SP0159 EULAR RECOMMENDATIONS FOR THE MANAGEMENT OF SJÖGREN'S SYNDROME

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Sjögren's syndrome (SjS) is a systemic autoimmune disease that affects 0.1% of the European population and that presents with a wide spectrum of clinical manifestations. Primary SjS has no cure, and their therapeutic management has not changed significantly over the past decades. The current approach is still based upon symptomatic treatment of sicca symptomatology and broad-spectrum immunosuppression directed against systemic disease. In spite of the several therapeutic options available, there is insufficient information on their differential efficacy and safety, making that treatment decisions remain challenging in clinical practice. Over the last decade, research has centred on investigating more effective SS-targeted therapies, with the development of various randomised controlled trials (RCT) unfortunately with the absence of game-changing results. With these discouraging results, national effort promoted by some scientific societies have led to the development of several sets of recommendations.

The European League Against Rheumatism (EULAR) promoted and supported in 2010 an international collaborative study (EULAR SS Task Force) aimed at developing consensual disease activity indexes in SS. This work was very successful with the development of the first international specific scores for SjS (ESSPRI and ESSDAI), now widely used both in clinical and research settings. The wide consensus obtained on the development of the ESSDAI/ESSPRI scores provided an excellent opportunity to advance in the development of international recommendations for the therapeutic management of SS. Therefore, we continued the work of the Task Force with the development of evidence-, consensus-based official EULAR recommendations following the Delphi methodology, with the aim to provide physicians and patients with a rational therapeutic approach to SjS patients.

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SP0160 2018 EULAR RECOMMENDATIONS FOR THE USE OF GLUCOCORTICOID THERAPY

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Glucocorticoids (GC) are potent anti-inflammatory and immunosuppressive drugs which are used successfully to treat many disorders, including rheumatoid arthritis, polymyalgia rheumatica, giant cell arteritis, myositis, systemic lupus erythematosus and other rheumatic diseases. However, these drugs also have the potential to cause severe adverse effects, particularly if high doses are used for prolonged periods. Therefore, the benefits of GC therapy must be balanced against the potential risks. Key strategies to achieve this goal include (i) following guideline recommendations regarding GC therapy dosing, monitoring for potential adverse events, and adverse event prevention and management, (ii) using or developing new therapeutic advances to improve the therapeutic balance.

The EULAR Glucocorticoid Task Force has already published several recommendations over the last years such as those on the standardised nomenclature for GC dosages and treatment regimens, on the management of systemic GC therapy in rheumatic diseases, and on monitoring adverse events of low-dose GC therapy. Recent work of this group dealt with the question under which conditions long-term treatment with GC has an acceptably low level of harm. As a result, the task force members agreed that the risk of harm is low for the majority of patients at long-term dosages of ≤5 mg prednisone equivalent per day, whereas at dosages of >10 mg/day the risk of harm is elevated. At dosages between >5 and ≤10 mg/day, patient-specific characteristics determine the risk of harm. This means general and glucocorticoid-associated risk factors and protective factors such as a healthy lifestyle should be taken into account when evaluating the actual and future risk.

Another new information on the use of GCs has been recently provided by EULAR 2016 update of RA recommendations: "Short-term GCs should be considered when initiating or changing csDMARDs, in different dose regimens and routes of administration, but should be tapered as rapidly as clinically feasible." This wording does acknowledge that there are several different regimens for oral use, intramuscular injection, and intravenous pulse therapy. It is now also being stated more clearly that GCs should be given as bridging therapy together with csDMARDs, either as part of the initial strategy or subsequently if this has failed.

In contrast, GCs are usually not needed as a bridging therapy when bDMARDs or tsDMARDs are used. This recommendations reconfirms that GCs should be gradually reduced and ultimately stopped, ideally within 3 to 6 months.

Finally, the role of GCs in the management of polymyalgia rheumatica as has been described and discussed by the recent EULAR/ACR recommendations will be briefly discussed.

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SP0161 2018 EULAR RECOMMENDATIONS FOR THE MANAGEMENT OF HAND OSTEOARTHRITIS

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Hand osteoarthritis is a prevalent osteoarthritis phenotype, with high disease burden. With regard to the management of patients with hand osteoarthritis European League Against Rheumatism (EULAR) recommendations have been formulated in 2007. However, new evidence has emerged and therefore the recommendations have been updated by an international task force of experts, including rheumatologists, health professionals, plastic surgeon and patient representatives.

First, a systematic literature review was performed, collecting evidence regarding non-pharmacological, pharmacological and surgical treatment options. Second, based on the evidence and expert opinion, five overarching principles were formulated, referring to treatment goal, information provision, individualised treatment, shared-decision making and need to consider multidisciplinary and multimodal treatment approaches. In addition ten recommendations were formulated, describing non-pharmacological, pharmacological and surgical options, and considerations about follow-up. Finally, a level of evidence, grade of recommendation and level of agreement were allocated by all experts to each statement.

With the updated EULAR recommendations evidence-based up-to-date guidance can be provided on the management of hand OA.

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Fractures: more than bone alone
SP0162 LOOK AT THE CASCADE : CLINICAL OBSERVATIONS

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Vertebral fracture (VF) is the most common osteoporotic fracture, and a strong risk factor of subsequent vertebral fracture. Prospective studies have shown that a recent VF increases an imminent risk of a subsequent one, and attention has been paid recently to a possible cascade phenomenon i.e. the occurrence of multiples VFs in less than one year after discontinuation of denosumab (rebound vertebral fractures) in postmenopausal osteoporosis and in patients receiving aromatase inhibitors. Vertebral fracture cascade has been reported in secondary causes of osteoporosis (after the initiation of systemic glucocorticoids, in endocrine disease, benign hemopathies as mastocytosis and in pregnancy-and lactation-associated osteoporosis). We reported the clinical observations of patients with multiples vertebral fractures occurring over one year and we discussed the causes, the potential risk factors and the appropriate management.

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SP0163 A CASE OF SEVERE OSTEOPOROSIS AND ISCHAEMIC HEART DISEASE; WHAT WILL BE THE FUTURE CARDIOVASCULAR RISK?

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Case description: A 63-year-old female recently presented to metabolic bone clinic with vertebral fractures at L4 and L5. DEXA scan showed osteoporosis with T-scores at the spine -4.0 and left hip -3.8. Patient was commenced on weekly

Alendronic acid 70 mg, calcium and vitamin D supplements. Patient had severe vitamin D deficiency and was commenced on treatment at primary care. She is on analgesics and physiotherapy for back pain, which was of moderate severity. She was on oral prednisolone for 5 years for polymyositis rheumatica in the past and weaned off year ago. Other past medical history includes coronary artery disease; a myocardial infarction requiring coronary artery bypass graft 3 years back. She does not have diabetes or hypertension. She has hypercholesterolaemia; probably polygenic in origin and is on lipid lowering medications since the age of 50 years.

Her alcohol intake is minimal, and is a non-smoker. There is no family history of osteoporosis or cardiovascular disease. Her milk consumption accounted for 300 mg of calcium per day. She has reached menopause at 43 years of age and has not been on hormonal replacement therapy. Her current medications are Aspirin, Atorvastatin, Bisoprolol and Omeprazole. There is no history of indigestion or dental concerns.

Except mild local tenderness at L4–5 region, rest of the clinical examination is unremarkable.

Recent blood investigations showed normal calcium, inorganic phosphate, PTH and vitamin D. Alkaline phosphatase (195 IU/L) and P1NP (55 ug/L) were raised. Renal functions showed normal creatinine with eGFR of 62 mL/min. Full blood count is unremarkable. Serum and urine electrophoresis excluded multiple myeloma.

Summary: This is a case of severe osteoporosis involving vertebral fractures requiring osteoporosis treatment. Possible causes for osteoporosis would be previous use of oral steroids for long time and severe undiagnosed vitamin D deficiency. Hypercholesterolaemia and significant history of ischaemic heart disease are other concomitant diagnoses.

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Emerging topics in the management of the antiphospholipid syndrome

SP0164 HOW TO MANAGE ASYMPTOMATIC CARRIERS OF ANTIPHOSPHOLIPID ANTIBODIES

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Individuals who do not display the classical features of the Antiphospholipid Syndrome (APS) (vascular and obstetric disease)¹ are referred to as “aPL carriers”. They can be patients affected by systemic autoimmune diseases who are screened for antiphospholipid antibodies (aPL). aPL may be found in patients with “non-criteria” manifestations or in women undergoing investigations for infertility. The presence of aPL can be serendipitously discovered before a surgical procedure because of a prolonged thromboplastin time. Are these subjects at increased risk for thrombosis and adverse pregnancy outcomes (APO)? Since aPL are pathogenic autoantibodies, the answer should be “yes”. However, the magnitude of the risk can be variable from patient to patient, accordingly to the multifactorial origin of aPL-related vascular and obstetric manifestations.

According to international consensus,² the thrombosis risk stratification should consider: 1) the aPL profile (type, titer, persistence), 2) the coexistence of other thrombotic risk factors, and 3) the presence of an underlying autoimmune disease, mainly systemic lupus erythematosus (SLE). The definition of “high-risk” aPL profile comprises positivity for Lupus Anticoagulant (LA), or ‘triple positivity’, i.e. LA+anti cardiolipin antibodies (aCL) +anti-beta2glycoprotein I antibodies (anti-B2GPI) or medium-high titers of IgG aCL or IgG anti-B2GPI. Conversely, patients with isolated, intermittently positive aCL or anti-B2GPI at low-medium titers could be considered at low risk for thrombosis.

According to the literature, aPL carriers seem to have a low annual incidence of acute thrombosis, ranging from 0% to 3.8%³. These figures are not much different from the estimated incidence of thrombosis in unselected cases (about 1% patient-years), which is also equivalent to that of major bleeding associated with the use of low dose aspirin (LDA), the most frequently used drug for primary prophylaxis.⁴ Therefore, the dilemma in clinical practice is to correctly select those aPL carriers for whom the expected benefit of therapy outweigh the risk.

Over years, the management of aPL carriers have been investigated in several studies enrolling different patients groups (SLE, pure obstetric APS, asymptomatic aPL carriers) and evaluating the efficacy of various interventions: LDA,⁵ low intensity warfarin,⁷ low molecular weight heparin (LMWH) in high risk situations such as surgery, prolonged immobilisation, and puerperium.⁹

Aside from drugs acting on platelets and on the coagulation system, there is evidence that immunomodulatory agents may be beneficial in primary prophylaxis of aPL carriers. Hydroxychloroquine (HCQ) is a well-recognised key-drug in the management of SLE patients and has an anti-thrombotic effect.¹⁰ The use of HCQ as primary prophylaxis has been proposed also for non-SLE patients.¹¹ Statins may be useful in aPL carriers not only for the correction of a proatherogenic lipid profile, but also for reducing proinflammatory and prothrombotic biomarkers.^{12,13}

Turning to the obstetric field, the detection of aPL antibodies has been increasingly performed in asymptomatic women, mainly for obstetrical reasons (e.g. before assisted reproductive techniques, after APO that are not included in APS classification criteria). Therefore, it is not infrequent to take the responsibility to recommend or not a treatment in “healthy” pregnant women carrying aPL. General obstetric risk should be assessed (age, hypertension, obesity, etc.). It is currently under discussion whether different aPL profiles confer the same degree of obstetric risk.¹⁴ LA¹⁵ and triple aPL positivity¹⁶ seem to be the major predictors of APO, although APO have been described also in patients with a “low-risk” aPL profile (e.g. IgM isotype or medium to low aPL titers).^{17,18} A key drug for primary obstetric prophylaxis is LDA and many physicians prescribe it to pregnant aPL carriers.²⁰ The immunomodulatory properties of HCQ have been advocated to be beneficial in pregnant patients with aPL²¹ and clinical retrospective studies supported its effectiveness in refractory obstetric APS.^{22,23} Puerperium is considered a high-risk period for thrombosis for all women. Women who carry aPL should be considered for LMWH for 4–6 weeks after delivery.¹⁹

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SP0165 THE COMPLEX INTERPLAY BETWEEN SYSTEMIC LUPUS ERYTHEMATOSUS AND ANTIPHOSPHOLIPID SYNDROME

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The antiphospholipid syndrome (APS) was first described in the 1980's. It is diagnosed when antiphospholipid antibodies (aPL) i.e. anti-cardiolipin (aCL), anti-β₂Glycoprotein-I (aβ₂GPI) or positivity in the functional lupus anticoagulant test (LA) occur together with any type of thrombosis (e.g. myocardial infarction (MI), stroke, venous or microvascular thromboses) or obstetric complications.⁵ aPL recognise protein co-factors, most importantly the scavenger protein β₂Glycoprotein-I (β₂GPI), that bind to membrane phospholipids. It is not fully understood how complexes of β₂GPI and anti-β₂GPI antibodies initiate a pro-thrombotic state, but activation of platelets, endothelial cells and the complement cascade are associated features.

Approximately 80% of APS patients are women, many are young and severely ill. There is a considerable overlap between APS and SLE. Approximately 30%–40% of SLE patients are aPL positive but only about half of them develop clinical symptoms fulfilling the APS classification criteria.