dsDNA and LAC positivity. The antiepileptic therapy was initially modified and then, as no more crises were present, interrupted. However, anemia, leukopenia, hypocomplementemia, ANA and anti-dsDNA persisted, and the diagnosis of idiopathic systemic lupus erythematosus (SLE) was made. After treatment with hydroxychloroquine and low-dose prednisone the girl clinically improved and her laboratory results normalized. This case report is suggestive of the complexity in differentiating SLE and DILE and underlines the importance of a long and careful follow-up.

Discussion Points
- Medications that can trigger lupus symptomatology
- Triggers of lupus or of lupus-like disease (autoimmunity in general) with TNF inhibitors used for arthritis
- Risks of prescribing such medications (i.e., anti-TNF) in patients with inflammatory arthritis and pre-existing autoantibodies or a family history of autoimmune disease

Case 5: Bleeding and thrombosis in juvenile systemic lupus erythematosus
Rolando Cimaz

A young girl with immune thrombocytopenic purpura was also found to have antiphospholipid antibody syndrome. This case describes the complexity of therapeutic management linked to the risk of bleeding and thrombosis.

Irene is 15-year-old. In recent hours she has experienced intermittent claudication and lower right limb pain. Her physical exam reveals swelling and tenderness of right calf, pain to compression and mobilization of right foot. Laboratory tests reveal WBC 21.610 (N 77%); Hb 13.1 g/dl; PLT 91000/ mmc; aPTT 48.4 sec, PT 99%; fibrinogen 236 mg/dl; D-Dimer 0.59 mg/L. Ultrasound revealed thrombosis.

Past medical history showed that 8 months before she had suffered from severe metrorrhagia. Laboratory tests had shown: PLT 7000, Hb 10.2 g/dl, Coombs direct test +, PT 1.18, aPTT 76 sec; IgM e IgG anticardiolipin +. Therapy consisted in intravenous immunoglobulin (two infusions) and then oral steroids. Five relapses occurred, and laboratory showed: ANA+ (1:160), ENA -, antiphospholipid -, C3 85, C4 7.5, LAC +. Repeat laboratory test showed ANA+, ENA-, antiphospholipid +, anti-b2 glycoprotein -, LAC +.

Discussion Points
- For thrombosis Heparin 6000U/x2/day. For how long? And for thrombocytopenia? Oral steroids (2 mg/kg/day); Mycophenolate mofetil (750 mg/m² x 2/day). But despite this therapy, she relapsed. So > rituximab 750 mg/m²/2 weeks. For SLE hydroxychloroquine was given.

Learning Objectives
- Distinguish between idiopathic and drug-induced SLE
- Describe the treatment of APS in children
- Discuss treatment options for hematologic SLE

Workshop

Abstract 21 Figure 1 T2 magnetic resonance sagittal slices showing a high intensity signal from the ponto-medullary junction to the cervical (A) and thoracic (B) medulla

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difficulties and was admitted at the Emergency Room. At physical examination, dysarthria and paresis of left upper limb was confirmed. Blood pressure was 160/95 mm Hg.

Discussion Point
- Recognising and treating focal cerebral involvement in SLE

Learning Objectives
- Recognise and describe clinical, laboratory and imaging features, which help assess focal cerebral involvement in patients with SLE.
- Explain the principles and strategies for the management of cerebral manifestations in SLE.
- Demonstrate clinical awareness of potential severe cerebral complications in SLE

Case 2: A woman with recurrent myelitis and SLE

Thomas Huizinga

A woman was diagnosed with systemic lupus erythematosus (SLE) at the age of 28 years old based on arthritis, skin erythema, mouth ulcers, sunlight hypersensitivity, ANF +, anti dsDNA + and anticyclicolipin antibodies. At the age of 34 she developed papillitis of both eyes with vision loss. Her MRI revealed no abnormalities and she was treated with methylprednisone 500 mg for 5 days. At the age of 35 she developed a recurrence, no neuritis bulbaris was observed and SLE was thought to be the most likely cause so she was treated with cyclophosphamide 750 mg/kg for 6 months and prednisone 60 mg for 4 weeks and then 10 mg/kg lowering every 4 weeks. After 6 months she had completely improved. At the age of 37 she gradually developed, over a number of weeks, problems urinating and sensory disturbances left thigh and right leg, physical exam revealed hypesthesia in her left leg, and feelings of a different temperature in legs compared to arms.

MRI revealed transverse myelitis at C6-Th2, compatible with SLE and she was retreated with cyclophosphamide and prednison (figure 1). Subsequently, she did quite well for a half year. In 2020 she started to suffer from recurrent infections and her IgG levels were 5.6 mg/ml after which intravenous IgG supplention was started and the infectious problems disappeared.

Discussion Point
- Diagnosis, management and recurrence of myelitis in SLE

Learning Objectives
- Describe the clinical presentation of myelitis and its recurrence
- Explain treatment options of myelitis
- Discuss the role of aquasporin-4 antibodies in myelitis