One week later, she suddenly deteriorated with acute severe respiratory distress, severe hypoxemia, unstable shock, and hemoptysis. Chest X-ray showed bilateral lung infiltrate suggestive of diffuse alveolar hemorrhage. She was transferred to paediatric intensive care and mechanical ventilation, including high-frequency ventilation, was required. Flexible bronchoscopy confirmed diffuse alveolar hemorrhage. She was treated with pulses of methylprednisolone, intravenous cyclophosphamide, and plasmapheresis.

Learning Objectives
- Explain pulmonary manifestations in lupus
- Discuss therapeutic approach to diffuse alveolar hemorrhage
- Describe prognosis of this life-threatening complication of SLE

14 MANAGEMENT OF CARDIOVASCULAR INVOLVEMENT IN SLE

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10.1136/lupus-2023-la.14

Case 1: A 35-year-old woman of Hispanic ancestry

A 35-year-old woman of Hispanic ancestry received a diagnosis of systemic lupus erythematosus (SLE) in June 2021, based on polyarthralgia/itis, malar rash, proteinuria of 1400 mg/24 h, positive antinuclear antibodies, anti-double-stranded DNA antibodies (anti-dsDNA), with hypocomplementemia. Lupus anticoagulant, anticardiolipin and anti-β_2-Glycoprotein-I antibodies were negative. Her SLEDAI score was 16. A kidney biopsy was performed showing a focal proliferative glomerulonephritis (Class III), with a score of 12 and 0 of activity and chronicity, respectively. She was treated with hydroxychloroquine 400 mg/day, prednisone 20 mg/day, and mycophenolate mofetil 3000 mg/day as induction therapy.

In September 2021 she came to the emergency room due to persistent tachycardia, dyspnea on moderate exertion, and chest pain. At admission she presented elevated ESR and C-reactive protein level, normal kidney function tests, proteinuria of 350 mg/24 h, and positive anti-dsDNA, with low C3 and C4. During hospitalisation she presented fever, and worsening dyspnea, for which she required oxygen therapy. The electrocardiogram showed sinus tachycardia and the echocardiography a systolic dysfunction and a hypokinetic left ventricle (inferior and lateral walls) with an ejection fraction of 40%. Troponin T and brain natriuretic peptides were elevated. The SARS-CoV2 RT-PCR was positive. With the suspected diagnosis of acute myocarditis in the context of SARS-CoV2 infection, the patient was treated with methylprednisolone pulses, IVIG, and respiratory support.

Learning Objectives
- Describe the different myocardial manifestations in a patient with SLE.
- Discuss complications and differential diagnosis with allied diseases.
- Discuss the treatment of myocarditis in a patient with SLE.

15 MANAGEMENT OF CARDIOVASCULAR INVOLVEMENT IN SLE

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Case 1: A 40-year-old woman with myocardial infarction

A 40-year-old female was diagnosed with systemic lupus erythematosus (SLE) at the age of 30 based on malar rash, arthritis, positive antinuclear antibodies, anti-double-stranded DNA antibodies, hypocomplementaemia, and biopsy-proven Class IV lupus nephritis. She was treated with glucocorticoids (GC), hydroxychloroquine (HCQ), and intravenous pulses of cyclophosphamide followed by mycophenolate mofetil (MMF) achieving complete remission 6 months later. Four years later, she suffered from a second SLE flare in the form of Class IV lupus nephritis as well as arthritis, receiving induction treatment with GC and MMF and achieving complete renal response 8 months later. She remained in lupus low disease activity for the next 5 years with prednisone 2.5 mg/day, HCQ 300 mg/day, and MMF 500 mg/12h. She was a current smoker, and her previous history included arterial hypertension and dyslipidaemia treated with enalapril 10 mg/day and atorvastatin 20 mg/day.

At the current admission, she presented at Emergency Department with thoracic pain and shortness of breath. She was diagnosed with myocardial infarction. Coronary angiography showed an atherosclerotic plaque in anterior descending coronary artery that required percutaneous coronary intervention and stenting. The patient was discharged without acute complications under treatment with dual platelet antiaggregation.

What could we have done to avoid this outcome?

Learning Objectives
- Discuss the general management of cardiovascular risk factors in patients with SLE
- Discuss the usefulness of different scoring tools to assess the atherosclerotic cardiovascular disease in SLE patients and the potential utility of imaging
- Discuss the objectives of treatment (primary prevention) of the different cardiovascular risk factors (hypertension, dyslipidaemia, tobacco) in SLE patients and the indications of aspirin in primary prevention

16 MANAGEMENT OF LUPUS NEPHRITIS

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The therapeutic armamentarium of the lupologist is expanding, notably when faced with one of the most frequent severe systemic lupus erythematosus (SLE) organ manifestation: Lupus nephritis (LN). Based on real-practice case studies, we will address the latest guideline recommendations for the management of LN. With novel agents at hand, we will make a journey through several treatment strategies for LN and provide key learnings by interactive discussions.

Case 1: 20-year-old woman with LN

A 20-year-old female with no significant pathological history presented with arthritis, facial rash and leg swelling. She was recently diagnosed SLE and treated prednisolone and hydroxychloroquine, calcium, and active vitamin D. Three months later she developed nephrotic syndrome and mild hypertension, her laboratory results showed a creatinine 123 mmol/L (1.5 mg/dL), positive antinuclear antibodies 1:1280, anti-double-stranded DNA 1230 U/mL and low C3 and C4. Her urine sediment showed glomerular erythrocyturia. A kidney biopsy was performed confirming active LN. After an interactive discussion on histopathological findings, a therapeutic decision must be made.

Learning Objectives

- Describe at least four treatment goals for your LN patient
- Summarize three treatment options for induction of remission in LN
- Discuss three treatment options for preventing relapses in LN
- Explain the pathophysiology of glomerulonephritis in SLE

17 MANAGEMENT OF LUPUS NEPHRITIS

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10.1136/lupus-2023-la.17

Case 1: 52-year-old female with SLE, LN and recurrent flare

A 52-year-old female was diagnosed with systemic lupus erythematosus (SLE) in 1983. She had positive antinuclear antibodies (ANA), anti-double-stranded DNA (anti-dsDNA) >666 IU/mL, C3 0.35 g/L, C4 0.07 g/L, CH50 20 IU/mL AI hemolytic anemia, lymphopenia, arthritis, probable lupus nephritis (LN) due to the presence of microhematuria, non-nephrotic proteinuria with preserved renal function, no renal biopsy was performed. She was treated with steroids, cyclophosphamide 6 x 500 mg, hydroxychloroquine 200 mg/d and subsequent maintenance with azathioprine 100 mg/d, prednisone 7.5 mg/d + calcium and vitamin D prophylaxis. Complete renal response was achieved after 18 months but with persistence of anti-dsDNA >600 and low levels of C3-C4. In 2006, she had clinical remission of SLE. GFR > 90 ml/min, inactive sediment and proteinuria 500 mg/d, persistently high anti-dsDNA titer and low C3-4 levels. The first renal biopsy revealed LN Class IIIC (IA 0/24, IC 2/12) and Grade 1 interstitial fibrosis and tubular atrophy. In 2012, she experienced a new flare. A renal biopsy revealed Class III (AC) (IA 6/24, CI 3/12. Complete renal remission was achieved after 1 year of treatment with steroids plus sodium mycophenolate, but persistent immunological activity. On March 2021 she experienced a new flare, with proteinuria up to 2.5 g/d, microhematuria, AKI 1, arthritis, malar rash and positive AAF. Renal biopsy showed LN Class IV (AC) AI: 10/24 CI: 4/12. Triple therapy with steroids, mycophenolate mofetil and belimumab was started. The response to triple therapy in this grumbling disease is discussed, from a renal and immunological point of view.

Case 2: 22-year-old woman with progressive worsening of proteinuria

A 22-year-old woman, diagnosed with SLE at 18 years, presented with deep vein thrombosis, a study was carried out, revealing positive ANA, anti-dsDNA >666 IU/mL, positive IgG anti-β_2-Glycoprotein-I and lupus anticoagulant, and low C3, C4 and CH50, microhematuria and proteinuria of 2 g/d, with normal renal function. Renal biopsy showed LN Class III, AI 4/24, IC 0/12. Steroids, mycophenolate mofetil and hydroxychloroquine, as well as anticoagulation with Vitamin K antagonists were initiated. Despite treatment, the patient presented progressive worsening of proteinuria up to 6 g/d. A new biopsy was performed 6 months later. The light micrograph showed LN Class IV-G (AC) IA 11/24 and IC 2/12 with immune deposits (IF and EM) compatible with associated membranous nephropathy. A sequential regimen of steroids plus cyclophosphamide 3 g (total dose) was started and later triple therapy with MMF-tacrolimus-steroids was continued due to persistent proteinuria in nephrotic range.

A third renal biopsy was performed for control of proteinuria stagnant in 3.5 g, revealing LN III + V with AI 4/24 and CI 3/12, IFTA ≥15%. Treatment with rituximab 1 g x 2 and nephroprotective treatment with losartan 50 mg/12 h were initiated. Under this treatment, a progressive decrease in anti-dsDNA titers up to 125 IU/mL and partial improvement in C3-C4 levels was confirmed, but there was an increase in proteinuria up to 4.5 g/d. In view of these findings and the high burden of immunosuppression administered, nephroprotective treatment was prioritized by sequentially adding dapagliflozin 10 mg/d and spironolactone 50 mg/d, achieving resolution of the nephrotic syndrome, with stable renal function and residual proteinuria of 1 g/d.

Currently, effective antiproteinuric treatments are available and they act by multiple mechanisms. The importance of establishing an adequate clinical correlation between the trajectory of immunological activity and proteinuria will be discussed in order to obtain better clinical results in LN.

Learning Objectives

- Describe clinical and histological findings that identify challenging cases of proliferative LN
- Explain the current role of therapy with anti-BAFF biologics in LN
- Discuss principles and strategies in the management and prevention of organ damage and preservation of kidney function in the long term

Interactive case study workshops

18 MANAGEMENT OF MUSCULOSKELETAL INVOLVEMENT IN SLE

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Case 1. A young female with SLE and relapsing-remitting arthritis

A 14-year-old Caucasian girl presented to her physician with malar and trunk photosensitivity rashes in Summer 2010. In October 2010, she developed fever, pleurisy, polyarthritis and mild proteinuria (<0.5 g/day). Laboratory tests showed positive antinuclear antibodies (ANA), anti-double-stranded DNA (anti-dsDNA, anti-U1RNP, anti-Sm and decreased C3. A diagnosis of systemic lupus erythematosus (SLE) was made and she was treated with IV pulses of methylprednisolone (MPN), 500 mg x 3, then oral prednisone starting from 25