CASE REPORT

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Systemic Lupus Erythematosus Flare Triggered by COVID-19 Infection: A Case-Based Review

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ABSTRACT

Introduction: The coronavirus disease 19 (COVID-19) and autoimmune disease has been associated bidirectionally, several reports has shown COVID-19 precipitate an exacerbation of an autoimmune disease that has already stable. Recognize and treatment of flare in SLE condition with COVID-19 is challenging in this pandemic era. This review aims to report a case SLE patient who experienced severe flares after being infected with COVID-19 and review of the literature.
Case report: We presented a 27-year-old female with a history of Systemic Lupus Erythematosus (SLE) who experienced severe flares after being infected with COVID-19 and a review.
Methods and Results: A total of 72 potentially relevant citations were identified. After removing the duplicate citations, the title, and abstracts of 61 articles were evaluated and 11 relevant articles were reviewed in detail. A total of 72 potentially relevant citations were identified. After removing the duplicate citations, the title, and abstracts of 61 articles were evaluated and 11 relevant articles were included.
Conclusion: The COVID-19 pandemic is a devastating situation all over the world. SLE patient has already been in a susceptible condition as the disease progressed and continued having immunosuppressant therapy also one of the risk factors. A Flare condition can be happened during the COVID-19 Infection or after the infection is resolved. In SLE patient having COVI-19, close monitoring, high adherence to the therapy, and the health protocol are needed.

Keywords: COVID-19, Systemic Lupus Erythematosus (SLE), Flare.
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INTRODUCTION

Coronavirus disease 2019 (COVID-19) is a respiratory infection caused by the newly emerging coronavirus, Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-CoV-2). The current COVID-19 outbreak is a particular concern for the management of patients with immunological disorders including Systemic Lupus Erythematosus (SLE). Patients with SLE have an increased risk of developing severe infections because of intrinsic interference with their immune response, the use of immunosuppressive drugs, and the potential for organ damage associated with their disease.[1,2] Infection can exacerbate SLE activity and be reported to be a significant cause of death among SLE patients (37.7%).[3] COVID-19 also has shown that can precipitate an exacerbation of an autoimmune disease that has already stable.[4] Here in this case report, we discussed an SLE patient who experienced severe flares after being infected with COVID-19 and we also present a review of the literature.

CASE DESCRIPTION

A 27-year-old female with a history of Systemic Lupus Erythematosus (SLE) came to the Emergency Department with the chief complaint of fever and swelling all over her body. She also complained of pain in both of her ankles, wrists, and knee joints. She has already been diagnosed with moderate SLE three months prior to admission with Anti-Nuclear Antibody (ANA) >1:1000 positive anti-nuclear antibody with Cytoplasmic dense fine speckled and had given treatment Chloroquine 300 mg BID, Methylprednisolone 16 mg twice daily, Methotrexate 7.5 mg once weekly, Folic Acid 5 mg twice daily, Calcium lactate 500 mg once daily (OD), D3 1.000 IU twice daily. Her symptoms were improved, and she was in a stable condition. Two months before admission she came to polyclinic care with symptoms of fever, cough, and runny nose. A Rapid Antigen Swab for SARS-CoV-2 performed with the result was positive. She was diagnosed with Mild COVID-19 Infection and was given antiviral therapy Favipiravir, supported treatment, continued her SLE medication, and performed a self-isolation. The patient missed her schedule to go to the polyclinic until the time she arrived at the Emergency Department. Below is the table of summaries of the symptoms and laboratory results before and after being diagnosed with COVID-19 infection.

At the time she arrived at the Emergency Department, her blood pressure was 160/100 mmHg, her axillary temperature was 39 degrees, and her respiratory rate was 24 times per minute. From the physical examination, she looked anaemic and pallor, from the chest examination we found a muffled heart sound. Shifting dullness on the abdomen also was found and swollen on both of her upper and lower extremities. She also complained of pain in both of her ankles, wrists, and knee joints. A chest X-Ray and emergency ultrasonography were

performed, and we found cardiomegaly with no sign of pneumonia (Figure 1), pericardial effusion, and ascites (Figure 2).

Initial laboratory studies showed hypochromic microcytic anaemia (7.2 gr/dl, normal range (NR): 11-16 gr/dL), leukopenia (2.900/µL, NR: 5.000-10.000/µL) thrombocytopenia (133.000/µL, NR: 150.000-450.000/µL), hyperazotemia (Urea 214 mg/dl, NR: 6-24 mg/dL), creatinine 4.6 mg/dl (NR: 0.6-1.0 mg/dL), hypoalbuminemia (1.7 g/dL, NR: 3.5-5.5 g/dL), and severe proteinuria 3+ from the urine dipstick, microhaematuria.

The patient was diagnosed with Severe SLE Flare probably induced by COVID-19 infection and planned to be treated with a high dose of methylprednisolone for 3 days and continued with cyclophosphamide every two weeks. During the treatment, her kidney function did not show any improvement and the patient was planned to refer for haemodialysis, but she refused due to financial issues and decided to refuse all medical advice. The patient was rehospitalisation to the hospital due to relapse ascites and shortness of breath, but she still refused to be referred for haemodialysis and she died on the fifth month after being diagnosed with SLE and kidney involvement.

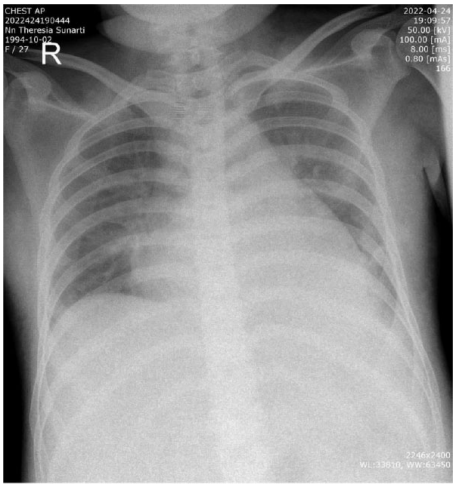

Figure 1. Chest X Rays showed Cardiomegaly.

SEARCH STRATEGY

According to the published guidance on narrative review[5] a literature search from December 2019 (when SARS-CoV-2 was first reported) to September 2, 2022, in PubMed and Scopus with MeSH search terms "Systemic Lupus Erythematosus", "Flare", "Severe Acute Respiratory Syndrome Coronavirus 2", "Infection" was performed. Case reports of a patient with SARS-CoV-2 infection and diagnosis of SLE with activity triggered (defined as a measurable increase in disease activity in one or more organs or systems involving new or worse clinical signs and symptoms and/or laboratory measurements) were included. Publications without restriction in the language were eligible for inclusion. We extracted the following data from the selected papers: age, sex, the clinical manifestation of SLE before and after contracting COVID-19, the onset of flare symptoms, laboratory test (including immunology profile), complication, and outcome.

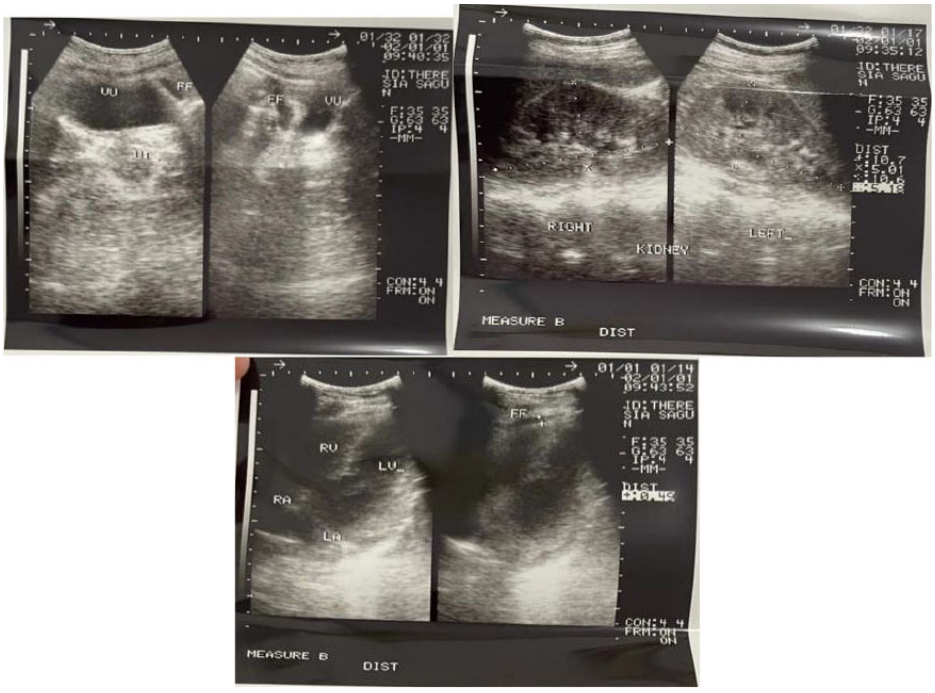

Figure 2. Emergency ultrasonography showed pericardial effusion and ascites.

DISCUSSION

A total of 72 potentially relevant citations were identified. After removing the duplicate citations, the title and abstracts of 61 articles were evaluated and 11 relevant articles were reviewed in detail. Nine case reports were found to be relevant for this research (Figure 3).[3,6–13]

In the present report, a total of ten cases were included for this review. Of the 10 patients (including ours) eight (80%) were women and two (20%) were men with a median age of 42 (range 22 – 68).

Diagnosing SLE Flare during COVID-19 infection or after the infection is resolved can be challenging because both SLE and COVID-19 share some of the symptoms and laboratory findings. The spectrum for COVID-19 manifestation can be divided into asymptomatic, mild, moderate, and severe according to the Indonesian guidelines.[14] A review article tried to conclude the characteristic of constitutional symptoms for COVID-19 with the resulting fever (78%), fatigue (31%), myalgia (17%), and arthralgia (11%)[15] and these findings are related SLE's constitutional.[16] SLE clinical manifestations after COVID-19 infection were Lupus Cerebritis[3]. Rowell Syndrome Like[12], Acute Lupus Pneumonitis overlapping with COVID-19 Interstitial Pneumonia[10], Diffuse Bilateral Lymphadenopathy[13], and Lupus Nephritis stage IV (in our case). From the immunology profiles a positive lupus anticoagulant found in 3 cases[6,8,13], hypocomplementemia[3,7,10,11,13], a positive or increased anti dsDNA.[3,7,10,11,13] There were other immunology profiles like anti-cardiolipin, anti b2 glycoprotein, anti-SS-A native, anti-SSA (Ro-52 recombinant), anti-ribosomal P antibodies, Anti Ro60, and anti-SM also positive (8,13,14). In our case, we did not repeat the immunology profile because of a lack of facilities and

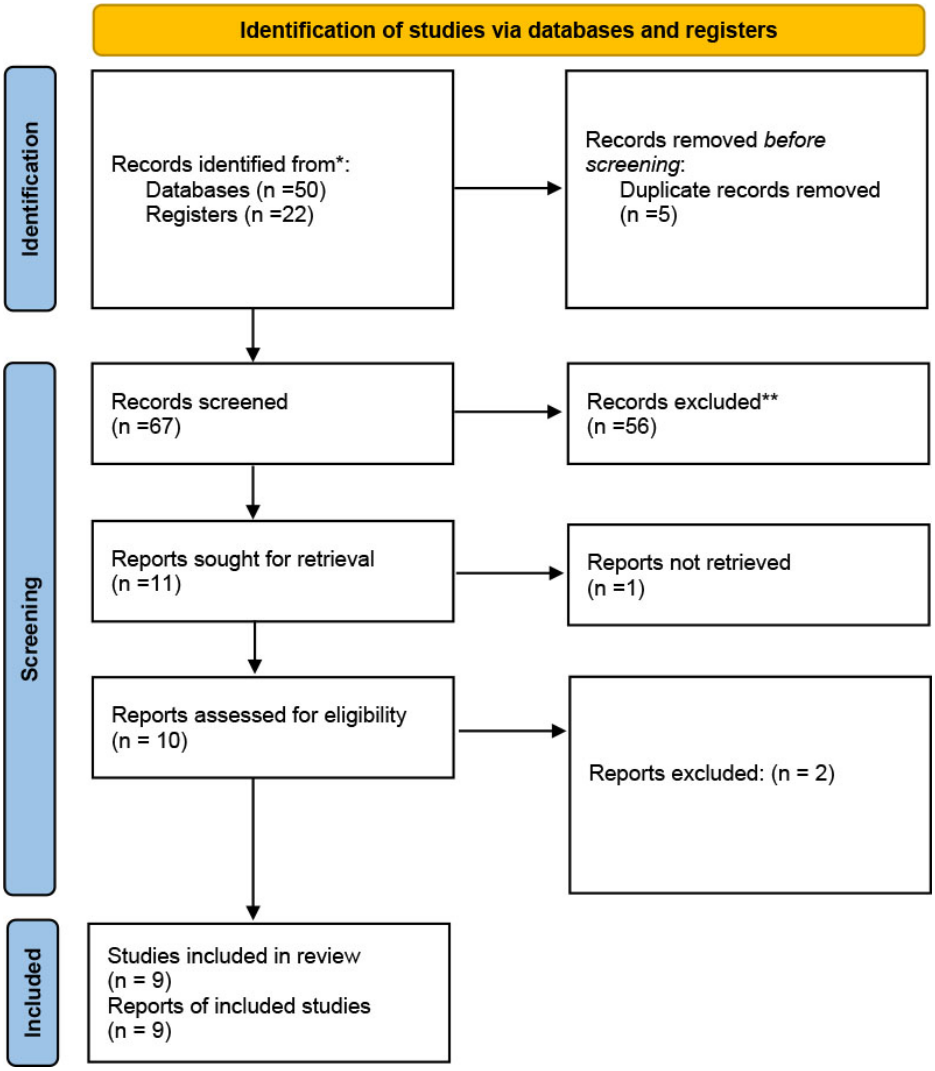

Figure 3. Flowchart of study selection.

funding.

Our findings on the patient with COVID-19 infection occurred before her flare-up condition and fever symptom was found in both conditions. Pulmonary manifestation can be found both in SLE (50-70%)[17] as moderate to severe COVID-19 infection even though the incidence of acute Lupus Pneumonitis was uncommon approximately 1-2% of SLE patients.[18] In our review, there were two cases with the complication of ARDS, one of them was an overlapping condition between acute lupus pneumonitis and COVID-19 Interstitial Pneumonia. However, in our patient, we did not find any sign of abnormality in the lungs from the Chest X-Ray.

The most frequent haematology abnormalities found in COVID-19 and SLE were lymphopenia and thrombocytopenia.[19] However, in COVID-19 infection the level of thrombocyte not too low and this can be used to differentiate between of these two conditions. In our case lymphopenia and mild thrombocytopenia were found and it was resolved during the remission condition but when she is in a flare-up it was found again. Another abnormal haematology finding in our case was hypochromic microcytic anaemia suspected a haemolytic anaemia from the peripheral blood smear show the target cell. Anaemia isn't a common finding in COVID-19 infection unless there is a sign of bleeding that was caused by the thrombosis or side effect of steroid usage as treatment in severe COVID-19 infection.[19] The mechanism of SARS-CoV-2 induced thrombocytopenia was still unclear. A hypothesis explained that there's a direct attack of the infection to the hematopoietic stem/progenitor cells through a mechanism of autoantibodies and immune complexes which is genetically more susceptible in SLE patients therefore we suspected that this is the pathophysiology of severe thrombocytopenia in SLE Flare in Covid patient.[20–22]

The renal disturbance was common in SLE conditions with the complication of AKI in flare-up to end stage renal disease when the disease has severely progress.[23] In COVID-19 infection the mechanism of dysregulation of the kidney was multifactorial. A direct viral effect caused a collapsing glomerulopathy, endothelial damage, coagulopathy, complement activation, and inflammation while indirect effects were more to be a pre-renal condition such as fever or sepsis, hypovolemic, mechanical ventilation, etc.[24] In our review, we found that lupus nephritis flares were found in two cases but the first one occurred along with the COVID Infection resulting with persistent nephrotic stage proteinuria, and in our patient which occurred 4 weeks after contracting COVID-19 infection. How COVID-19 infection triggered or increased the severity of lupus nephritis is still a doubtful mechanism. The possible mechanism is that viral infections such as Epstein Barr Virus or Influenza may trigger SLE or flare through molecular mimicry and epitope spreading which has been considered for the last 40 years.[25] A hypothesis can be made through the mechanism of direct viral effect on the kidney and the indirect viral effect through the immunologic effect of cytokine storm combined with the dysregulation of the kidney in an SLE patient triggered the new onset of lupus nephritis or worsening.[26]

The complications from out review show two cases with AKI[11] in our case , acute Respiratory Distress Syndrome[7,10], bleeding symptoms et: gingival bleeding[6], haemoptysis[9], Intracranial Hemorrhage[8], severe skin lesions[3,12], and altered mental status[3] with the outcome eight patients (80%) were survived and two patients including our case (20%) were passed away.

Managing a patient with SLE flare with COVID-19 infection also becomes a tricky

situation. Before the RECOVERY trial glucocorticoid are contraindicated because they will cause a delay in viral clearance but on the other hand a high-dose steroid IV is needed in the condition of SLE Flare. Other immunosuppressant drugs also must be postponed in COVID-19 Infection because they will disrupt the mechanism to eliminate the virus.[6–11] Fortunately, in our case, the patient has resolved from the COVID-19 infection and a high-dose steroid IV followed by other immunosuppressant drugs to treat lupus nephritis can be given.

This review has several limitations owing to its retrospective nature, limited size, and heterogeneity. Overall, the mechanism of SARS Cov-2 infection can trigger a flare condition in an inactive SLE still cannot be fully understood and more research is highly needed. However, our review has given some meaningful information that COVID-19 infection can trigger an SLE flare which can happen along with the infection or after the infection is resolved, the clinical manifestation, laboratory findings, and the outcome.

CONCLUSION

The COVID-19 pandemic is a devastating situation all over the world. SLE patient has already been in a susceptible condition as the disease progressed and continued having immunosuppressant therapy also one of the risk factors. A Flare condition can be happened during the COVID-19 Infection or after the infection is resolved. For every SLE patient, close monitoring, high adherence to the therapy, and the health protocol are needed.

DISCLOSURES

None.

FUNDING

None.

CONFLICT OF INTEREST

The authors declared that there are no conflicts of interests.

AUTHOR CONTRIBUTION

Both authors contributed equally to the study.

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Table 1. **Major clinical manifestations, findings of complementary studies, and outcomes in patients with SLE triggered by SARS-CoV-2 infection.**

First Author	Sex	Age	Clinical Manifestation		Onset of Flare	Specific Laboratory Finding	Immunology profile	Complication	Outcome
			Before COVID	After COVID					
Kondo[6]	F	58	Hematology: Mild thrombocytopenia	Hematology: Severe thrombocytopenia	Ongoing COVID-19 infection	Lymphopenia (650/µL Thrombocytopenia (5000/ µL)	Lupus anticoagulant (+)	Gingival bleeding	Survive
Kichloo[7]	F	22	Pulmonary: Chronic Lupus pneumonitis	Pulmonary: Acute Lupus pneumonitis Overlapping with COVID-19 interstitial pneumonia	Ongoing COVID-19 infection	Lymphopenia ↑ D-dimer ↑ LDH	↑anti-dsDNA (19 IU/mL) ↓C3 (84 mg/ dL) ↓C4 (9 mg/dL)	ARDS	Survive
Raghavan[8]	M	62	Hematology: Mild thrombocytopenia	Hematology: Evans syndrome	Ongoing COVID-19 infection	Anemia (9.1 g/dL Thrombocytopenia (2000/µL) Indirect hyperbilirubinemia (2.9 mg/dL) ↑ PT (15.4 s) ↑ aPTT (41.8 s) ↑ fibrinogen (561 mg/ dL) ↑ D-dimer ↑ CRP (11.9 mg/dL)	Lupus anticoagulant (+) anticardiolipin (+) anti-beta2-glycoprotein (+)	ICH	Death
Hayden[9]	F	51	Hematology: Mild thrombocytopenia	Hematology: Severe thrombocytopenia	Ongoing COVID-19 infection	Thrombocytopenia (< 10,000/µL) ↑ INR (1.94	ND	Hemoptysis	Survive

First Author	Sex	Age	Clinical Manifestation		Onset of Flare	Specific Laboratory Finding	Immunology profile	Complication	Outcome
			Before COVID	After COVID					
Altharty[10]	F	28	Renal Disease: Lupus Nephritis stage IV on regular HD	Pulmonary: Acute Lupus Pneumonitis	Ongoing COVID-19 infection	Lymphopenia (590/µL) ↑ CRP (354 mg/L) ↑ D-dimer (1.9 mcg/ml) ↑ DHL (737 U/L) ↑ ferritin (1126 ng/ml)	↑ anti-dsDNA (22 U/ml) ↓ C3 (64 mg/dl) ↓ C4 (6 mg/dL	ARDS	Survive
Shamsi[11]	M	30	Renal Disease: Mild Lupus Nephritis	Renal Disease: Lupus Nephritis stage III	Ongoing COVID-19 infection	Urine PCI (0.63 g/mmol ↑) Creatinine (3,63 mg/dL) Urea (150,15 mg/dL) CRP (33.8 mg/dL)	ANA 1:320 anti-dsDNA positive ↓ C3 (30 mg/dL) ↓ C4 (5 mg/dL)	AKI	Survive
Khalid[3]	F	29	Cutaneous Lupus	Neuropsychiatric lupus Lupus Cerebritis	Three weeks after the COVID-19 infection	Thrombocytopenia (135000/µL) Creatinine (1.4 mg/dL)	↑ anti-dsDNA (55 IU/mL) ↓ C3 (88 mg/dL)	Severe skin lesions	Survive
Drenovska[12]	F	67	Cutaneous Lupus Photosensitivity Discoid lesions	Cutaneous Lupus Erythematosus Sub-acute cutaneous lupus presenting as Rowell syndrome	Two weeks after the COVID-19 infection	Normal	anti-SS-A native (60 kDa) (Ro/SSA), 34 U/ml (<10 U/ml) anti-SSA (Ro-52 recombinant), 44 U/ml (<10 U/ml) anti-ribosomal P antibodies, 63 U/ml (<10 U/ml),	Increased skin lesions	Survive

First Author	Sex	Age	Clinical Manifestation		Onset of Flare	Specific Laboratory Finding	Immunology profile	Complication	Outcome
			Before COVID	After COVID					
Karsulovic[13]	F	25	Arthritis Non-scarring alopecia Mild thrombocytopenia	Lupus headache Axillary and cervical bilateral lymphadenopathy	Four weeks after the COVID-19 infection	CRP ×2 times normal value ESR: 46 mm/h	Lupus anticoagulant (+) C4 (12 mg/dL)	None	Survive
Karsulovic[14]	F	68	Arthritis Non-scaring alopecia Hypercomplentemia	Asthenia Multiple lymphadenopathy axillar and cervical	Four weeks after the COVID-19 infection	CRP ×5 times normal value ESR: 66 mm/h	Anti-dsDNA: Positive Anti-Ro60: Positive Anti-Ro52: Positive Anti-Sm: Positive	None	Survive
Current case	F	27	Hemolytic Anemia Arthritis Non-scarring alopecia	Lupus nephritis stage 4 Anemia Thrombocytopenia	Four weeks after the COVID-19 infection	Anemia (7.2 mg/dL) Thrombocytopenia (133000/µL) Proteinuria 3+ Urea (214 mg/dL) Creatinine (4.6 mg/dL) Albumin (1.7 mg/dL)	N/A	AKI	Death