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Corona Virus Disease 2019 (COVID-19) Infection in a Patient with Systemic Lupus Eritematosus (SLE)

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Abstract: ***Corona Virus Disease 2019 or called COVID-19 is an infectious disease caused by infection from Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-CoV-2), COVID-19 is an extraordinary threat that has occurred globally since 2019. Since the COVID-19 pandemic, there has been concern about the risk SARS-CoV-2 infection and complications in patients with systemic autoimmune rheumatic diseases, especially is SLE. Currently published studies regarding the risk of SLE patients to become infected with COVID-19 give mixed results. We report a case of a woman with a confirmed diagnosis of severe COVID-19 with a newly diagnosed SLE. The possibility of spreading COVID-19 infection in SLE patients is increased due to the overexpression of ACE-2 in peripheral blood mononuclear cells. Based on the data obtained, it is presumed that the recurrence of SLE will increase the possibility of SARS-CoV-2 infection due to increased oxidative stress and DNA demethylation of ACE-2.***

Keywords: COVID-19, SLE

1.Introduction

Corona Virus Disease 2019 or often called COVID-19 is an infectious disease caused by infection from SARS-CoV-2, COVID-19 is an extraordinary threat that occurs globally, this disease can attack anyone, without exception. SARS-CoV-2 is a new type of coronavirus that has never been previously identified in humans.[1, 2] There are at least two types of coronavirus that are known to cause diseases that can cause severe symptoms such as Middle East Respiratory Syndrome (MERS) and Severe Acute Respiratory Syndrome (SARS). This disease can be more dangerous if it is suffered by the elderly group and those who have a congenital disease (comorbid). The first cases were reported in Wuhan, Hubei Province, China, from December 2019 to February 2020, cases jumped sharply to 14, 840 and at the same time the spread of cases was reported in 25 countries with 60, 329 cases. In Indonesia, according to the report of the Gugus Tugas Percepatan Penanganan COVID-19, as of July 8, 2020, there were 68, 079 positive cases and 3, 359 deaths.[2, 3] Most patients infected with SARS-CoV-2 show symptoms of the respiratory system such as fever, coughing, sneezing and shortness of breath. Based on data from 55, 924 cases, the most common symptoms were fever, dry cough and fatigue. Other symptoms that can be found are productive cough, shortness of breath, sore throat, headache, myalgia/arthralgia, chills, nausea/vomiting, nasal congestion, diarrhea, abdominal pain, hemoptysis and conjunctival congestion.[4]

Systemic Lupus Erythematosus (SLE) is a multisystem autoimmune disease in which organs, tissues and cells are damaged mediated by tissue-binding autoantibodies and immune complexes. The highest incidence and prevalence of SLE were found in North America at 23.2/100, 000 population/year and 241/100, 000 population. Based on gender, SLE was more common in women than men (2: 1 to 15: 1 ratio). Based on ethnicity, the incidence of SLE among black population is 31.9/100, 000 population/year, Asian 0.9-4.1/100, 000 population/year and Caucasian 0.3-4.8/100, 000 population/year. The clinical picture of SLE can change, both in terms of disease activity and organ involvement. The immunopathogenesis of SLE is complex and associated with a variety of clinical features. The management of SLE requires a holistic approach which includes education, rehabilitation programs as well as non-pharmacological and pharmacological therapies, the target of SLE management is to achieve remission and prevent recurrence.[5]

Since the pandemic, there have been concerns about the risk of SARS-CoV-2 infection and complications in patients with systemic autoimmune rheumatic diseases, especially SLE. On one hand, SLE patients have a higher risk of infection because they are in a state of decreased immunity caused by immunosuppressant treatment, on the other hand, immunosuppressants can suppress the abnormal immune response in COVID-19 which is responsible for the occurrence of more severe complications of the disease. However, to date, there have been very few reports of an increase in the number of autoimmune rheumatic patients, especially SLE with COVID-19, or an increase in the morbidity and mortality of SLE patients with COVID-19. This case report aims to provide information about the relationship and management of COVID-19 in patients with SLE.

2.Cases

The female patient Mrs. S aged 50 years, came to the ER on September 29, 2021, with the main complaint is shortness of breath since two weeks ago and has been getting worse since three days before being admitted to the hospital. The shortness of breath is said to not improve with a change in position and interfere with the patient's activities. Besides shortness of breath, the patient also complained of coughing since two weeks ago, coughing

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up phlegm with yellow phlegm. The patient also complained of feeling weak, nauseated and vomiting, vomiting three times mixed with food, without blood. There was also hair loss, intermittent fever, recurrent ulcer at mouth and weight loss of ten kg over the past two years.

The patient had previously been hospitalized about one year ago with a diagnosis of urinary tract infection, hypertension, cholelithiasis, acute on chronic kidney disease and autoimmune rheumatoid arthritis. However, it has been a year since the patient had no control over his illness for fear of going to the hospital due to the COVID-19 pandemic.

On physical examination, the general condition was severe pain, compos mentis consciousness, GCS E4V5M6 with blood pressure 180/100 mmHg, pulse rate 108 times per minute, respiratory rate 24 times per minute, temperature 36.6 C, oxygen saturation 90% on room air, with a VAS of 3/10 of the lower limb area. On examination of the head and neck, the eyes were not anemic, the pupillary reflex was positive isocor, there was no enlargement of the cervical lymph nodes. On examination of the thorax, on auscultation, vesicular breath sounds were found in both lung fields, there were rhonchi sounds in both lung bases and no wheezing was found. On examination of the abdomen, bowel sounds were normal, the liver and spleen were not palpable, but tenderness was found in the epigastric region. On examination of the extremities, all four extremities were warm and there was no edema.

The results of complete blood count showed leukocytosis (Wbc 13, 200), Hb 10.8 g/dL, Hct 34% and platelets 252, 000 cells/mm^3. Examination of kidney function increased (ureum 43 mg/dL and creatinine 2.92 mg/dL). On urinalysis examination found urine looks cloudy yellow, protein +3. The chest X-ray on September 27, 2021 showed cardiomegaly (RVH) with alveolar pulmonary edema and bilateral pleural effusions, on October 6, 2021, minimal lesions were seen with left interstitial pneumonia and cardiomegaly. On the ECG examination, a sinus rhythm was found. Then, the patient underwent a SARS-CoV2 PCR swab examination with a positive result, D-Dimer examination was performed with the results were 1539.07 ng/ml and the results of the ANA (IF) >1: 1000.

This patient was diagnosed with severe COVID-19 and Moderate SLE (MEX-SLEDAI score 6). While being treated, the patient was given O2 nasal cannula 4-5 lpm, asering infusion 12 tpm, levofloxacin 1x750mg (iv), cefuroxim 3x1g (iv), pantoporazole 2x40mg (iv), ondansetron 3x4mg (iv), methylprednisolone 1x40 mg (iv), furosemide 2x20mg (iv), favipiravir 2x1600mg (po) day-1 followed by 2x600mg (po) day 2 to day 5, goldtrion 1x1 (po), heparin 2x5000 iu (sc), N-ace 2x600mg (io), candesartan 1x16mg (po), spironolactone 1x50mg (po), vitamin D 2x400mg (po), azathioprine 1x50mg (po), hydroxychloroquine 1x200mg (po).

3.Discussion

COVID-19 is a respiratory infection caused by infection of SARS-CoV-2. The pathogenesis of COVID-19 is the entry of the virus into host tissues through a special receptor, the human angiotensin converting enzyme 2 (ACE2) receptor. Viral replication occurs primarily in the mucosal epithelium of the upper respiratory tract, subsequently spreads to the lower respiratory tract, the gastrointestinal tract and mucosa where further multiplication is thought to occur resulting in mild viremia. Multiplication of the virus can also occur in many organs such as the lungs, heart, kidneys, stomach and bladder. The mechanism of viral invasion into host cells consists of 5 stages, namely attachment, penetration, biosynthesis, maturation and release. Binding of the virus to the ACE2 receptor (adherence) results in mediating viral entry into host tissues via the plasma membrane or by endocytosis (penetration) via protease release. After entry, the viral contents are released inside the host cell and the viral RNA enters the host cell nucleus for replication where the viral mRNA will produce viral proteins (biosynthesis). The viral protein will then create new viral particles (maturation) which are then released into the host tissue from the nucleus. The entry of the virus into the host cell, triggers a host immune response, initially an innate immune response, but an adaptive immune response can be stimulated as the infection progresses. ACE2 receptors are predominantly expressed on the apical side of pulmonary epithelial cells in the alveolar space and therefore the first immune response is stimulated by three major components for innate immunity in the airway: epithelial cells, dendritic cells (DCs) and alveolar macrophages. DCs are found mainly beneath the epithelium, while macrophages are located on the apical side of the epithelium. Thus, DCs and macrophages function as innate immune cells to fight the virus until adaptive immunity takes effect. DCs and macrophages can phagocytize cells infected with SARS-CoV-2, then present viral particles to antigen presenting cells (APCs). The APCs then migrate to the lymphnodes to present viral antigens to T cells which initiate a T cell response with CD4+ and CD8+ T cells playing the most important roles: CD4+ T cells activate B cells to increase the production of virus-specific antibodies, whereas CD8+ T cells can kill virus-infected cells. In severe cases of COVID-19, there is an increased level of activated proinflammatory immune cells such as cytokines, including interleukin (IL-6), IL-10, granulocyte-colony stimulating factor (G-CSF), monocyte chemoattractant protein 1 (MCP1), macrophage inflammatory protein (MIP) 1α, tumor necrosis factor (TNF)-α and this condition is called Cytokine Storm (cytokine storm) which can result in an uncontrolled (excessive) immune response that can result in lung tissue damage, impaired function and death. decreased lung capacity. Cytokine Storm is an uncontrolled systemic inflammatory response that occurs due to the release of pro-inflammatory cytokines and chemokines by immune effector cells resulting in an exaggerated inflammatory immune response that contributes to acute respiratory distress syndrome (ARDS), multiple organ failure and ultimately death in severe cases of SARS-CoV-2 infection. In addition, elevated levels of these proinflammatory cytokines can cause shock and tissue damage in the heart, liver, kidneys, as well as respiratory or multiple organ failure. In the lungs there is extensive damage, which is characterized by

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massive infiltration of neutrophils and macrophages, diffuse alveolar damage with the formation of hyaline membranes and thickening of the alveolar walls caused by high levels of these proinflammatory cytokines. An autopsy of the deceased patient showed lymph node necrosis and splenic atrophy, damage mediated by the immune system.[1, 6] The diagnosis of COVID-19 is confirmed by anamnesis, physical examination and supporting examinations. The history, especially a description of the travel history or history of close contact with confirmed cases or working in health facilities that treat patients with COVID-19 infection or being in the same house or environment with confirmed COVID-19 patients accompanied by clinical symptoms and comorbidities. Clinical symptoms vary depending on the degree of disease but the main symptoms are fever, cough, myalgia, dyspnoea, headache, diarrhea, nausea and abdominal pain. The most common symptoms are fever (98%), cough and myalgia. Other investigations according to the degree of morbidity. In pneumonia, a chest x-ray is performed, followed by a computed tomography scan (CT scan) of the thorax with contrast. Chest radiography of pneumonia caused by COVID-19 infection ranging from normal to ground glass opacity, consolidation. Chest CT scan can be done to see more details of abnormalities, such as ground glass opacity, consolidation, pleural effusion and other pneumonia features. Examination of procalcitonin (PCT) showed normal results, except when a bacterial infection is suspected, the PCT will increase. Other examinations were carried out to see comorbidities and evaluate possible complications of pneumonia, namely kidney function, liver function, albumin, arterial blood gas, electrolytes, blood sugar, bacterial cultures and sensitivity tests to see possible causes of bacteria or if a double infection with bacterial infection is suspected. A definite diagnosis or confirmed case is determined based on the results of the SARS-CoV-2 RNA extraction. COVID-19 uses reverse transcription polymerase chain reaction (RT-PCR) to extract 2 SARS-CoV-2 genes. An example of a test that can be used is a sample in the form of a throat swab. Nasopharyngeal swabs are good for evaluation of influenza but for other coronaviruses nasopharyngeal swabs are taken using swabs from dacron or rayon instead of cotton.[7]

Systemic Lupus Erythematosus (SLE) is a complex autoimmune disease that attacks various body systems. The pathogenesis of SLE is influenced by genetic and environmental factors (such as UV light). The course of SLE begins with a preclinical phase, often mimicking other diseases. Formation and deposition of immune complexes that are increasingly developing cause the course of SLE to enter a more advanced stage with increasingly diverse and multiorgan clinical manifestations. The final stages of the course of SLE are generally caused by long-term complications of SLE that cause organ damage. The main characteristic of SLE is characterized by the emergence of an immune response to endogenous nuclear antigens. Damage to various organs in SLE occurs due to the formation and deposition of autoantibodies and immune complexes. Hyperactive B cells originate from the stimulation of T cells and antigens which will increase the production of antibodies against antigens exposed to the surface of apoptotic cells. The antigen causes T cell and B cell stimulation which contributes to the incomplete clearance of apoptotic cells. During the process of apoptosis, there are pieces of cellular material that form on the surface of the dead cells. Normally, the antigen is not present on the cell surface, but in SLE, the antigen is found on the cell surface. All pathways in SLE lead to endogenous nucleic acid-mediated interferon (IFN-α) production. Increased production of autoantigens during apoptosis (spontaneous or induced by UV light), decreased clearance and deregulation play important roles in the initiation of autoimmune responses. Nucleosomes contain harmful endogenous ligands that can bind to pathogen-associated molecular pattern receptors, combining with apoptotic blebs which then activate dendritic cells, B cells then produce IFN and autoantibodies. Cell surface receptors such as BCR and FcRIIa facilitate endocytosis of immune complexes or nucleic acid-containing materials and bind to endosomal receptors of the innate immune system such as TLRs. In SLE disease in the early stages, autoantibodies and immune complexes are not yet formed, there is a process of releasing antimicrobial peptides by damaged tissues such as LL37 and neutrophil extracellular traps. The two products released may be bound to nucleic acids so that they can inhibit their degradation and facilitate their endocytosis and stimulation of TLR-7/9 in the plasmacytoid of dendritic cells. Increased levels of endogenous nucleic acids associated with apoptosis stimulate IFN production and autoimmunity with the breakdown of self-tolerance through activation and maturation of conventional dendritic (myeloid) cells. Immature dendritic cells cause tolerance whereas activated mature dendritic cells cause autoreactivity. The production of autoantibodies by B cells in SLE is controlled by the availability of endogenous antigens and is highly dependent on T cell assistance mediated by cell surface interactions (CD40L/CD40) and cytokines (IL21). Immune complexes containing chromatin stimulate B cells via BCR/TLR cross-linking. SLE disease develops when T lymphocytes are activated by antigens presented by Antigen Presenting Cells (APC) through the Major Histocompatibility Complex (MHC), then these activated T lymphocytes will release cytokines, inflammation and stimulate B cells. Stimulation of B cells and production of immunoglobulin G autoantibodies (IgG) can cause tissue damage. T cells and B cells that are specific for autoantigens will interact and produce autoantibodies.[8, 9]

Systemic lupus erythematosus is a systemic disease, where the clinical manifestations of this disease involve almost all organ systems, including:

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Table 1: Manifestations of Systemic Lupus Erythematosus based on the organs involved.

Organs involved	Symptom
Constitutional manifestation	Fever, fatigue, lymphadenopathy, malaise, decreased appetite, weight loss.
Musculoskeletal	Joint pain, arthritis, muscle weakness, muscle pain, myositis, osteoporosis, tenosynovitis.
Skin and mucosa	Photosensitivity, malar rash (Butterfly rash), discoid rash, alopecia, oral ulcers, subacute cutaneous lupus erythematosus.
Kidney	Proteinuria, hematuria.
Neuropsychiatry	Seizures, chorea, cognitive impairment, peripheral neuropathy, headache, depression, mood disorders, psychosis, organic brain syndrome.
Lung	Pleurisy, alveolar haemorrhage, chronic pulmonary interstitial disease, airway obstruction
Cor	Pericarditis, myocarditis, cardiomyopathy, cardiac conduction disorders
Blood vessel	Vasculitis, Raynaud's phenomenon
Gastrointestinal	Nausea, vomiting, diarrhea, acute abdominal pain, anorexia, pharyngitis, esophagitis, peptic ulcer, malabsorption, ascites, peritonitis, pancreatitis, gastrointestinal bleeding, motility disorders, liver enzyme abnormalities, hepatomegaly, jaundice
Ocular	Dry eye, keratitis, keratoconjunctivitis sica, episcleritis, optic neuropathy, scleritis, uveitis, retinal vasculitis
Obstetric	Premature delivery, low birth weight, small neonates for gestational age, spontaneous abortion, stillbirth, preeclampsia
Endocrine	Vitamin D deficiency, hyperprolactinemia
Hematologic	Iron deficiency anemia, anemia of chronic disease, autoimmune hemolytic anemia, thrombocytopenia, leukopenia and lymphopenia.

The diagnosis of SLE is based on clinical symptoms and laboratory examination. The SLE classification criteria can help in establishing a diagnosis, there are several criteria and the latest currently is SLE classification criteria from ACR/EULAR 2019.[8, 10] Table 2. SLE ACR/EULAR 2019 classification criteria.

Entry criterion
Antinuclear antibodies (ANA) at a titer of ≥1:80 on HEp-2 cells or an equivalent positive test (ever)

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If absent, do not classify as SLE
If present, apply additive criteria

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Additive criteria
Do not count a criterion if there is a more likely explanation than SLE.
Occurrence of a criterion on at least one occasion is sufficient.
SLE classification requires at least one clinical criterion and ≥10 points.
Criteria need not occur simultaneously.
Within each domain, only the highest weighted criterion is counted toward the total score§.

Clinical domains and criteria	Weight	Immunology domains and criteria	Weight
Constitutional		***Antiphospholipid antibodies***	
Fever	2	Anti-cardiolipin antibodies OR	
Hematologic		Anti-β2GP1 antibodies OR	
Leukopenia	3	Lupus anticoagulant	2
Thrombocytopenia	4	***Complement proteins***	
Autoimmune hemolysis	4	Low C3 OR low C4	3
Neuropsychiatric		Low C3 AND low C4	4
Delirium	2	***SLE-specific antibodies***	
Psychosis	3	Anti-dsDNA antibody* OR	
Seizure	5	Anti-Smith antibody	6
Mucocutaneous			
Non-scarring alopecia	2		
Oral ulcers	2		
Subacute cutaneous OR discoid lupus	4		
Acute cutaneous lupus	6		
Serosal			
Pleural or pericardial effusion	5		
Acute pericarditis	6		
Musculoskeletal			
Joint involvement	6		
Renal			
Proteinuria >0.5g/24h	4		
Renal biopsy Class II or V lupus nephritis	8		
Renal biopsy Class III or IV lupus nephritis	10		

Total score:

↓

Classify as Systemic Lupus Erythematosus with a score of 10 or more if entry criterion fulfilled.

Management for SLE consists of non-pharmacological and pharmacological management. Non-pharmacological management are education about disease, healthy lifestyle, exercise according to ability, balanced nutrition, avoid smoking, avoid exposure to direct sunlight for those who are sensitive, routine control and regular drug

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consumption. Pharmacological management is based on the severity of disease activity.[10]

Table 3: Pharmacological management of SLE

SLE Ringan	SLE Sedang	SLE Berat
Terapi Awal		
• Prednison oral ≤20mg/day ~ 1-2 week or • Metilprednisolon injection 80-120mg IM/IA and • Hiroksiklorokuin ≤6, 5mg/kgbb/day or • Metrotreksat 7, 5-15mg/week or • OAINS	• Prednison ≤0, 5mg/kgbb/day with or without metilprednisolon injection ≤ 250mg IV/day ~ 3 day and • Azatioprin 1, 5-2, 0 mg/kgbb/day or • Metrotreksat 10-25mg/week • or • Mofetil mikofenolat 2-3g/day • or • Asam mikofen0lat 1, 44-2, 16 g/day or • Siklosporin ≤ 2, 0mg/kgbb/day and • Hiroksiklorokuin ≤6, 5mg/kgbb/day	• Prednison ≤0, 5mg/kgbb/day and • metilprednisolon injection 500-750mg IV/day ~ 3 day or • Prednisolon ≤0, 75-1mg/kgbb/day and • Azatioprin 2-3 mg/kgbb/day • or • Mofetil mikofenolat 2-3 g/day • or • Asam mikofenolat 1, 44-2, 16 g/day or • Siklosporin ≤ 2, 5 mg/kgbb/day or • Sikofosfamid IV and • Hiroksiklorokuin ≤6, 5mg/kgbb/day

In our case, a woman aged 50 years came with complaints of shortness of breath, cough. From the physical examination on auscultation of the thorax, vesicular breath sounds were found in both lung fields, then there were rhonchi sounds in both lung bases and no wheezing was found. On a chest X-ray on October 6, 2021, a minimal lesion was seen with left interstitial pneumonia and cardiomegaly (RVH). Furthermore, the SARS-CoV2 PCR swab examination with positive results and a D-Dimer examination was carried out and the results were 1539.07 ng/ml. So based on clinical symptoms, auscultation of the thorax, chest X-ray images and the positive results of the SARS-CoV2 PCR swab examination, the patient was diagnosed with COVID-19. With symptoms of pneumonia (shortness, cough) accompanied by 90% oxygen saturation in room air, the patient is included in the classification of COVID-19 with Severe Symptoms. The management of this patient is given antivirals to treat viral infections that form the basis of the disease, antibiotics, antimucolytics to treat cough complaints, as the management of heart disorders, diuretics are given, to treat pulmonary edema due to heart failure and anticoagulants are given to treat coagulopathy in patients. Furthermore, the patient was suspected of having SLE because the anamnesis showed symptoms of hair loss, recurrent stomatitis, pain throughout the body, especially in the joints, and the patient had a previous history of autoimmune rheumatoid arthritis. On investigation, the results of the patient's ANA (IF) >1: 1000, then the patient can be included in the SLE classification criteria from ACR/EULAR 2019. With the SLE classification from ACR/EULAR 2019, the patient scored 11 (pleural effusion, joint involvement), so that with the ACR/EULAR 2019 criteria met (1 clinical criteria and ≥10 points) the patient was diagnosed with SLE. The Indonesian Rheumatology Association generally distinguishes the determination of the degree of SLE activity based on clinical manifestations and the SLEDAI / MEX-SLEDAI score. The patient in this case had a MEX-SLEDAI score of 6 so it was included in the category of Moderate SLE. Patients were given therapeutic management according to the guidelines of the Indonesian Rheumatology Association (2019), namely steroids, immunosuppressants and antimalarials.

Since the COVID-19 pandemic, there have been concerns about the risk of SARS-CoV-2 infection and complications in patients with systemic autoimmune rheumatic diseases. Currently published studies regarding the risk of autoimmune survivors to become infected with COVID-19 have yielded mixed results. Some studies show an increased risk, but some studies show the opposite result. There are not many studies related to the risk of autoimmune disease patients to be infected with SARS-CoV2 to date, but recommendations from several rheumatology associations such as EULAR and the Global Rheumatology Alliance show that the case fatality rate is 9-15% higher than the general population. Furthermore, according to Sawalha H, the data obtained support that ACE-2 release is regulated by DNA methylation and that the methylation defect in lupus extends to the regulatory sequence of the ACE-2 gene, which may result in excess ACE2 secretion in lupus patients. It should be noted that excessive ACE2 secretion in other cells may facilitate viremia and organ damage in COVID 19 patients. It is possible that DNA methylation defects in lupus patients, exacerbated by oxidative stress resulting from SARS-CoV-2 infection, would further increase viral entry in lupus patients through epigenetic ACE-2 depression and increased ACE-2 secretion. Furthermore, epigenetic dysregulation may increase the risk and severity of SARS-CoV-2 infection in lupus patients. The possibility of spreading SARS-CoV-2 infection in lupus patients is increased due to the overexpression of ACE-2 in peripheral blood mononuclear cells. Based on the data obtained, it was hypothesized that lupus recurrence would increase the possibility of SARS-CoV-2 infection due to increased oxidative stress and DNA demethylation of ACE-2. Therefore, maintaining remission in lupus patients is very important during this pandemic. In conclusion, the data obtained indicate that lupus patients are more susceptible to SARS-CoV-2 infection; this is consistent with recent evidence showing that lupus patients are inherently more susceptible to viral infections.[1, 11]

Currently, several recommendations have been issued from the American College of Rheumatology (ACR) and the European League Against Rheumatism (EULAR) regarding the therapeutic management of rheumatic diseases in the COVID-19 pandemic. The ACR

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recommends that rheumatic patients with COVID-19, regardless of the severity of COVID-19, continue taking hydroxychloroquine or chloroquine, but that sulfasalazine, methotrexate, leflunomide, immunosuppressants, non-IL-6 biologics and JAK inhibitors should be stopped or postponed. The treatment of glucocorticoids can be continued at the lowest dose to control rheumatic disease. In contrast to EULAR, where EULAR recommends that if a rheumatic disease patient develops COVID-19 with mild symptoms, change the dose of DMARDs on a case-by-case basis and if the patient is treated with glucocorticoids, the therapy should be continued. Furthermore, if a rheumatic disease patient with COVID-19 gets worse, it can be treated according to expert opinion (such as a pulmonary specialist, internal medicine specialist or infectious disease consultant) adapted to local guidelines. Apart from ACR and EULAR, the Indonesian Rheumatology Association also provides several recommendations regarding the management of autoimmune-rheumatic diseases during the COVID-19 pandemic, in confirmed COVID-19 patients who have just been diagnosed with autoimmune-rheumatic diseases, then[1]:

- NSAID therapy can be given as part of the treatment for rheumatic diseases. However, if the patient has severe COVID-19 symptoms with severe respiratory, cardiac, gastrointestinal and renal manifestations, then the NSAID should be discontinued because of the poor prognosis and NSAID administration may lead to worsening of these symptoms.
- Corticosteroids can be given to patients with confirmed COVID-19 who are asymptomatic or with symptoms of mild to moderate infection with the smallest effective dose according to their rheumatic disease activity. In COVID-19 patients with symptoms of severe infection, the determination of the dose of corticosteroids takes into account the clinical condition on a case-by-case basis and the risk-benefit ratio of each patient.
- The conventional DMARD that can be given is HCQ. Another conventional DMARD can be given after it is proven that you no longer have COVID-19 and other infections have resolved.
- The biologic DMARD that can be given is anti-IL-6. Anti-IL-6 for the management of rheumatic diseases can be given after the COVID-19 infection is over. Administration of anti-IL-6 in rheumatoid-autoimmune patients suffering from COVID-19 and experiencing a cytokine storm refers to the recommendations issued by the IRA. Other biologic DMARDs can only be given after it is proven that they are no longer suffering from COVID-19 and other infections have been resolved.
- IVIG may be given if needed as indicated by the underlying autoimmune rheumatic disease.
- ACE inhibitors and ARBs may be given.

In this case, the patient was given empirical antibiotics for bacterial pneumonia until PCR results were available, as the possibility of bacterial pneumonia infection could not be ruled out. After the results of the PCR were confirmed, the patient was given COVID-19 therapy according to the patient's disease, namely severe COVID-19. The patient was given favipiravir as an antiviral, antibiotic because there is still a possibility of bacterial co-infection. Treatment for SLE is given hydroxychloroquine and for high doses of glucocorticoids because of the condition of the patient who has severe COVID-19.

4.Summary

We report a case of a 50-year-old woman with a diagnosis of severe COVID-19 in a newly diagnosed patient with SLE. COVID-19 is a respiratory tract infection caused by infection with SARS-CoV-2, the diagnosis is made based on clinical symptoms and there is a positive PCR swab result. The diagnosis of SLE is made based on clinical symptoms, then an antibody titer is obtained which after the titer exceeds normal, it can be continued to enter the SLE classification from ACR/EULAR 2019, where the patient's score has met the SLE diagnosis. Based on the data obtained, it should be noted that SLE patients may be more susceptible to COVID-19 infection compared to the general population.

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