as incidental imaging findings in asymptomatic patients.
However, they can also cause chronic or acute arthropathy, generating symptoms. In the chronic setting, imaging features are usually characteristic and allow the differentiation of the type of crystal arthropathy. In the acute phase and in the early stages of the crystal deposition, the signs are often non-specific, and the final diagnosis still relies on the analysis of synovial fluid.
Radiography is the main imaging modality for the workup of these conditions. It can confirm the diagnosis and often characterizes the type of crystal arthropathy. In recent years, US has played an increasingly important role in this setting, and is a useful tool in superficially located crystal-induced arthropathies. CT nicely complements radiography for deeper sites, especially the axial skeleton. DECT is a promising tool for the characterization of crystal arthropathies, in particular gout as it permits a quantitative assessment of deposits, and may help in the follow up of patients.
When performed in the acute stage, MRI may show severe inflammatory changes that could be misleading and correlation to radiographs or CT should help to distinguish crystal arthropathies from infectious or tumoral conditions.
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THURSDAY, 15 JUNE 2017

Fifty shades of remission in RA

SP0059 REMISSION: MORE THAN CLINICAL ...?

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This session is about defining remission. Most examples will be about rheumatoid arthritis (RA) because most experience has been gathered in that disease. But the concept of remission should be viewed from a wider perspective than one disease. The session includes two pro-con debates (on the utility of including imaging and biomarkers in a definition of remission).
As in all things, when embarking on a scientific project, one must ask:
"Why are we doing this?"
To even begin with answering the question, we must first agree on a clear conceptual definition of remission. When we started on the development of the ACR-EULAR definition of remission in RA, we used dictionary sources and discussions to settle on this:
"The state of absence of disease activity in patients with a chronic illness, with the possibility of return of disease activity." [1]
It is clear that choices are made from the beginning, especially with the concept "disease activity", and the possibility that disease activity returns (as opposed to "healing", where this possibility does not exist). Disease activity is a tangible concept adequately defined in RA, but less so in many other rheumatological diseases. Also, disease activity is conceptually separate from (mostly irreversible) consequences of the disease, such as damage. Finally, note that the above concept does not contain the elements "duration" or "treatment".
If we continue with the above concept, why do we want to proceed to operationalize the remission definition? The two main reasons are research and patient care. For both, it is clear that we are defining a very good, perhaps even the best state a patient can be in, given that we are talking about chronic disease, i.e. the root cause of the disease cannot be taken away to heal the patient. Being in such a good state has immediate benefits (minimal disease impact) and probably also future benefits, if lack of disease activity translates to less consequences (damage etc). In both research and patient care, we want a definition that is both valid (favorable test characteristics; links to prognosis) and feasible (time, costs, interpretability). Validity and feasibility oppose each other to a certain extent (eg, definitions with better sensitivity and specificity are usually more expensive). Research and patient care differ in their use of the definition. In research, validity and feasibility can be lower than in patient care, because research is about groups, and cost and interpretability are less of an issue than in patient care.
Most of the people criticizing the current ACR-EULAR remission definition of RA are confused over its purpose: whereas it was intended for use in trials, they criticize it for lack of validity in the clinic. For instance, it is felt that the patient global criterion is too strict, so that patients with no apparent inflammatory activity but a patient global score of 2 or higher (scale 0–10) are "unjustly" not classified as in remission. Also, the lack of a duration or treatment criterion is felt to be a problem, but this is not an issue in research.
In the following pro-con debates, please consider the following:
Proposals to change existing criteria for remission must also be held to the question: "Why are we doing this?"
References:
[1] Remission. (Accessed 21–02–2017, at http://en.wikipedia.org/wiki/Remission.)
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SP0060 BIOMARKERS ARE REQUIRED FOR REMISSION: PRO

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1. Conceptually, remission is thought of as a state where the disease is absent. As we approach a better understanding of the underlying pathophysiological process of a disease, it becomes more and more relevant to include in a definition of remission appropriate biochemical markers of that process.
2. From a practical point of view, definitions of remission in RA have been built upon clinical parameters of disease activity, supplemented in some cases with a single biomarker. However, it is clear that in practice these definitions are insufficiently precise: held against a gold standard of expert opinion, they perform at around 80–90%, misidentifying one or two out of every ten patients. And while there is an understandable and in many ways desiriable development of more patient-reported emphasis in outcomes, it has considerable practical value to be able to objectify an important disease state such as remission.
3. There is convincing evidence to show that biomarkers can be employed succesfully to predict some aspects of RA. In the day-to-day care of patients with this disease, the most important prediction may be whether the effective drug can be tapered or not. Current evidence indicates that biomarkers may be invaluable at helping clinicians and their patients make this important decision.
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Innate immunity

SP0061 A DAY IN THE NEUTROPHIL'S LIFE

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Immunity is essential for life, yet the strength of immune responses are not constant throughout the day. This oscillatory immunity reflects an adaptation of organisms to environmental changes that occur through day-night cycles, so as to optimize and concentrate effective responses to the times of maximal environmental threat. In my talk I will discuss our ongoing efforts to uncover the mechanisms by which neutrophils, the most abundant and aggressive of all immune cells, orchestrate temporal immunity. These mechanisms are reflected in diurnal changes in phenotype and function of neutrophils, which we refer to as neutrophil aging. We propose that the existence of a timed response of neutrophils governed by cell-intrinsic and –extrinsic mechanisms suggests that inflammatory disease co-opts ancestral processes to damage tissues.
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SP0062 DIFFERENTIAL SCAVENGING OF APOPTOTIC CELLS AND BACTERIA

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During inflammation and infection, we are simultaneously confronted with both self and non-self in form of dying cells and microbes, respectively. Mechanisms that facilitate the non-immunogenic clearance of self-antigens derived from apoptotic and necrotic cells and that, in parallel, allow the initiation of an immune response against invading pathogens are incompletely understood. Recent data from our laboratory show that the immune system actively sorts apoptotic cells (ACs) and bacteria into distinct subspecies of phagocytes thereby enabling a segregated processing of self and non-self as well as a differential immune response against these two entities. During inflammation, ACs were cleared by tissue resident macrophages (resMφ) that performed a non-immunogenic disposal of self antigens, whereas bacteria were preferentially ingested by monocyte-derived inflammatory macrophages. We identified the enzyme 12/15-lipoxygenase and the nuclear receptor Nr4a1, both specifically expressed by resMφ, as key factors that control the coordinated and non-immunogenic phagocytosis of ACs by these specialized macrophage subset. Incorrect sorting and aberrant uptake of AC-derived self-antigens by pro-inflammatory and immunocompetent phagocytes, however, resulted in the break of self-tolerance and autoimmunity. Our data thus demonstrate the importance of a sorted clearance of ACs for the maintenance of immunologic self-tolerance.
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SP0063 THE ROLE OF MUSCLE IN INNATE IMMUNE RESPONSES

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The skeletal muscle represents a unique site from the immunological point of view. Leukocytes are virtually absent in healthy conditions. However they are quickly recruited upon muscle injury, persist during the regenerative phases to disappear again after tissue healing. Thus, it represents and ideal scenario to study the