Learning Objectives
- Explain that shorter time to remission of LN protects against rapid progression to ESRD
- Explain that preventing flares in LN protects against rapid progression to ESRD
- Explain that longer duration of immunosuppressive therapy in LN protects against rapid progression to ESRD
- Describe the unusual features of LN that might lead to catastrophic progression to ESRD

INCREASED CV RISK IN WOMEN WITH SLE: STORIES FROM A PANEL OF BIOMARKERS

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Cardiovascular (CV) mortality and morbidity are significant challenges in managing patients with systemic lupus erythematosus (SLE). Patients with SLE have a 2-fold higher risk of stroke and a 3-fold higher risk of myocardial infarction than in the general population. Still, the risk is much greater in young women with SLE compared to age-matched women without SLE.1

The higher risk of CV disease in SLE patients is mainly related to accelerated atherosclerosis, due to sub-clinical disease and clinical manifestations earlier than the general population. A complex interplay between traditional risk factors and SLE-related features is responsible for accelerated atherosclerosis, even though its pathogenesis is not fully understood.2 SLE-related factors contributing to accelerated atherosclerosis include cytokines (e.g., IFN-α, BAFF) and auto-antibodies production responsible for endothelial cells dysfunction, hyperactivated T-cells directed against peptides from vascular cells, impairment in lipid profile with increased oxidized LDL and pro-inflammatory (pi)HDL, high dose or high duration of glucocorticoids therapy. Therefore, although there is an increased prevalence of Framingham traditional CV risk factors (e.g., age, hypertension, dyslipidemia, diabetes, current smoking), it is not surprising that they do not fully account for the high CV risk observed in SLE patients.3

New biomarkers have been developed to identify potentially high CV risk SLE patients, including increased circulating leptin, antibodies against apolipoprotein A1, serum cardiac troponin T, and the soluble tumor necrosis factor-like weak inducer of apoptosis (sTWEAK). In addition, a combination of biomarkers factors (age, piHDL, homocysteine, sTWEAK) has shown better sensitivity (89%) and specificity (79%) than any single biomarker to predict the formation or enlargement of carotid plaques.4 Nevertheless, a biomarker or combination of markers to predict cardiovascular risk accurately in SLE patients is still missing.

Presently, the main objectives to lower the CV risk in SLE should be to monitor and correct traditional CV risk factors while treating the disease to target and maintain remission or low disease activity, prescribing hydroxychloroquine to virtually all patients and minimising corticosteroid use as much as possible.

REFERENCES

Learning Objectives
- Describe the morbidity associated with cardiovascular diseases in SLE
- Explain the interplay between traditional risk factors and SLE related features responsible for accelerated atherosclerosis
- Discuss the most promising biomarkers of CV risk in SLE patients

Plenary III: difficult-to-treat lupus

APS ANTIBODIES: MANAGING PATIENTS WHEN LABORATORY SCENARIOS DON’T FIT GUIDELINES

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Some patients with positive antiphospholipid antibodies (aPL) have not been included in randomised clinical trials or observational registries and, therefore, information on their risk of thrombotic or obstetric recurrence and optimal treatment is scarce.

In this session, the existing evidence regarding the management of two laboratory scenarios not covered by the guidelines is presented: (1) patients with antiphospholipid syndrome (APS) clinical manifestations and anti-phospholipid (aPL) positivity not fulfilling APS laboratory criteria, and (2) the possibility of discontinuing anticoagulation in APS patients whose aPLs become persistently negative.

Growing evidence suggests a role for low titres and ‘non-criteria’ aPL, especially in obstetric APS. Treatment is not formally recommended but might be considered according to the individual’s risk profile. Regarding the question of whether or not to discontinue anticoagulants after the ‘spontaneous’ disappearance of aPL, there is no definite answer. Retrospective studies seem to suggest that withdrawal of anticoagulation could be safe in certain patients with APS, especially in those with a first provoked venous thrombosis and whose aPL became persistently negative during follow-up.1,2 Still, before the withdrawal can be recommended in routine clinical practice, multicentre and prospective studies are required to validate this hypothesis.

REFERENCES

Learning Objectives
- Explain the main challenges in managing patients with antiphospholipid antibodies when laboratory scenarios don’t fit guidelines