Strategies for Minimizing Corticosteroid Exposure in SLE

Zahi Amoura. French National Reference Center for SLE and APS, Pitie-Salpetriere Hospital, Paris, France

10.1136/lupus-2021-la.15

Glucocorticoids (GCs) play a central role in the treatment of active systemic lupus erythematosus (SLE). Long-term GC-related side effects (i.e., infections, diabetes mellitus, cataract, osteoporosis, gastrointestinal bleeding and cardiovascular disease), leading to the development of irreversible organ damage, mean that clinicians must develop strategies for minimizing GC exposure in SLE.1 Initiation of oral GC should be avoided, especially when there are effective therapeutic alternatives as for cutaneous and articular manifestations. In patients with lupus nephritis, starting GC with a medium prednisone dose (0.5 mg/kg/day) is as effective as high-dose one (1 mg/kg/day) prednisone.2 Use of intravenous methylprednisolone (MP) pulses (usually 250–1000 mg/day for 3 days) may allow for a lower starting dose and faster tapering of oral GC.2 Early initiation of immunosuppressive drugs can facilitate a more rapid GC tapering and may prevent SLE flares.4 Long-term GC administration with doses of ≤5 mg/day prednisone produces an acceptably low level of harm, with the exception of patients at high cardiovascular risk who may require preventive measures.3 Withdrawal of low dose prednisone is also recommended by EULAR, when possible,1 but recent data suggest that this exposes SLE patients to an increased risk of flare, whereas its long-term maintenance is not associated with increased damage scores.6

Learning Objectives

- Describe strategies for minimizing corticosteroid dose at treatment initiation for lupus nephritis, cutaneous lupus and lupus arthritis
- Describe strategies for minimizing corticosteroid exposure when SLE remission has been reached

REFERENCES

There is little question that our colleagues in rheumatology who treat patients with rheumatoid or psoriatic arthritis have outdone us ‘lupologists’ in bringing new therapies to the community. Biologics have truly been transformative for those patients with inflammatory arthritis. We are now starting to see the same successes in lupus. As we celebrate the 10th anniversary of the FDA approval of belimumab, lupus community recently witnessed the approval of two drugs, belimumab and voclosporin, for the treatment of lupus nephritis. In addition, several Phase 2 studies yielded favorable results and will be progressing to Phase 3.1 Favorable results and will be progressing to Phase 3.

Learning Objectives

- Describe unmet needs in SLE treatment
- Discuss biologic targets for SLE drug development
- Explain recent clinical trial results

REFERENCES


LUPUS TREATMENT IN THE NEXT DECADE: THE NEXT DECADE IS UPON US

Richard Furie, Zucker School of Medicine at Hofstra/Northwell, New York, USA
10.1136/lupus-2021-la.17

There is little question that our colleagues in rheumatology who treat patients with rheumatoid or psoriatic arthritis have outdone us ‘lupologists’ in bringing new therapies to the community. Biologics have truly been transformative for those patients with inflammatory arthritis. We are now starting to see the same successes in lupus. As we celebrate the 10th anniversary of the FDA approval of belimumab, the lupus community recently witnessed the approval of two drugs, belimumab and voclosporin, for the treatment of lupus nephritis. In addition, several Phase 2 studies yielded favorable results and will be progressing to Phase 3.1 Drug development activity is currently unprecedented, and there is no doubt that research advances will improve outcomes and ensure brighter futures for our patients with systemic lupus.

Learning Objectives

- Describe unmet needs in SLE treatment
- Discuss biologic targets for SLE drug development
- Explain recent clinical trial results

Case 1: 35-year-old Mestizo female

Bernardo Pons-Estel

A 35-year-old Mestizo female was diagnosed with systemic lupus erythematosus (SLE) in 2005 based on polyarthritis, malar rash, photosensitivity, mucosal ulcers, positive ANA and anti-dsDNA, and low complement. She was treated with prednisone 20–30 mg/day and hydroxychloroquine (HCQ) 400 mg/day. In June 2010, lupus pneumonitis was diagnosed.

In July 2018, she was first admitted to our hospital. She was cushingoid and had fever, fatigue, malar rash, oral ulcers, alopecia, polyarthritis, oedema, multiple purplish-red streaks, and active erythematous, palpable and painful subcutaneous indurated nodules/plaques located on her face, proximal lower extremities and abdomen; some were ulcerated.

Laboratory tests

- RBC 3.8 (x10 12/L), hemoglobin 11.8 g/dl, WBC 2.3 (x10 9/L), platelets 62 (x10 9/L), ESR 8 mm, CRP 0.8 mg/L, serum ferritin 1,487 ng/ml, ALAT 70 U/ml, ASAT 26 U/ml, GGT 64 U/L, BUN 43 mg/dl, serum creatinine 1.54 mg/dl, GFR 67 mL/min, cholesterol 186 mg/dL, triglycerides 149 mg/dl, proteinuria 210 mg/24 h, ANA 1/320, speckled pattern, anti-Sm (+) and anti-dsDNA, anti-U1RNP, anti-Ro and anti-La all (-). C3 75 mg/dl, C4 10, Coombs test (-), procalcitonin 0.29 ng/ml (<0.5). VDRL and viral serologies were negative.

A cutaneous ulcer culture showed proteus mirabilis. Her SLEDAI was 19. Three skin biopsies of indurated lesions all showed lobular panniculitis.

She was treated with IV methylprednisolone (500 mg/day/3 days), and high-dose intravenous immunoglobulin, and discharged with mycophenolate mofetil 1 g/day, HCQ 400 mg/day, prednisone 20 mg/day and hydroxychloroquine 200 mg/day and TMS-SMX 860/160 BID for 5 days. A week later she was re-admitted with fever, pancytopenia, hepatosplenomegaly, lymphadenopathy and several painful, indurated erythematous lesions. Her SLEDAI was 13. A bone marrow aspiration and biopsy was interpreted as a macrophage activated syndrome in the context of SLE exacerbation and treated with IV dexamethasone, colony stimulating factor, and rituximab 1000 mg. Despite treatment, she remained severely ill with fever, asthenia, petechiae and purpura on her abdomen and thighs, and pancytopenia. Due to disease severity, treatment with etoposide was indicated. Finally, the patient presented an acute episode of respiratory distress followed by death.

Discussion Points

- Interpreting the different skin manifestations in a patient with SLE
- Analyzing complications and differential diagnoses with other associated diseases

Case 2: 28-year-old Caucasian female

Annegret Kuhn

A 28-year-old Caucasian female was diagnosed with systemic lupus erythematosus (SLE) in 1996 and presented with severe, erythematous, scarring discoid lesions on the scalp,