only disrupted when I realised that my experience of illness was quite common to other persons with a RMD. I can still remember my fascination, in a Patient Reported Outcomes workshop, by discovering that our experience as patients had a bigger relevance that the one we usually conceive.

Then, I felt the need of understanding the research process and specific jargon, so I could relate them with my knowledge on living with a RMD. Otherwise, any insight on the patient perspective would be "lost in translation", due to an inability to establish a correspondence between the scientific terminology and mindset regarding the disease, and the experience of illness and sickness by the individuals. Training opportunities were also recognised as desirable by the above mentioned EULAR recommendations, to increase expertise and understanding of research methods and to promote the patients' self-confidence on their contribution to research.

The *Patient Research Partners* (PRP) training by EULAR, in 2013, was the first step towards a better understanding of what could be the role of patients and on how to provide a meaningful input from the patients' perspective into research processes. It was followed in 2014 by the training provided by European Patients' Academy on Therapeutic Innovation (EUPATI) for *Patient Expert on the Medicines Research and Development Process*. This expert-level training was organized in a mixture of independent e-learning coursework and face-to-face training events over a 14-month period. The syllabus involved six modules: Discovery of Medicines & Planning of Medicine Development, Non-Clinical Testing and Pharmaceutical Development, Exploratory and Confirmatory Clinical Development, Clinical Trials, Regulatory Affairs, Medicinal Product Safety, Pharmacovigilance and Pharmaco-epidemiology, HTA principles and practices. Additionally, in 2016, I have attended the 1st EULAR course on Health Economics in Rheumatology.

In the meantime, my background as an anthropologist led me to become interested in Medical Anthropology. Between 2007–2015 my academic research (MA Phil. and PhD) was oriented towards a specialization in Anthropology of Health, with a special interest in Anthropology of Pharmaceuticals.

Based on my personal experience, on this lecture I will focus on the challenges of the role of the PRP trying to fill the gap between the mindsets and practices of different stakeholders.

Navigating through different meanings of symptoms and treatments, the educated patient representative must act like a translator, decodifying the biologic impact of the disease and intervention over the experience of illness on the everyday aspects of living with a RMD. The biggest expectation and challenge might be to bring these aspects forward, as relevant for the other stakeholders, since they shape individual values and patient's preferences.

Although recognised as having a pivotal role, patient's involvement in research may be limited by tokenism or by ineffective patients' participation. Patients' involvement is now a requirement and an added value to any project. But, is the project team ready and willing to listen to patients? Are PRP duly involved in the project, or are they just expected to be recipients without any input of their perspectives into the development and implementation of the research?

The knowledge and education acquired to perform our task enables us to understand science enough to communicate the patient experience in a meaningful way, improving the research. Our added value is, undoubtedly, our experience with the disease, our understanding of the individual values and preferences shaped by the everyday aspects of living with a RMD. We should be taken more seriously, for the benefit of science and patients.

Disclosure of Interest: None declared

DOI: 10.1136/annrheumdis-2017-eular.7243

SP0052 BUILDING PATIENT PARTNERSHIP IN HEALTH CARE SERVICE DESIGN AND DELIVERY

H. Lempp. *Rheumatology, Kings College London, London, United Kingdom*

This paper will present details of the approach to patient and public involvement in health service delivery, health service research and health care education in England. The presentation will be based upon a Logical Framework with the following key elements: inputs, processes, outputs, and outcomes/impacts. Key barrier acting to minimise the impact of building patient partnerships will be discussed, illustrated by examples from our experience in the preparatory stages for our departmental strategy to formalise close Patient Partnership for our research portfolio: (i) establish honorary contracts for patients for the academic Institution and local Hospital Trust; (ii) include patients on the interview panels to appoint project researchers (iii) build in a separate funding within the overall project budget for the costs associated with patient partnership and (iv) manage patient expectations of rapid implementation of the results of research after the completion of projects. Patient Partnership is essential and feasible to make health care service and design relevant.

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Disclosure of Interest: None declared

DOI: 10.1136/annrheumdis-2017-eular.7123

THURSDAY, 15 JUNE 2017

WIN & HOT session

SP0053 HOT SESSION: VASCULITIS TREATMENT

R.A. Luqmani. *University of Oxford, Oxford, United Kingdom*

The systemic vasculitides are characterized by inflammation of blood vessels, which if untreated, results in tissue or end organ damage or death. Chapel Hill nomenclature is widely used to define different forms of vasculitis according to the size of the predominantly affected vessels. Other forms of vasculitis are not defined by a predominant vessel size (e.g. Behcet's syndrome). Vasculitis associated with the presence of anti-neutrophil cytoplasm antibody (ANCA), termed AAV, is less common than giant cell arteritis (GCA), but considerable advances have been made in understanding the pathogenesis and evidence based treatment for AAV. AAV is divided into three major forms: granulomatosis with polyangiitis (GPA) (Wegener's granulomatosis), microscopic polyangiitis (MPA) and eosinophilic granulomatosis with polyangiitis (EGPA) (Churg–Strauss syndrome). ANCA are implicated in their pathogenesis but not all patients with AAV are ANCA positive.

We will review recent EULAR guidelines on therapy for AAV, based on careful structured clinical evaluation of patients, with stratification according to severity. Cyclophosphamide or rituximab (plus glucocorticoid) is used for severe disease, followed by maintenance with azathioprine (AZA) or methotrexate (MTX), and reducing doses of glucocorticoids; or maintenance rituximab. Additional plasmapheresis is indicated for very severe disease; by contrast for less severe disease, MTX or AZA or mycophenolate (plus glucocorticoids) can be used. The evidence for effectiveness is clear for MPA and GPA. A number of studies are underway to improve our use of these existing agents and to test newer, mechanism based treatments such as inhibition CTLA4Ig or of the C5 complement pathway in GPA and MPA. For EGPA with severe manifestations, cyclophosphamide and glucocorticoids are recommended. A trial of mepolizumab (inhibitor of interleukin 5, a potent driver of eosinophil production) in EGPA has recently been completed. IL-6 inhibition with tocilizumab is a significant advance over glucocorticoid monotherapy in treatment of GCA. Apremilast is effective in treating mucocutaneous manifestations of Behcet's syndrome. Relapse is a common feature of many forms of vasculitis and needs to be monitored by structured clinical evaluation. Monitoring of ANCA titres in AAV or the acute phase response in most forms of vasculitis can be misleading and should not serve as sole guide to therapy in the absence of clinical evidence of active disease. Early survival is over 94% of patients with most forms of vasculitis. Five year survival is 70–75% for AAV with current therapy. However, if the condition is more severe disease, especially with significant renal impairment, mortality is worse.

Vasculitis remains a challenge. Whilst mortality has dramatically reduced as a result of effective immunosuppression, relapse and chronic damage are significant problems for all forms of vasculitis. We need a better understanding of how to manage and limit the long term chronic effects of vasculitis and its therapy.

Disclosure of Interest: R. Luqmani Grant/research support from: Arthritis Research UK, GSK, MRC, UCSF/OIF, Canadian Institutes of Health Research, The Vasculitis Foundation; Consultant for: GSK, Medpace, MedImmune, Roche

DOI: 10.1136/annrheumdis-2017-eular.7162

THURSDAY, 15 JUNE 2017

Treat-to-target in axSpA: reality or utopy?

SP0054 THE CONCEPT OF TREAT-TO-TARGET

J. Braun. *Rheumazentrum Ruhrgebiet, 44649 Herne, Germany*

Many illnesses including most rheumatic diseases have substantial effects on well-being and quality of life, including deterioration of physical and mental function and a reduced life expectancy, since they can cause damage to organs and cells. If healing and regeneration cannot be achieved an impairment of organ function can be expected. In acute diseases this may occur rapidly over hours to days and weeks, while it often takes months to years in chronic diseases. However, if treatment is instituted early enough, organ damage may be prevented or diminished.

Critical for an optimal management of diseases with potentially severe outcomes is to determine the responsible thresholds for, for example, disease activity or to define the maximum level of a surrogate marker at which damage is unlikely to occur and, thus, will not be harmful in the long term. Although the optimal aim of therapy is cure, and appropriate therapy may even normalize life expectancy, many chronic diseases such as hypertension, diabetes, rheumatoid arthritis (RA) and ankylosing spondylitis (AS) have remained without curative therapies in the last decades – even though considerable progress has been made. Thus, a strategic therapeutic approach should aim for prevention of future damage, and maximal improvement of compromised organ function. Therefore, a clearly defined threshold of a validated measure that predicts future harm or no or minimal harm, is a target of critical importance for chronic diseases with potentially severe outcomes. Treat-to-target strategies having been developed to achieve this have widespread implications. They should be routinely followed - as long as the potential harm from treatment is carefully balanced against its benefit. Clearly, if inappropriately managed, the consequences of diabetes and hypertension in

the long run include, myocardial infarction, stroke, renal failure, blindness, etc. Therefore, target values for biological markers have been determined below which organ damage does usually not occur and life expectancy is normalised. Examples for domains in which such thresholds have been defined are blood pressure, glycosylated haemoglobin (HbA1c), and others.
Inflammatory rheumatic diseases lead to organ damage not only in the musculoskeletal system but they may also harm internal organs. A target level of a measure related to its long-term outcome, can be a surrogate measure like the cholesterol level for cardiovascular diseases, or a composite measure of disease activity as used in RA (DAS28) or in AS (ASDAS). The treat-to-target strategy can be reduced to a simple algorithm of, on the one hand measuring activity and on the other hand, in consequence, adapting treatment. Treatment adaptation does not necessarily mean changing a medication or increasing the dosage of a drug but may even also mean life style changes, as long as the therapeutic target is attained or nearly attained - importantly within a prespecified time frame. Therapeutic adaptations should always take patient factors, including comorbidities, adverse events and patient preferences, into account.
However, the musculoskeletal system can mostly not be assessed by using a simple surrogate or direct “gold standard” measures, since rheumatic diseasese with multiple signs and symptoms are mostly rather complex. In RA information derived from physical examination using a quantitative joint count is considered very important. This is different in AS. Additionally, information from the history, which can be collected through patient self-report multifaceted questionnaires, has proven effective in determining patient status and its change. This is even more important in AS. However, functional impairment has reversible and irreversible components. Damage is a consequence of high and/or persisting disease activity. The most important variables contributing to joint damage in RA are swollen joint counts and C-reactive protein (CRP). The latter in combination with questions on back pain is also important in AS, while in RA the use of composite measures of disease activity that comprise joint counts is critical. There is good evidence that, if this strategy is consequently followed, physical function will improve and joint damage be reduced in patients with RA. To determine optimal treatment targets in RA it is necessary to define the thresholds of disease activity measures at which progression of joint destruction is halted. Of note, only remission is associated with maximal reversal of functional impairment and a stop of progression of damage as well as work disability. However, it needs to be realized that some remission criteria are more stringent than others.
Disclosure of Interest: None declared
DOI: 10.1136/annrheumdis-2017-eular.7129

SP0055 WHAT ARE THE CHALLENGES FOR APPLYING TREAT TO TARGET IN AXIAL SPONDYLOARTHRITIS?

M. Dougados. *rheumatology Hopital Cochin, René Descartes University, paris, France*

The concept of Treat to Target (T2T) applied in rheumatoid arthritis has been evaluated in psoriatic arthritis and is currently under investigation in two different strategy trials in patients suffering from axial spondyloarthritis (axSpA). Whatever le results of these trials will be, the acceptation of this concept and consequently its implementation in daily practice might be challenging for several reasons.
The concept of T2T necessitates 3 different steps and also the close collaboration of the patient and the availability of different treatment modalities.
The three different steps consist in:
a) the choice of the most relevant outcome measure (e.g. a measure evaluating a domain recognized as predisposing to subsequent clinically relevant damage (either structural damage or important comorbidities such as cardiovascular diseases)).
b) the determination of the threshold of the outcome measure to reach (threshold below which the risk of subsequent damage is abolished or significantly decreased).
c) The time to reach the target is usually related to the treatment modality (a few days for NSAIDs and several weeks for DMARDs).
A part these different steps, two points have to be considered a) this T2T approach is impossible without embarking the patient in a true share decision b) this T2T strategy requires the possibility to adapt/increase the treatment in case the target is not reached after one or several “conventional” treatment modalities.
For each of these different points we will consider past-on ongoing initiatives proposing to resolve the different encountered issues in order to facilitate the elaboration and the implementation of a T2T strategy in AxSpA.
Disclosure of Interest: M. Dougados Grant/research support from: UCB,ABBVIE,PFIZER,MERCK, Consultant for: UCB,ABBVIE,PFIZER,MERCK
DOI: 10.1136/annrheumdis-2017-eular.7119

SP0056 UPDATE OF THE T2T RECOMMENDATIONS IN SPA

D. Van Der Heijde on behalf of T2T in SpA working group. *Rheumatology, Leiden University Medical Center, Leiden, Netherlands*

In 2013 the first recommendations for treating spondyloarthritis to target (T2T) were published. These followed the reasoning for the T2T recommendations for rheumatoid arthritis. Although the systematic literature review at that time did not provide evidence to support the recommendations, five overarching principles and 11 recommendations were formulated. There were 9 commen recommendations for axial SpA, peripheral SpA and psoriatic arthritis and 2 additional recommendations for each subgroup specifically. In 2017 the T2T working group met again to update the recommendations. This was based on an updated systematic literature review. Data had been published that there is indeed a clear link between inflammation and subsequent longterm outcomes, which is the basis for the T2T principles.
SpA is characterised by musculoskeletal signs and symptoms (arthritis, enthesitis, dactylitis, axial disease) but also extra-articular manifestations (psoriasis, inflammatory bowel disease, anterior uveitis) are important manifestations. Moreover, comborbidities (such as osteoporosis, cardiovascular disease). All these manifestations are taken into account in the formulation of the recommendations. The overarching principles were kept largely identical. Some changes in the wording were made for a better understanding, but no fundamental changes were made. A total of 11 recommendations were formulated. These are now for all subgroups of SpA and no specific recommendations are proposed. In principle, the treatment target is remission or inactive disease of musculoskeletal and extra-articulaur manifestations, and the target should be individualised. It is important that remission/inactive disease should be based on a combination of clinical and laboratory parameters, and disease activity should be measured on the basis of clinical signs and symptoms as well as acute phase reactants. This is important to realise, e.g. in axial SpA as patient reported outcomes only are at best weakly correlated with structural damage. In certain circumstances, low disease activity may be an alternative target. Because of the heterogeneous presentation of SpA, not only the target, but also the assessments should be individualised. Both in the overarching principles and in the recommendation the shared decision between patient and rheumatologist is listed as the basis of the T2T management.
The updated recommendations will be presented.
Disclosure of Interest: D. Van Der Heijde Consultant for: AbbVie, Amgen, Astellas, AstraZeneca, BMS, Boehringer Ingelheim, Celgene, Daiichi, Eli-Lilly, Galapagos, Gilead, Janssen, Merck, Novartis, Pfizer, Regeneron, Roche, Sanofi, UCB, Employee of: Imaging Rheumatology
DOI: 10.1136/annrheumdis-2017-eular.7158

THURSDAY, 15 JUNE 2017

Calcium crystal deposition in rheumatic diseases —

SP0057 CALCIUM CRYSTALS AND THEIR LINK TO OSTEOARTHRITIS

J. Bertrand. *Department of Orthopeadic Surgery, Otto-von-Guericke University Magdeburg, Magdeburg, Germany*

Calcification of cartilage is a common finding during osteoarthritis (OA). We have shown that it is mainly of the BCP type and not the CPPD type of crystal formation. BCP cartilage calcification is directly linked to the severity of cartilage degradation and, therefore, OA severity. We have also shown that with increasing hypertrophic differentiation of chondrocytes, the amount of calcification increases in vivo and in vitro. This indicates a link between chondrocyte hypertrophy andcartilage calcification. The pyrophosphate pathway is known to be involved in tissue calcification. It functions to keep the sensitive balance of pyrophosphate (PPi) and phosphate (Pi), thereby preventing the generation of calcium-phosphate crystals. One key player in this pathway is the nucleotide pyrophosphatase phosphodiesterase (NPP1), which has been demonstrated to be regulated by inflammatory mediators such as IL-1. In our cohort of OA patients, the expression of collagen X and NPP1, but not ANK and TNAP, correlated with cartilage calcification and also with the Mankin-Score. NPP1 expression inverse correlated with the calcification, whereas collagen X was upregulated. This finding was confirmed in experimental murine OA using the DMM mouse model. Furthermore, NPP1mut/mut mice (ttw/ttw) exhibit more calcification activity than wild type controls in joints as well as in cartilage of non weight bearing areas, including ear cartilage, suggesting that mechanical stress is not required for the induction of calcification. NPP1mut/mut (ttw/ttw) mice developed typical OA-like changes as evaluated by histological analysis as well as in vivo imaging and histological stainings. Intriguingly, calcification was associated with increased expression of the hypertrophic cartilage marker collagen X and the bone marker collagen I. Additionally, BCP crystals are able to activate chondrocyte differentiation via the WNT signaling pathway.
NPP1 is an important player in OA-associated cartilage calcification. Pathologic calcification of cartilage resembles in many aspects cartilage transformation into bone. Taken together, the data suggest that OA is characterized by the re-activation of molecular signalling cascades that at least in part resemble endochondral ossification.
Disclosure of Interest: None declared
DOI: 10.1136/annrheumdis-2017-eular.7192

SP0058 REVIEW OF THE DIFFERENT IMAGING MODALITIES TO DETECT CALCIUM DEPOSITION DISEASES

P. Omoumi. *Lausanne University Hospital, Lausanne, Switzerland*

Crystal deposits in and around the joints are common and most often encountered