. The sharp increase may be due to the situation of severe psychological stress to which also the doctors were subjected in the emergency phase: people could not find the comfort of being empathically listened to.

Conclusion: The research shows that the most frequent symptoms among people with rheumatic diseases were depression and high levels of anxiety due to strong emotional stress. Psychological malaise caused direct effects in worsening the symptoms of rheumatic disease as well as other related effects, for example, insomnia. The forced isolation due to the lockdown has made people lack the social support that is fundamental for the psychological well-being especially for those suffering from some chronic pathology. Starting from the data collected, APMARR promptly activated a completely free psychological support service with 6 professional psychologists, two of them specialized in emergency psychology. The service is accessible online and is still going on for all who are not able to overcome the anxiety and fear related to the pandemic and its evolution. Thousands of accesses to the service have been measured to date.

References: S Mingolla[1], A Celano[1], M Santopietro[2]

[1] NATIONAL ASSOCIATION OF PEOPLE WITH RHEUMATIC AND RARE DISEASES - APMARR APS

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Disclosure of Interests: None declared

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The origins of pain in RMDs

OP0083 INFECTIOUS AND AUTOINFLAMMATORY MODIC TYPE 1 CHANGES HAVE DIFFERENT PATHOMECHANISMS

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Background: Modic type 1 changes (MC1) are vertebral bone marrow (BM) edema that associate with non-specific low back pain (LBP). Two etiologies have been described. In the infectious etiology the anaerobic aerotolerant *Cutibacterium acnes* (*C. acnes*) invades damaged intervertebral discs (IVDs) resulting in disc infection and endplate damage, which leads to the evocation of an immune response. In the autoinflammatory etiology disc and endplate damage lead to the exposure of immune privileged disc cells and matrix to leukocytes, thereby evoking an immune response in the BM. Different etiologies require different treatment strategies. However, it is unknown if etiology-specific pathological mechanisms exist.

Objectives: The aim of this study was to identify etiology-specific dysregulated pathways of MC1 and to perform in-depth analysis of immune cell populations of the autoinflammatory etiology.

Methods: BM aspirates and biopsies were obtained from LBP patients with MC1 undergoing spinal fusion. Aspirates/biopsies were taken prior screw insertion through the pedicle screw trajectory. From each patient, a MC1 and an intra-patient control aspiration/biopsy from the adjacent vertebral level was taken. If *C. acnes* in IVDs adjacent to MC1 were detected by anaerobic bacterial culture, patients were assigned to the infectious, otherwise to the autoinflammatory etiology.

Total RNA was isolated from aspirates and sequenced (Novaseq) (infectious n=3+3, autoinflammatory n=5+5). Genes were considered as differentially expressed (DEG) if p-value < 0.01 and log2fc > ± 0.5. Gene ontology (GO) enrichment was performed in R (GOseq), gene set enrichment analysis (GSEA) with GSEA software.

Changes in cell populations of the autoinflammatory etiology were analyzed with single cell RNA sequencing (scRNAseq): Control and MC1 biopsies (n=1+1) were digested, $CD45^+CD66b^-$ mononuclear cells isolated with fluorescence activated cell sorting (FACS), and 10000 cells were sequenced (10x Genomics). Seurat R toolkit was used for quality-control, clustering, and differential expression analysis.

Transcriptomic changes (n=5+5) of $CD45^+CD66b^+$ neutrophils isolated with flow cytometry from aspirates were analyzed as for total bulk RNAseq. Neutrophil activation (n=3+3) was measured as $CD66b^+$ expression with flow cytometry. $CD66b^{high}$ and $CD66b^{low}$ fractions in MC1 and control neutrophils were compared with paired t-test.

Results: Comparing MC1 to control in total bulk RNAseq, 204 DEG in the autoinflammatory and 444 DEG in the infectious etiology were identified with only 67 shared genes (Fig. 1a). GO enrichment revealed "T-cell activation" (p = 2.50E-03) in the autoinflammatory and "complement activation, classical pathway" (p=1.1E-25) in the infectious etiology as top enriched upregulated biological processes (BP) (Fig 1b). ScRNAseq of autoinflammatory MC1 showed an overrepresentation of T-cells (p= 1.00E-34, OR=1.54) and myelocytes (neutrophil progenitor cells) (p=4.00E-05, OR=2.27) indicating an increased demand of these cells (Fig. 1c). Bulk RNAseq analysis of neutrophils from the autoinflammatory etiology revealed an activated, pro-inflammatory phenotype (Fig 1d), which was confirmed with more $CD66b^{high}$ neutrophils in MC1 (+11.13 ± 2.71%, p=0.02) (Fig. 1e).

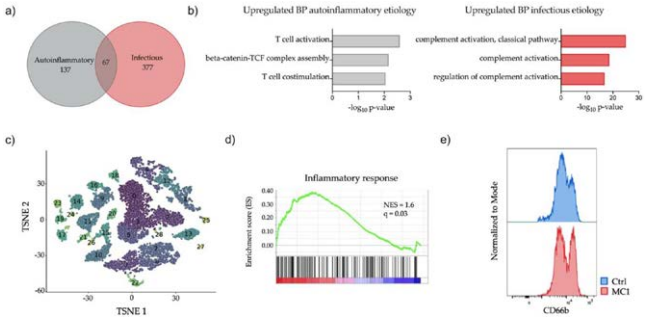

Figure 1. (a) Venn diagram of DEG from total bulk RNAseq (b) Top enriched upregulated BP of autoinflammatory (left) and infectious (right) etiology (c) Cell clustering of autoinflammatory MC1 BM (d) Enrichment of "inflammatory response" gene set in autoinflammatory MC1 neutrophils (e) Representative histogram of $CD66b^+$ expression in MC1 and control neutrophils.

Conclusion: Autoinflammatory and infectious etiologies of MC1 have different pathological mechanisms. T-cell and neutrophil activation seem to be important in the autoinflammatory etiology. This has clinical implication as it could be explored for diagnostic approaches to distinguish the two MC1 etiologies and supports developing targeted treatments for both etiologies.

Disclosure of Interests: None declared

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OP0084 CENTRAL MECHANISMS TRAIT PREDICTS PERSISTENT KNEE OSTEOARTHRITIS PAIN AT 24-MONTHS: DATA FROM THE OSTEOARTHRITIS INITIATIVE

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Background: In the UK, 10% of men and 18% of women over the age of 60 suffer from symptomatic osteoarthritis (OA), and rising. OA knee pain can worsen without significant radiographic changes and pain remains a major problem for up to 20% of patients after total knee joint replacement. Chronic knee OA pain is