recommended by the Canadian guidelines [3], which recommended "patients should undergo a physical examination which should be within normal limits except for tenderness on pressure of soft tissues however the specific tender point examination according to the 1990 ACR diagnostic criteria is not required to confirm a clinical diagnosis of fibromyalgia". Furthermore, it stated that "Fibromyalgia should be diagnosed as a clinical construct, without any confirmatory laboratory test, and with testing limited to simple blood testing including a full blood count, erythrocyte sedimentation rate, C-reactive protein, creatine kinase, and thyroid stimulating hormone. Any additional laboratory or radiographic testing should depend on the clinical evaluation in an individual patient that may suggest some other medical condition." However, many primary care physicians do not feel competent/confident to exclude other rheumatic conditions by physician examination.

Over the last decade, comorbid FM is increasingly recognized in many chronic musculoskeletal diseases including patients with inflammatory arthritis. A systematic review found that the prevalence of FM is 21%, 13% and 18% in RA, axSpA and PsA respectively [4]. Patients with comorbid FM have higher diseases activity score and more severe pain.

In 2016, the ACR diagnostic criteria for FM were modified and stated that "A diagnosis of fibromyalgia is valid irrespective of other diagnoses. A diagnosis of fibromyalgia does not exclude the presence of other clinically important illnesses." [5].

Conclusion: The motion that "FM is a diagnosis of exclusion" is no longer valid. FM should be a positive diagnosis and does not exclude other rheumatic diagnosis.

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SP0120 PERSONALISED TREATMENT FOR FIBROMYALGIA; THE IMPACT OF COMORBIDITIES

Serge Perrot. France, France

Background: The heterogeneity of the clinical presentation, the co-morbidities and the pathophysiologic mechanisms associated with fibromyalgia (FM), and the modest results on average for any therapy, call for a more individualized management strategy.

How to develop personalized treatment?

Individualized treatment can be developed on the basis of subgrouping of patients according to associated conditions and co-morbidities (mental health problems, chronic overlapping pain conditions, other somatic diseases) or on disease severity. Personalizing treatment needs to classify FM in specific subgroups. It can be proposed on the basis of clinical assessment (eg, degree of daily functioning), on co-morbidities or on dedicated questionnaires. Lastly, shared decision-making regarding treatment options can be directed according to patient preferences, co-morbidities, and availability in various health care settings.

What is currently recommended as a personalized approach?

The European League Against Rheumatism guidelines recommend a tailored approach directed by FM key symptoms (pain, sleep disorders, fatigue, depression, disability), whereas the German guidelines recommend management tailored to disease severity, with mild disease not requiring any specific treatment, and more severe disease requiring multicomponent therapy (combination of drug

treatment with aerobic exercise and psychological treatments). When indicated, treatments should follow a stepwise approach beginning with easily available therapies such as aerobic exercise and amitriptyline. We will present how successful application of a tailored treatment approach that is informed by individual patient characteristics should improve outcome of FM.

Key points: This presentation will presents suggestions for an individualized treatment strategy for FM patients on the basis of symptoms, disease severity and co-morbidities.

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FRIDAY, 14 JUNE 2019

13:30:00 – 15:00:00

Joint EULAR – EFIS: Combatting or harnessing ILC in the battle against autoimmunity

SP0121 DEBATE: ILC AS INNOCENT BYSTANDER

Mikael Ebbo. Hopital de la Timone, AP-HM, Aix-Marseille Université, Internal Medicine, Marseille, France

Innate lymphoid cells (ILC) are recently (10 years ago) described tissue-resident lymphoid populations [1]. They lack adaptive antigen receptor (generated by the recombination of genetic elements) and are innate counterparts of T lymphocytes. Indeed ILC1s, ILC2s, and ILC3s mirror CD4⁺ T helper (Th)1, Th2, and Th17 cells, respectively, in terms of transcription factor expression and cytokine production.

In this lecture, an overview of the different protective immunological functions of these helper ILC will be provided, in particular their role for maintenance of gut homeostasis, for control of parasitic, bacterial or viral infections, for anti-tumor immune responses, for metabolism, or for tissue repair mechanisms. The concept of ILC plasticity, which represents the capacity of these cells to change their phenotype and functional capacities in response to diverse microenvironment stimuli, will be also addressed. It directs the heterogeneity of these cells in the tissues, and may be important for their effective immune responses.

In this context, targeting ILC for therapeutic use, for example in the battle against autoimmune or inflammatory diseases in which they may also play roles [2], could be associated with deleterious consequences. However, recent data concerning patients with a profound and selective ILC deficiency and no susceptibility to disease will be also presented here. This last observation suggests that most protective functions mentioned above may be redundant and potentially dispensable for human health, at least in modern conditions of hygiene and medicine, when T cell and B cell functions are preserved.

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FRIDAY, 14 JUNE 2019

13:30:00 – 15:00:00

Optimizing the access to new treatments for RMD patients

SP0122 OPTIMIZING HOLISTIC TREATMENTS: THE CHALLENGE OF IMPLEMENTATION

Krysia Dziedzic. Keele University, School for Primary, Community and Social Care, Stoke on Trent, United Kingdom

International guidelines present recommendations for holistic care for RMDs, building upon best evidence. However, there is still a gap between what we know about best care and what we do to implement best care in our clinical services. Having EULAR guidelines alone (and multiple updates and revisions) is not enough to change complex systems. European funders are now investing in projects to accelerate the uptake of best care across multiple European partners.

Using one example of a completed EIT-Health funded implementation project, JIGSAW-E (Joint Implementation of Guidelines for oSteoArthritis in Western Europe), this presentation will highlight some of the challenges of implementation such as: understanding the context; competing priorities; lack of time and funding; stability of the workforce; and differing health care systems. It will describe the use of Knowledge Mobilization and Communities of Practice as methods for overcoming these challenges and optimizing holistic treatments.

The presentation will offer some of the key factors for implementation such as 'leading from the middle' and brokering across silos; working with patients and the public '*Patients are the most under-used resource in any health care system*'; using theories, models and frameworks to plan, understand and evaluate implementation; using pilot sites to build momentum with dedicated Communities of Practice; and agreeing what success should look like.

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SP0123 NEW TREATMENTS: WHY SHOULD WE WAIT?

Daniel Morales. *University of Dundee, Population Health and Genomics, Dundee, United Kingdom*

Background: Improving access to new and established medicines for rheumatological disease is of public health importance. However, drug development processes, medicines regulation and health technology requirements may significantly impact on when a new medicine become available to be used by health care professionals.

Objectives: The objectives of this session are to present some of the challenges and opportunities for accessing new and established medicines for rheumatological disease.

Methods: Drug development processes, medicines regulation and health technology requirements may significantly impact on when a new medicine become available to be used by health care professionals. Although these requirements may be perceived as barriers, many of these requirements are in place to ensure a medicines benefit risk profile is favorable and is cost effective in terms of quality of life. This session will briefly discuss issues around the process of making medicines newly available from drug development to gaining new market approvals with an emphasis on rheumatological disease, including the post-authorization safety requirements that are in place to safe guard public health.

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New assessments in clinical practice

SP0124 WHICH MEASURES TO USE TO ASSESS REMISSION IN RHEUMATOID ARTHRITIS

Annamaria Iagnocco. *Academic Rheumatology Unit Università degli Studi di Torino, Academic Rheumatology Centre, Turin, Italy*

Remission is the ideal target of treatment in rheumatoid arthritis (RA) patients. However, the definition and characterization of remission is still a theme of discussion and its assessment can be a challenging issue in RA clinical practice. Indeed, nonetheless the new treatments and therapeutic strategies for RA make remission possible, sub-clinical inflammation can still be present both at the joint site and at the patient level, leading to damage progression and to occurrence of flares.

Clinically, remission entails persistent absence of any sign of joint and systemic inflammation. However, the presence of some residual joint inflammatory activity can be accepted by current clinical indexes such as DAS28, SDAI, CDAI and Boolean remission, although the latter is clearly based on more stringent parameters (1,2). Based on those criteria, and particularly with the use of SDAI and Boolean cut-offs, complete and persistent remission is then quite uncommon in late RA, while it seems to be more easily possible in early disease (3). Another interesting issue to consider in clinical practice is the presence of sustained remission which seems to occur less frequently than remission at different point times and with only a minority of cases reaching a complete disease recovery (4).

Depending on the modality that is used, the assessment of remission by imaging encompasses the evaluation of a wide spectrum of lesions. While ultrasound is able to show intra- and peri-articular signs of inflammation and to differentiate active from inactive synovitis with the use of Doppler modes, MR shows also bone marrow inflammatory-related abnormalities. Moreover, ultrasound detects inflammation at multi-site level and allows the measurement of stable remission over

time on longitudinal evaluation. Interestingly, also tenosynovial Doppler inflammation has been demonstrated to be a frequent finding in patients in clinical remission and, compared to joint synovitis, it is more associated with self-perceived unstable remission (5). In addition, tenosynovial and joint Doppler positivity has been demonstrated to be an independent risk factor of flare in RA patients in remission (6). Residual joint inflammation in patients in remission can be demonstrated also by other imaging techniques, such as PET which shows enhanced tracer uptake at different joint sites (3). Basically, the capability of imaging to demonstrate sub-clinical disease activity even in patients in clinical remission, by allowing the detection of residual inflammation at different joint sites and at different disease stages gives it an added value compared to clinical assessment alone.

In conclusions, clinical evaluation of remission alone appears frequently inadequate to capture a real persistent lack of inflammation at different joint sites. Imaging can complement the assessment of patients in clinical remission, offering additional data that may still reveal a certain amount of sub-clinical disease activity. With new therapeutic strategies RA management has become increasingly complex over the last years (7). The relevance of this information is then substantial, being structural damage the result of both previous and current phases of disease activity.

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SP0125 HOW TO ASSESS PATIENT WITH SUSPECTED VASCULITIS

Raashid Luqmani. *University of Oxford, Nuffield Department of Orthopaedics, Rheumatology and Musculoskeletal Sciences, Oxford, United Kingdom*

Background: Systemic vasculitides are multi-organ, multi-system conditions which can mimic many other diseases. They are rare but very significant because they can rapidly lead to end organ damage or death if left untreated. The difficulty faced by clinicians in diagnosing a patient with vasculitis is further compounded by the presentation of limited forms of vasculitis, which can be difficult to recognise as due to vasculitis, the effects of co-existing co-morbidity and the use of drugs either for medicinal or recreational purposes. Giant cell arteritis is one of the most common serious forms of vasculitis in adults, leading to blindness if untreated. In view of this risk, many clinicians will initiate treatment of any patients with suspected giant cell arteritis before definitive investigations have been performed. Unfortunately this can result in inadequate outcomes from diagnostic tests such as ultrasound or biopsy of the temporal artery. There are no diagnostic criteria for most forms of vasculitis apart from Behcet's syndrome. Some tests can be very useful in ruling out other causes as well as helping to confirm a diagnosis of vasculitis. We will review some of the strategies for diagnosing vasculitis, complementing a thorough history and examination with selected investigations to improve diagnostic certainty.

Objectives: To consider the strategies used to assist the diagnosis of systemic vasculitis; to assess the frequency of multi-system involvement in systemic vasculitis; to critically evaluate the role of diagnostic testing in systemic vasculitis.

Methods: A review of current literature on the approach to diagnosis and classification of vasculitis.

Results: Table 1 summarises the clinical manifestations of some forms of primary systemic vasculitis, comparing the different frequencies of the common presenting features. Patients with large vessel vasculitis are usually more distinct as a clinical group from other forms of vasculitis but increasingly we see an overlap between GCA and older patients (especially males) with Takayasu arteritis. There is considerable overlap of clinical features amongst the small vessel vasculitides, despite different immuno-pathogenetic pathways. Amongst the anti-neutrophil cytoplasm antibody (ANCA) associated vasculitides (AAV), the presence of active nephritis characterises most patients with MPA; it is less common in GPA and least common in EGPA. Lung involvement is prevalent in all three forms of AAV, but has different characteristics: bronchial wall inflammation with ulceration plus nodules and infiltrates in GPA; transient infiltrates and bronchospasm in EGPA; lung haemorrhage at presentation in MPA, with subsequent risk of developing lung fibrosis. The different patterns of disease activity help in differentiating