CASE REPORT

Lupus Flares in COVID-19 Patients: A Case Report

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ABSTRACT

Coronavirus disease 2019 (COVID-19) is closely related to Systemic Lupus Erythematosus (SLE). COVID-19 can cause SLE with the formation of autoantibodies. SLE patients have dysregulation and weakness in the immune system, so they are vulnerable to COVID-19 infection. A 29-year-old man came to the Emergency Unit with fever and weakness complaints. Follow-up examination revealed thrombocytopenia, increased neutrophil to lymphocyte ratio, and elevated hsCRP. A chest X-ray showed pneumonia, PCR examination showed a positive result for the SARS-CoV-2 virus, the ANA IF level was 1/320, and the ANA profile test was positive for PCNA antibodies. Diagnosis and treatment of SLE patients with COVID-19 coinfection become problematic because COVID-19 is a hyperinflammatory disease like SLE. Immunosuppressant drugs in SLE will cause weakness of the immune system and broader spread of the virus, so it needs strict evaluation and proper timing of drug administration.

Keywords: COVID-19, Lupus Flare, SLE

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INTRODUCTION

COVID-19 is a global health problem that affects many sectors of life. On March 6, 2021, in Indonesia, there were 1.373.836 cases of COVID-19 with 147.172 active patients, and the number of deaths reached 37.154 people. There were 157.812 cases of COVID-19 (11.4% of the total national cases), with 6.089 active cases and 9.939 people dying in Central Java alone (1). There is concern about the risk of infection and complications of SARS-CoV-2 in SLE patients. SLE patients are at higher risk for COVID-19 due to immunosuppressant treatment. Immunosuppressants can suppress the abnormal immune response in COVID-19, which is the cause of complications of severe diseases such as acute respiratory distress syndrome, so it needs a strict evaluation and the right time of drug administration (2). There is still controversy over the administration of immunosuppressants and corticosteroids in SLE patients with COVID-19 (3).

Flares in SLE and COVID-19 are rare, so we would like to present a case of Lupus Flare in COVID-19 patients. This report aims to describe Lupus Flare in COVID-19 patients, also how to treat and manage these patients for excellent care.

CASE REPORT

We report a rare case of a 29-year-old man with COVID-19 and Lupus. Informed consent and ethical consent for publication were obtained from the patients. The patient came to the UNS (Universitas Sebelas Maret) Hospital emergency unit with the main complaints of fever and weakness one week before admission. Another complaint felt by the patient was joint pain in the hands and hair loss which was felt one week before admission to the hospital. In Table I, laboratory tests showed thrombocytopenia ($11x10^6$/µL), lymphocytopenia (940/µL), an increase in the Neutrophil to Lymphocyte Ratio (6.98), an increase in hsCRP (high sensitive C reactive protein) 10.26 mg/dL. The chest X-ray in Figure 1 shows pneumonia, the PCR (Polymerase Chain Reaction) examination is positive for the SARS-CoV-2 (Severe acute respiratory syndrome coronavirus-2), the level of the ANA IF (Anti Nuclear Antibody Indirect Immunofluorescence) test shows 1/ 320, and the ANA profile shows positive results for PCNA (Proliferating Cell Nuclear Antigen) antibodies. Treatment of patients using standard therapy favipiravire and intravenous methylprednisolone 62.5 mg per 12 hours. The patient's condition recovered after therapy, and after PCR evaluation, the SARS-CoV-2 virus became negative on the 7th day.

DISCUSSION

This patient was diagnosed with lupus according

Table I: laboratory examination in SLE patients with COVID-19

Laboratorium	Data	Normal range
Hemoglobine	14.6	12.1-17.6 g/dL
Leucochyte	4.890	4500-11000 per micro-litres
Thrombocyte	64000	150.000-450.000 per microlitres
Lymphocyte	1050	>1500 per microlitres
Neutrophyl to Lymphocyte ratio	3	
SGPT	70	8-40 U/L
SGOT	60	0-37 U/L
Creatinine	1.13	0.5-1.1 mg/dL
Ureum	26	10-45 mg/dL
IG M dengue	Negative	Negative
IG G dengue	Negative	Negative
ANA IF	1/320	Negative
ANA profile	Anti PCNA	Negative

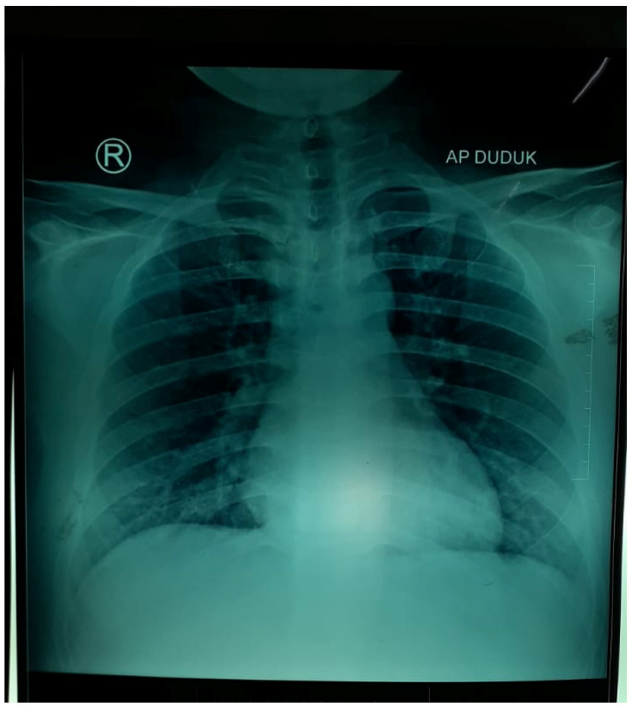

Figure 1: Chest X-ray shows bronchopneumonia with infiltrates in both lung fields. X-ray image of the lungs shows the heart is within normal limits and there is no enlargement. X-ray showing infiltrates in both lung fields.

to American College of Rheumatology criteria. The diagnostic variables included a positive ANA test, accompanied by joint pain, hair loss, low lymphocytes, and thrombocytopenia. This patient was diagnosed with COVID-19 because the PCR for Sars-CoV-2 was positive.

This patient has a low lymphocyte count. Low lymphocytes (lymphopenia) may occur in lupus and COVID-19 patients. Lymphopenia occurs in 90% of SLE patients. The main cause is not only due to disease activity but also due to infection and immunosuppressant drugs. In lupus, the occurrence of lymphopenia has several causes, such as antibodies to CD-8 T lymphocytes, cell apoptosis, decreased lymphocyte production, and excessive destruction of lymphocytes due to complement-mediated sequestration and T-lymphocyte cytolysis (4). Low lymphocytes are also common in COVID-19 patients. Lymphopenia occurs in 96% of COVID-19 patients. Lymphopenia in COVID-19 patients can be due to a cytokine storm. The degree of lymphopenia is related to the severity of the disease (5).

COVID-19 causes respiratory distress, an exaggerated inflammatory response, and a hypercoagulable state through activation of the innate and adaptive immune systems. A wide variety of cytokines are involved in the pathogenesis of severe COVID-19, and immunosuppressant drugs may be a potential therapy (2). In confirmed COVID-19 patients with newly diagnosed SLE, NSAID (Nonsteroidal Anti-Inflammatory Drugs) therapy can be given as part of therapy for SLE. However, if the patient has severe COVID-19 symptoms with severe respiratory, cardiac, gastrointestinal, and renal manifestations, NSAID administration should be discontinued because of the poor prognosis, and NSAID administration may worsen the symptoms. Corticosteroids can be given to patients with confirmed COVID-19 who are asymptomatic or with mild to moderate infection symptoms with the lowest effective dose according to SLE disease activity. In patients with severe COVID-19 symptoms, corticosteroid doses are determined according to clinical conditions on a case-by-case basis and the risk-benefit ratio for each patient. The conventional DMARD (Disease-modifying antirheumatic drugs) that can be given is hydroxychloroquine. Other conventional DMARDs can be used as a treatment after the patient recovers from COVID-19 (2). This patient had mild symptoms of COVID-19 because of only pneumonia and fever without a decrease in oxygen saturation, so we decided to give a high dose of corticosteroid 125 mg per day. The therapy results showed rapid clinical improvement with joint pain loss and a negative PCR Sars-Cov-2.

CONCLUSION

COVID-19 and SLE have similar symptoms and complex management. Understanding the details on a case-by-case basis is essential to achieve proper management and a good patient outcome.

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REFERENCES

1. Gugus Penanganan COVID-19. Peta Sebaran [Internet]. Peta Sebaran. 2020[cited 2020 april 29].

Available from: https://covid19.go.id/peta-sebaran.
2. Perhimpunan Reumatologi Indonesia. Penatalaksanaan Penyakit Rematik-Autoimun pada Masa Pandemi COVID-19. Jakarta: PB IRA; 2019.
3. Viner RM, Whittaker E. Kawasaki-like disease: emerging complication during the COVID-19 pandemic. Lancet. 2020;395(10239):1741–3.
4. Martin M, Guffroy A, Argemi X, Martin T. Systemic lupus erythematosus and lymphopenia: Clinical and pathophysiological features. Rev Med Interne. 2017 Sep;38(9):603-613.
5. Liu J, Li H, Luo M, Liu J, Wu L, Lin X, et al. Lymphopenia predicted illness severity and recovery in patients with COVID-19: A single-center, retrospective study. PLoS One. 2020 Nov 18;15(11):e0241659.