Six months following the beginning of Covid-19 pandemic in China, data on the risk of SARS-CoV-2 infection among patients with autoimmune rheumatic diseases are now available. However, the rapid spread of the pandemic has not allowed proper design of prospective studies, thus evidence came mostly from case series and observational studies.

The early enthusiasm on hydroxychloroquine (HCQ) antiviral properties should not suggest that patients who are long-term treated with antimalarials, such as patients with systemic lupus erythematosus (SLE), are protected against SARS-CoV-2 infection. Indeed, a French report on 17 HCQ-treated SLE patients damped the enthusiasm.1

A recent report from Covid-19 Global Rheumatology Alliance has described 80 SLE patients with Covid-19, mostly females under 65 years of age, 64% of whom were already taking HCQ before the infection: the rate of hospitalisation and the need for intensive care did not differ between patients who were and those who were not taking HCQ.2 A study group from Northern Italy – the Italian epicentre of the pandemic – reported an incidence of 2.5% of Covid-19 (higher compared to the general population of the same region) in 165 patients with SLE.3

Patients with SLE are possibly at risk of developing symptomatic or severe Covid-19, not only because of their disease or treatment but as a consequence of associated comorbidities known to worsen the outcome of SARS-CoV-2 infection.4 5

What do we know so far? SLE patients should not withdraw their medication. Before drawing any other conclusion, large registry data are needed to clarify the incidence and the outcome of Covid-19 in patients with SLE.

Learning Objectives
- Describe the current evidence for risk of SARS-CoV-2 infection among patients with autoimmune rheumatic diseases, notably SLE
- Explain why it is important to ensure robust evidence are available to clarify the outcome of Covid-19 in patients with SLE

REFERENCES
aggressive intervention, about a quarter of patients continue to progress to end stage renal disease (ESRD). The factor of drug adherence, however, may draw the fine distinction between refractory (or resistant) and relapsing LN, with the latter substantially more common than the former. Nearly half of patients with proliferative LN who initially achieve a complete response to immunosuppressive therapy will have a relapse or renal flare following cessation or reduction of immunosuppression. Other risk factors for refractoriness include genetics and comorbidities like hypertension, diabetic nephropathy and antiphospholipid antibodies, each of which must be effectively addressed in the overall management of these patients.

Treatment options for refractory LN include switching or multitargeted therapy with immunosuppressives cyclophosphamide, mycophenolate derivatives and calcineurin inhibitors (cyclosporine A, tacrolimus, and recently voclosporin). Literature abounds with the use of rituximab in refractory LN including trials exploring the sequential use of rituximab plus cyclophosphamide followed by belimumab. Other modalities such as extracorporeal treatment (plasma exchange or immunoadsorption) and stem cell transplantation may be tried in special situations. Novel insights of LN pathogenesis have led to the development of new or re-purposed drugs including obinutuzumab, itolizumab, and iguratimod which can significantly prevent progression to ESRD. Finally, the timely, aggressive and highly individualised management of LN which reinforces strict patient adherence is key to enhanced outcomes.

Learning Objectives
- Define refractory and/or relapsing LN
- Identify risk factors for refractoriness among LN patients
- Describe the management approach to refractory LN
- Describe advances in drug development for LN

REFERENCES

The aim of this presentation is to provide concise information regarding diagnosis and management of immune mediated hematological manifestations of systemic lupus erythematosus (SLE), specifically autoimmune hemolytic anemia (AIHA), immune mediated thrombocytopenia and immune mediated leukopenia. Studies on hematological manifestations of SLE are scarce and with low quality of evidence, therefore most existent guidelines are supported by hematological data from non-SLE patients. Of note, SLE patients have some peculiarities that must be taken into consideration when choosing the best treatment approach for their hematological conditions.

The key points of each cytopenia are:

a. Autoimmune hemolytic anemia (AHAI) in SLE is usually mediated by warm antibodies (IgG). First-line treatment is glucocorticoid, but there is no consensus to guide second-line therapy. Rituximab and immunosuppressive drugs can be used in refractory cases.

b. Immune thrombocytopenia is an important prognostic indicator of survival in SLE patients. Treatment is usually indicated for patients with a platelet count <30 × 109/mm3. First-line therapy remains glucocorticoids (dexamethasone is the hematologists 1st choice), but sustained response after glucocorticoid discontinuation in SLE patients is unlikely to happen. Rituximab and immunosuppressive drugs can be used in refractory cases. Thrombopoietin agonists and splenectomy should be avoided due to high risk of infection and thrombosis.

c. Neutropenia may be severe and can lead to morbidity and mortality from increased susceptibility to infection. Severe neutropenia can be successfully treated with granulocyte colony-stimulating factor. Lymphopenia is most often related to drug toxicity and disease activity. Severe lymphopenia may require the use of prophylactic therapy to prevent opportunistic infections. For all cytopenias an extensive evaluation of other causes, particularly drugs and infection should be performed.

Learning Objectives
- Identify and manage immune mediated anemia in lupus
- Discuss diagnosis and treatment options for thrombocytopenia in lupus
- Explain the diagnosis and management of leukopenia in lupus

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The aim of this presentation is to provide concise information regarding diagnosis and management of immune mediated hematological manifestations of systemic lupus erythematosus (SLE), specifically autoimmune hemolytic anemia (AIHA), immune mediated thrombocytopenia and immune mediated leukopenia.