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Case Report

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COVID Infection in A Systemic Lupus Erythematosus Patient (About One Case)

Y. HAFIANI*, M.A. ELABIDI *, A. LAKRAFI, M. ELAIASSI, A. NSSIRI*, R. ALHARRAR

Service de réanimation COVID 19 pavillon 11 CHU ibnrochd

***Corresponding authors:** Yassine Hafiani and Mohammed Amine El Abidi, Service de réanimation COVID 19 pavillon 11 CHU ibnrochd. Email: Elabidimohammedamine@gmail.com

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Introduction

COVID-19 is a systemic infectious disease with significant respiratory involvement, which can cause acute respiratory distress syndrome. COVID-19 is currently spread around the world with a dramatic impact on public health.

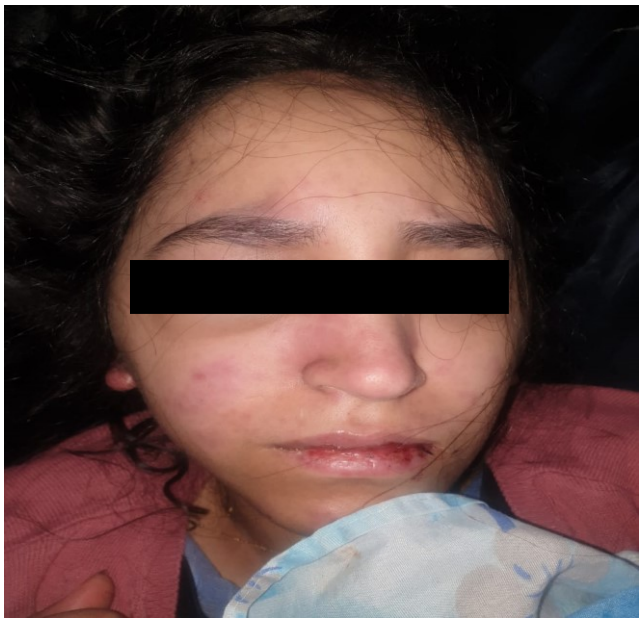
The virus is spread mainly by droplets, but direct contact and fecal excretions are other possible sources of infection. Vertical transmission may be possible. The primary target of SARS-CoV-2 is the upper respiratory mucosa and the angiotensin-converting enzyme 2 (ACE2) acts as a functional receptor for viral peaks and ultimately viral entry into host cells. Expression of the ACE2 gene from the SARSCoV-2 cellular receptor has been demonstrated in a number of human tissues, including skin and adipose tissue.

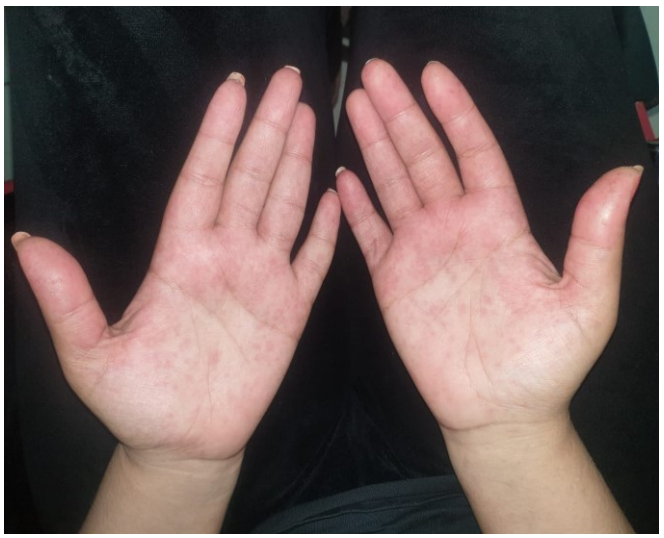
Systemic lupus erythematosus (SLE) is an autoimmune disease with broad clinical polymorphism characterized by the production of antinuclear antibodies, particularly native anti-DNA antibodies. Its pathogenesis is still debated. Many genetic, endocrine, immunological and environmental factors contribute to the onset and then maintenance of the disease.

Patients with systemic lupus erythematosus (SLE) may be at increased risk for SARS-COV 2 infection due to the organ damage from SLE, immunosuppressive drugs and hypercoagulability.

Case report

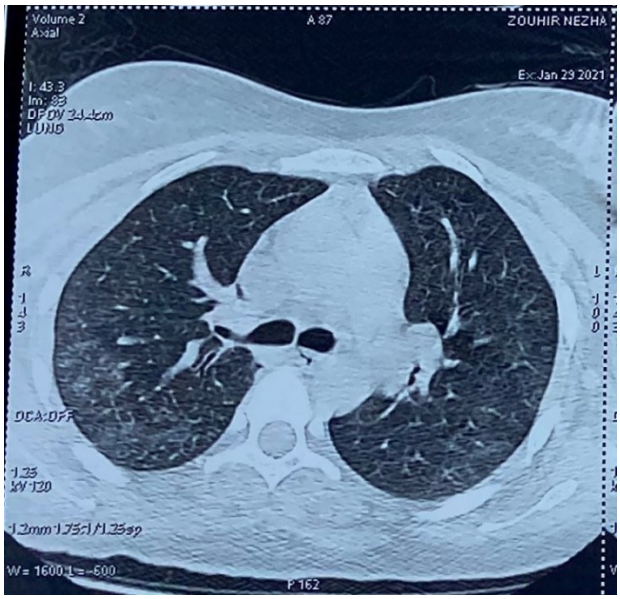
Patient aged of 27-year-old with no particular pathological history. 15 days before her admission she presented a maculopapular erythema on the face, trunk and palmoplantar area, the evolution was marked by worsening of the lesions with installation of facial, palpebral and limb edema; 10 days later, the patient presented fever and diffuse myalgia with difficulty in breathing, which motivated the patient to consult.





On admission the patient was conscious patient (GSW 15/15), afebrile, eupneic at 18 cpm, the pulse oxymetry was at 93% in ambient air and 99% using nasal canula 4l / min, a blood pressure at 130/70 and a heart rate of 82 bpm; Cardiac and pleuropulmonary auscultation was without abnormality.

SARS-COV2 PCR was ordered and tested positive, chest CT scan showed a ground glass appearance with an extent measured at 10%. The ECG demonstrated a regular sinus rhythm at 92 bpm, a fixed PR space at 0.2s, fine QRS with QTc at 436ms without repolarization disturbances. Transthoracic ultrasound revealed a 52% ejection fraction and grade III aortic insufficiency.



In the biological assessment: CRP at 3.2 mg / l, hemoglobin at 9.5 g / dl, GB at 1230 with PNN at 730 and lymphocytes at 320 and platelets at 145,000. Urea at 0.5 and creat at 7.4, serum sodium at 135 mmol / l, serum potassium at 3.9 mmol / l, fibrinogen at 1.34 g / l, prothrombin level at 127% and a TCA at 28.5 / 28, ferritinemia at 1010, LDH at 510, pro calcitonin at 0.22. The HIV serology was negative, the syphilitic serology was negative the C3 fraction of the complement was reduced to 0.33 and CH50 to 13.80.

Therapeutically, the patient was put on oxygen therapy using 4l / min oxygen glasses, hydroxychloroquine 200 mg three times a day, azithromycin 500 mg / day on the first day then 250 mg / day for six days, dexamethasone 6 mg / day for 10 days, aspirin 100 mg / day, enoxaparin 100 IU / kg / day, zinc 90 mg / day, vitamin C 1 g / day and vitamin D 25,000 IU / week.

In the follow up the anti-nuclear and anti-DNA antibodies were positive; therefore, the patient was put under methylprednisolone 1 g per day for 3 days, the evolution was presented with an improvement in the respiratory system, with a withdrawal from oxygen therapy and regression of facial and limb edema and regression of erythema.

Discussion

COVID-19 is an infectious disease with respiratory tropism, the symptoms have been widely described with the most frequent signs, cough, dyspnea, fever, and myalgia and in critical cases a severe acute respiratory syndrome. However cutaneous manifestations are not to be excluded.

A study in Spain using a representative sample of 375 cases found five clinical patterns of cutaneous manifestations of COVID-19: acral areas of erythema with vesicles or pustules (19%), other vesicular eruptions (9%), urticarial lesions (19%), maculopapular eruptions (47%), livedo or necrosis (6%). These patterns appear at different times in the disease, and are associated with different duration, severity and probably prognosis.

Systemic lupus erythematosus (SLE) is a chronic systemic autoimmune disease with an innate and adaptive immune response disorder. SLE patients could be at increased risk from COVID-19 due to immunosuppressive medications, hypercoagulability and the multiorganic damage. The reciprocal interaction between dysregulation of the immune response and infections in SLE is complex and only partially understood and it constitute potential triggers for SLE flares.

Viral infections among immunosuppressed patients can cause significant morbidity and mortality. The overall rates of mortality among SLE patients remains higher than the general population and it's is due to infection. A lot of research have shown that SLE patients have an increased infectious risk and it depends on the level of disease activity and the treatment protocols. Angiotensin converting enzyme two (ACE2) is a functional receptor for the SARS-CoV-2 spike glycoprotein, with SARS-CoV-2 binding to target host cells though ACE2. There is evidence to show that in SLE patients, ACE2 is over expressed on CD4 positive T-helper cells compared to healthy controls, and this has led some to suggest that SLE patients may be at higher risk of viraemia.

SLE and COVID-19 have been shown to cause respiratory, cardiac and hematological damages. Due to similarities between them, the treatment bases of patients with SLE may help discovering therapeutic options for COVID-19.

The mainstay of treatment for SLE involves corticosteroids and other immunosuppressive therapies that may cause further vulnerability to COVID-19 infection. Meanwhile, immunosuppressant treatment has been proved to lower inflammation and to reduce the chances of developing acute respiratory distress syndrome (ARDS) in patients already infected with coronavirus.

The hydroxychloroquine has been used as a COVID-19 treatment due to its antiviral effects. However, recent studies show no benefit from its use. Furthermore, there is no association between hydroxychloroquine and protection from COVID-19. An Italian survey and Spanish registry data looking at rheumatic patients on immunosuppressive therapies have recorded a low number of patients with SLE and confirmed COVID-19 with no ICU admissions or deaths.

Many SLE patients are on long-term corticosteroid treatment which is associated with a higher risk of infection.28 Data from the C19-GRA registry has shown that over a third of enrolled patients have been on corticosteroids prior to COVID-19 diagnosis 17 and that a higher dose of corticosteroids exposes to an increased risk of hospitalisation.19 stopping the use of corticosteroids suddenly may lead to disease flare and Addisonian symptoms. The European League Against Rheumatic Disease (EULAR), the American College of Rheumatology (ACR) and the National Institute of Clinical Excellence (NICE) have maid guidelines suggesting that patients on longterm steroids should not have these stopped suddenly and that discussions with patients regarding starting new treatment should include consideration of deferring starting treatment and the risks of delaying treatment.

Antiphospholipid Syndrome (APS) is characterized by the presence of at least one clinical criterion: thrombosis or pregnancy morbidity such as foetal loss or early delivery; and at least one laboratory criteria: lupus anticoagulant (LAC), anti-cardiolipin antibodies or anti-b2 glycoprotein antibodies. The presence of antiphospholipid antibodies is often associated with thrombosis and can be transient or persistent. Many studies have linked COVID-19 with a hypercoagulable state and thrombosis. There is evolving evidence for high thrombotic risk in patients with severe COVID-19 infection. A Dutch study of 184 intensive care patients with COVID-19 pneumonia found 31% of patients had thrombotic complications.87 A study from Wuhan, China found that patients who did not survive had a significantly higher D-dimer levels, prolonged thrombin time and activated partial thromboplastin time.88 A meta-analysis of coagulation in COVID-19 noted that prothrombin time and D-dimer were significantly higher in patients with severe COVID-19.89 Unfortunately, antiphospholipid antibodies were not assayed; however, there are numerous case studies demonstrating the presence of antiphospholipid antibodies in COVID-19 related thrombosis .

Conclusion

Patients with autoimmune diseases are vulnerable to infections because of the aberrant immune responses the corticosteroid treatment, all along with other immune-suppressants and immune-modulator drugs. Many aspects of lupus and COVID-19 are shared including some demographics of patient populations affected and aberrant immune responses while some such as gender-bias are strikingly distinct in the two diseases, with lupus predominantly afflicting women and COVID-19 with worse outcomes in men. Aberrant cellular, humoral and cytokine immune responses including lymphopenia, proinflammatory cytokines, aberrant B and T cell responses may likely influence the severity and disease outcomes of COVID-19 in patients with immune-mediated and autoimmune diseases. Better understanding of the

intricacies of the immune response will be important in guiding management strategies for these patients.

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