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 levotiroxyne 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

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, respiratory 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 (0.53 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!
to severe fatigue or psychological issues, amongst other reasons. As such reduced work capacity and/or work loss may occur.  

So, how should we assess and monitor patients with SLE?  

Disease activity (active disease [i.e. every 1–3 months] versus stable disease [i.e. every 6–12 months]), and specific conditions (i.e. pregnancy, thrombosis and bone fractures). Measuring disease activity with validated instruments (i.e. SLEDAI-2k, BILAG 2004) at each visit is recommended, in addition to a history and clinical examination, laboratory assessments (including blood, urine and immunology test) and eventually other investigations (i.e. imaging or biopsy); using these instruments results in better outcome, although the evidence is of low-moderate quality. However, high disease activity is associated with poor outcome (higher mortality rate, higher level of organ damage and comorbidities). Conversely, lower levels of disease activity and remission are associated with better outcome. It is important that clinical symptoms can be due to one or any combination of the following: disease activity, thrombosis or active inflammation, drug toxicity, chronic damage due to the disease or to its treatment, or to comorbidity (i.e. infection). Yearly assessment of disease damage (with SLICC Damage Index, SDI) is recommended, although the evidence of better outcome is of low-moderate quality. However, early and late damage is associated with small to moderate increase in mortality, further damage in the future and reduced quality of life and therefore is justified to assess. Regular assessment of cardiovascular (CV) risk factors in particular as well as corticosteroid-associated adverse events is recommended, with moderate-high quality evidence for CV assessment.

The rationale and principles of treat-to-target (T2T) in SLE and how to incorporate T2T in daily clinical practice will be discussed.  

Learning Objectives

- Describe the relevance of systematic assessment of disease activity, disease damage, comorbidities and quality of life
- Explain the current clinical practice guidelines in SLE, and define unmet needs in order to improve our daily practice
- Discuss the current state of T2T in SLE

REFERENCES