daily, increase immunosuppression, either by increasing dose of immunosuppressive being given (e.g. azathioprine) or adding a new immunosuppressive.[4] During this time, consider whether the patient is compliant with the regimen you established prior to flare: Compliance (defined as taking the medication as directed 80% of the time) occurs in only 50–70% of SLE patients: Poor compliance is associated with young patients, those with poor social and economic support systems, less educated, and those with strong beliefs in adverse effects of Western medications and/or utility of other healing approaches.[5] I may choose to use intravenous medications in situations such as these. Most SLE patient use complementary supplements: Those that should be discouraged include St John's wort, which interferes with metabolism of many drugs. Vitamin D levels should be normalised. N-acetylcysteine, polyphenols, omega-3 fatty acids, fish oil and thundervine herb may all have benefits but have not reached general acceptance in the medical community. Since the number of SLE flares is strongly correlated with damage to many body systems, with poor quality of life, and with most of the causes of death of SLE patients, physicians and other caregivers are obligated to identify SLE flares early and suppress them.

Learning objectives

- Differentiate between infection and flare in patients with SLE
- Describe key biomarkers associated with SLE flare
- Explain how best to differentiate infection from SLE flare

REFERENCES

1. Parker B, Bruce I. *Clinical markers, metrics, indices and clinical trials*. Dubois' Lupus Erythematosus and Related Syndromes. 9th edition, Wallace D and Hahn B eds: Elsevier, 2019.
2. Ospina FE, Echeverri A, Zambrano D, *et al*. Distinguishing infections vs flares in patients with systemic lupus erythematosus. *Rheumatology (Oxford, England)* 2017;**56**(suppl_1):i46-i54.
3. Fanouriakis A, Kostopoulou M, Alunno A, *et al*. 2019 update of the EULAR recommendations for the management of systemic lupus erythematosus. *Ann Rheum Dis* 2019;**78**(6):736–45.
4. Lu R, Guthridge JM, Chen H, *et al*. Immunologic findings precede rapid lupus flare after transient steroid therapy. *Scientific reports* 2019;**9**(1):8590–90.
5. Costedoat-Chalumeau N, Houssiau F, Izmirly P, *et al*. A Prospective International Study on Adherence to Treatment in 305 Patients With Flaring SLE: Assessment by Drug Levels and Self-Administered Questionnaires. *Clinical pharmacology and therapeutics* 2018;**103**(6):1074–82.

02 MANAGEMENT OF REFRACTORY DISCOID LUPUS

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Discoid lupus erythematosus (DLE) is the most common form of chronic cutaneous lupus erythematosus (CCLE) and occurs as localised form (ca. 80%) or disseminated/generalized form (ca. 20%). The localised form presents with lesions on the face and scalp, especially the cheeks, forehead, ears, nose, and upper lip, whereas the generalized form presents with lesions involving the upper part of the trunk and the extensor aspects of the extremities.[1] The lesions of DLE consist of sharply-demarcated, coin-shaped ('discoid') indurated erythematous plaques with adherent follicular hyperkeratosis.[2] During the course of the disease the lesions may expand at the periphery with an active erythematous border and hyperpigmentation, resulting in atrophy, scarring, telangiectasia and hypopigmentation in the center of the lesions. At the scalp, eyebrows and bearded regions of the face, DLE can progress to total, irreversible scarring alopecia. In the perioral region, the lesions can lead to characteristic pitted acneiform ('vermicular') scarring.[3] Mucosal DLE presents with chronic buccal plaques, showing typical roundish lesions with peripheral white hyperkeratotic striae and central atrophy, erosion or ulceration. Exposure to the sun or irritating stimuli ('Koebner phenomenon'), such as trauma, can provoke or exacerbate the disease.[4] DLE lesions occur in approximately 15–25% of patients in the course of SLE, but more than 95% of patients with DLE lesions suffer from cutaneous disease only. First-line treatment options in DLE include topical corticosteroids or calcineurin inhibitors; in patients with disfiguring and widespread disease, systemic agents need to be applied.[5] The first-line systemic treatment is antimalarials, but some patients are therapy-resistant and immunosuppressive agents, such as methotrexate or mycophenolate mofetil, are used as alternative therapeutic option. The monoclonal antibody belimumab, which is approved for SLE as an adjunct therapy for patients with autoantibody-positive disease who despite standard therapy show high disease activity, may be effective, but needs to be evaluated using validated skin scores.

Learning objectives

- Describe the different types of skin manifestations in DLE
- Explain the preventive strategies for DLE including photoprotection
- Discuss the topical and systemic treatment options for DLE

REFERENCES

1. Costner M, Sontheimer R, Provost T. Lupus erythematosus. In: Sontheimer R, Provost T, eds. Cutaneous manifestations of rheumatic diseases. Philadelphia: Williams & Wilkins, 2003.
2.. Kuhn A, Landmann A, Bonsmann G. Cutaneous lupus erythematosus. In: GC T, ed. Systemic Lupus Erythematosus. 1st ed. Amsterdam: Systemic Lupus Erythematosus, 2016:333–40.
3. Chang YH, Wang SH, Chi CC. Discoid lupus erythematosus presenting as acneiform pitting scars. *International journal of dermatology* 2006;**45**(8):944–5.
4. Ueki H. Koebner phenomenon in lupus erythematosus with special consideration of clinical findings. *Autoimmun Rev* 2005;**4**(4):219–23.
5. Kuhn A, Aberer E, Bata-Csorgo Z, *et al*. S2k guideline for treatment of cutaneous lupus erythematosus - guided by the European Dermatology Forum (EDF) in cooperation with the European Academy of Dermatology and Venereology (EADV). *Journal of the European Academy of Dermatology and Venereology : JEADV* 2017;**31**(3):389–404.

03 MEMBRANOUS NEPHROPATHY: HOW AGGRESSIVE SHOULD I BE?

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Compared to proliferative lupus nephritis (PLN), membranous lesions are less inflammatory, have a more benign course, require less aggressive therapy, and have better prognosis.[1] The 2012 EULAR/EDTA recommendations for lupus nephritis[2] were recently updated (Fanouriakis A, *et al* 2019 to be submitted).

Goals of therapy Optimisation (preservation or improvement) of renal function with at least 25% reduction in proteinuria at 3 months, 50% at 6 months and a urine protein/creatinine ratio (UPCR) target below 0.5–0.7 mg/g by 12 months (complete renal response).

Initial therapy Glucocorticoids and immunosuppression if UPCR exceeds 1 mg/g despite the optimal use of renin-

angiotensin-aldosterone system blockers, or from the beginning when nephrotic-range proteinuria is present. In pure Class V nephritis, mycophenolate mofetil (MMF) (dose 2–3 g/day; or mycophenolic acid [MPA] at equivalent dose) in combination with pulses IV methylprednisolone (total dose 500–2500 mg) followed by oral prednisone (20 mg/day, tapered to ≤5 mg/day by 3 months) can be used as initial treatment based on better efficacy/toxicity ratio. Alternative options include high-dose IV cyclophosphamide (0.5–0.75 g/m^2 monthly for 6 months), calcineurin inhibitors (ciclosporin, tacrolimus) or their combination with MMF/MPA, particularly in patients with severe nephrotic syndrome.

Subsequent therapy MMF/MPA (dose: 1–2 g/day) – especially if it was used as initial treatment – or azathioprine (AZA); 2 mg/kg/day – preferred if pregnancy is contemplated – for at least 3 years, in combination with low-dose prednisone (2.5–5 mg/day) when needed. If sustained complete response, gradual drug withdrawal, glucocorticoids first, can then be attempted, with immunosuppressives following after 3–5 years in complete response. Continuation, switching or addition of calcineurin inhibitors can be considered in pure Class V nephritis at the lowest effective dose taking into consideration the possibility for nephrotoxicity.

Refractory disease Treatment may be switched to one of the alternative initial therapies mentioned above or rituximab (1000 mg on days 0 and 14). In a recent randomised controlled trial of rituximab in idiopathic membranous nephropathy, rituximab was equal to cyclosporine in achieving remission at 12 months (60% vs 52%) but superior to cyclosporine in maintaining remission at 24 months (60% vs 20%).[3]

Adjunct therapy ACE-inhibitors or angiotensin receptor blockers for patients with UPCR >0.5 mg/g or hypertension. Antilipidemics and hydroxychloroquine at a dose not to exceed 5 mg/kg/day. Anticoagulant treatment in cases of nephrotic syndrome with serum albumin <20 g/L, presence of antiphospholipid antibodies or other pro-thrombotic conditions.

Learning objectives

- Discuss how membranous differs from proliferative disease
- Describe how to treat nephropathy membranous
- Explain targets of therapy
- Describe treatment of refractory disease
- Explain adjunct therapy and what it entails

REFERENCES

1. Ward F, Bargman JM. Membranous Lupus Nephritis: The Same, But Different. *American journal of kidney diseases : the official journal of the National Kidney Foundation* 2016;**68**(6):954–66.
2. Bertsias GK, Tektonidou M, Amoura Z, *et al*. Joint European League Against Rheumatism and European Renal Association-European Dialysis and Transplant Association (EULAR/ERA-EDTA) recommendations for the management of adult and paediatric lupus nephritis. *Ann Rheum Dis* 2012;**71**(11):1771–82.
3. Fervenza FC, Appel GB, Barbour SJ, *et al.* Rituximab or Cyclosporine in the Treatment of Membranous Nephropathy. *The New England journal of medicine* 2019;**381**(1):36–46.