methylprednisolone (250 mg), AZA shifted to tacrolimus (3 mg/bd) and HCQ started. When discharged, prednisone was reduced to 25 mg/day; LMWH, LDA, HCQ, vitamin D and levothyroxine unchanged. Proteinuria rapidly decreased (1.3 g on 15th May and 0.260 g on 10th July).

On 23rd July she had vaginal delivery at 38 weeks' gestation, her baby was 3.390 kg and 51 cm high; APGAR 10/10. Tacrolimus was suspended on the delivery day and the patient continued with prednisone 5 mg every other day with LMWH during puerperium. A subsequent kidney biopsy confirmed Class V glomerulonephritis.

At last evaluation (October 2019), the patient and the baby were fine, urinalysis did not show proteinuria.

Discussion Points

- Pre-pregnancy counselling for women with a history of LN
- Management of acute flares of LN in pregnancy
- Diagnosing pre-eclampsia in women with active LN
- Discuss the use of immunosuppressive medications in pregnancy

Learning Objectives

- Explain the importance of pre-pregnancy counselling in women with LN
- Discuss the best management of flares of LN in pregnant women
- Discuss how to recognise pre-eclampsia in women with LN
- Distinguish lupus nephritis from APS nephropathy

18 RARE DISEASE MANIFESTATIONS IN SLE

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Case 1: A 20-year-old female with SLE and myocarditis

Zahir Amoura

A 20-year-old female with systemic lupus erythematosus (SLE) was referred for chest pain and skin lesions. The diagnosis of SLE was made 4 years earlier, when she developed arthritis, proteinuria, with positive antinuclear antibodies (1/1280), anti-dsDNA, anti-SS-A, and anti-Sm antibodies. The renal biopsy showed Class I lupus nephritis. The patient was treated with low-dose steroids and hydroxychloroquine. One year later, she had pulmonary embolism and received warfarin because of the presence of persistent lupus anticoagulant. Two months before admission, while the patient was receiving hydroxychloroquine (400 mg/day), hand skin lesions gradually developed.

At admission, the patient complained of chest pain and went to the emergency department. Her temperature was 36.6°C, blood pressure 130/70 mmHg, pulse rate 90 beats per minute, respiration rate 18 breaths/minute, and oxygen saturation 98% while she was breathing ambient air. There was normal breath sounds in the lungs' fields. Laboratory tests showed hemoglobin 10.3 g/dL, lymphocytes 0.9 G/L, normal platelets, international normalised ratio (INR) for prothrombin time 2.3, creatinine 69 µmol/L, elevated ultrasensitive troponin T (248 ng/L), low C3 (0.55 mg/dL), positive anti-dsDNA antibodies (45 U/mL) and proteinuria 1.5 g/24h. Serum protein electrophoresis showed a normal pattern and a diffuse increase

in immune globulin. The tests for lupus anticoagulant, anticardiolipin antibodies, were positive.

Discussion Point

- Diagnosis and management of lupus myocarditis

Learning Objectives

- Describe the clinical presentation of lupus myocarditis
- Discuss the diagnosis workshop for lupus myocarditis
- Discuss treatment options of lupus myocarditis

Case 2: A 22-year-old male with thrombotic microangiopathy

Richard Furie

A 22-year-old male was admitted to the hospital because of headache, confusion and fever. He had been previously well. At the time of admission, he was on no medications. Initial laboratory test results were notable for WBC 2,800, Hb: 6.7; platelets: 64,000 and creatinine: 1.9. The patient was given broad spectrum antibiotics for the treatment of sepsis and/or bacterial meningitis. However, the patient's mental status worsened and he became comatose. All cultures were sterile. A 'shotgun' diagnostic approach revealed ANA 1/2560 (H) and DNA 883 IU/dL. The creatinine continued to rise.

The impression was that of SLE complicated by anemia, thrombocytopenia, nephropathy and CNS disease. 'Pulse' steroids were administered for 3 days without subsequent improvement. Intravenous gamma-globulin failed to raise the platelet count. Rheumatology to the rescue!

Discussion Point

- Diagnosis and management of thrombotic microangiopathy (TMA)

Learning Objectives

- Describe the clinical presentation of TMA
- Discuss treatment options of TMA
- Review proposed modifications to the classification of lupus nephritis

Hot Topic

19 OPTIMAL ASSESSMENT AND MONITORING OF SLE PATIENTS IN CLINICAL PRACTICE

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Systemic lupus erythematosus (SLE) is a heterogeneous disease, characterised by a wide spectrum of clinical manifestations (ranging from arthritis and skin manifestations to renal insufficiency or central nervous system involvement) with an unpredictable relapsing-remitting course. SLE primarily affects young women in their childbearing lifetime, and perinatal complications due to SLE and/or the coexistence of the presence of antiphospholipid antibodies, can occur. Also, low complement levels and many antibodies can be found in SLE, among which anti-ds DNA and anti-Sm. During the disease course, irreversible organ damage can accrue due to SLE and/or its treatment. In addition, comorbidities can develop, including infections, osteoporosis and atherosclerosis. Compared to the general population, quality of life is reduced, frequently due