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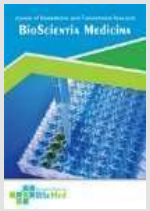

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Systemic Lupus Erythematosus with Lupus Nephritis, Community-Acquired Pneumonia, Bilateral Pleural Effusion, Pericardial Effusion, and Hypoalbuminemia in a 20-Year-Old Male Patient: A Case Report

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A B S T R A C T

Background: Systemic lupus erythematosus (SLE) is a chronic autoimmune disease characterized by multisystem inflammation. Lupus nephritis is a serious complication of SLE that can cause kidney failure. Community acquired pneumonia (CAP), bilateral pleural effusion, pericardial effusion, and hypoalbuminemia are other complications that can occur in SLE patients. **Case presentation:** We report the case of a 20-year-old man with SLE who presented with lupus nephritis, CAP, bilateral pleural effusion, pericardial effusion, and hypoalbuminemia. Patients were diagnosed with SLE based on the American College of Rheumatology (ACR) classification criteria. The diagnosis of lupus nephritis is made based on the presence of proteinuria, hematuria, and casts on urinalysis, as well as findings on kidney biopsy. CAP is diagnosed based on the presence of fever, cough, cough with phlegm, and infiltrates on chest X-ray. Bilateral pleural effusion and pericardial effusion were diagnosed based on physical examination and findings on chest ultrasound. Hypoalbuminemia is diagnosed based on low serum albumin levels. Patients are treated with steroids, antimalarials, diuretics, and antibiotics. The patient's symptoms improved and complications resolved. **Conclusion:** SLE is a complex disease that can cause a variety of serious complications. Early diagnosis and treatment of these complications are essential to improve the patient's prognosis. This case shows that SLE can cause a variety of serious complications, including lupus nephritis, CAP, bilateral pleural effusion, pericardial effusion, and hypoalbuminemia. Early diagnosis and treatment of these complications are essential to improve the patient's prognosis.

1. Introduction

Systemic lupus erythematosus (SLE) is a chronic autoimmune disease characterized by multisystem inflammation. The immune system in SLE sufferers attacks the body's own tissues and organs, causing damage and various symptoms. SLE is a complex disease with causes that are not yet fully understood, but genetic, hormonal, and environmental factors are thought to play a role in its development. The prevalence of SLE in Indonesia is estimated to be around 0.02%, with a higher prevalence in women than men (female: male ratio around 9:1). SLE generally attacks young adults, between 15 and 45 years. The exact cause of SLE is not fully known, but a combination of genetic, hormonal, and environmental factors is thought to play a role in its development. Individuals with a genetic predisposition to SLE are more susceptible to triggering this disease when exposed to trigger factors, such as infections, sun exposure, or certain medications. In SLE, the

immune system produces abnormal antibodies called autoantibodies. These autoantibodies attack the body's own healthy tissues and cells, triggering inflammation and tissue damage. This inflammation can occur in various organs of the body, including the skin, joints, kidneys, lungs, heart, and brain.[1,2]

The clinical manifestations of SLE vary greatly between individuals and can change over time. Joint pain and stiffness can occur in various joints, especially in the hands, wrists, knees, and ankles. Joint pain is usually worse in the morning and improves with activity. The most typical skin rash in SLE is a butterfly rash which appears around the nose and cheeks. Other rashes that can occur on the SLE include photosensitive rash (a rash that appears or worsens when exposed to sunlight), maculopapular rash, and mouth ulcers. Fever is a common symptom of SLE and is usually a mild fever. Fatigue is a common symptom of SLE and can be so severe that it interferes with daily activities. Weight loss for no apparent reason can occur in SLE. Hair loss and thinning of hair can occur in the SLE. Oral thrush is a painless sore that appears in the mouth. Kidney disorders are a serious complication of SLE that can cause kidney failure. Symptoms of kidney problems can include proteinuria (protein in the urine), hematuria (blood in the urine), and decreased kidney function. Lung disorders are a serious complication of SLE that can include pneumonia, pleural effusion (fluid buildup around the lungs), and pulmonary fibrosis (scar tissue in the lungs). Heart problems are a serious complication of SLE which can include myocarditis (inflammation of the heart muscle), pericarditis (inflammation of the membrane around the heart), and endocarditis (inflammation of the inner lining of the heart). Neurological disorders are serious complications of SLE that can include headaches, seizures, confusion, and stroke. Psychiatric disorders are a common complication of SLE and can include depression, anxiety, and psychosis.[3,4]

Lupus nephritis is a serious complication of SLE that can lead to kidney failure. Approximately 50% of SLE patients will develop lupus nephritis during the course of their disease. Lupus nephritis is diagnosed based on the presence of proteinuria, hematuria, and casts on urinalysis, as well as findings on renal biopsy. Community-acquired pneumonia (CAP) is a lung infection acquired outside of a hospital. CAP is a common complication in SLE patients, especially in those with lupus nephritis. CAP is diagnosed based on the presence of fever, cough, cough with phlegm, and infiltrates on chest X-ray. Pleural effusion is a buildup of fluid in the space between the lungs and the chest wall. Pleural effusion can occur in SLE patients for various reasons, including lupus nephritis, pneumonia, and heart failure. Pleural effusion is diagnosed based on physical examination and findings on chest ultrasound. Pericardial effusion is a buildup of fluid in the space between the heart and the pericardium. Pericardial effusion can occur in SLE patients for various reasons, including lupus nephritis, myocarditis, and pericardial tamponade. Pericardial effusion is diagnosed based on physical examination and findings on ECG and echocardiography. Hypoalbuminemia is a low serum albumin level. Hypoalbuminemia can occur in SLE patients for various reasons, including lupus nephritis, malnutrition, and protein-losing enteropathy. Hypoalbuminemia can cause edema, ascites, and hypotension.[5-7]

2. Case Presentation

A 20-year-old man came to the hospital with complaints of pain and stiffness in the joints, fever, cough, cough with phlegm, and shortness of breath. The patient had a history of being diagnosed with Systemic lupus erythematosus (SLE) 6 months previously and had been undergoing treatment with methylprednisolone and hydroxychloroquine for 4 months, but had not taken medication for 2 months. The patient also has a history of mouth ulcers. Physical examination showed blood pressure 130/90 mmHg, pulse 100 beats per minute, temperature 38.5°C, respiratory rate 24 breaths per minute, and oxygen saturation 92%. A cardiac examination revealed a pericardial rub murmur, and a lung

examination revealed bilateral crackles. Abdominal examination revealed ascites and joint examination revealed tenderness and swelling of the wrist. Investigations showed proteinuria 2+, hematuria 3+, and granular casts 5-10/field on urinalysis. Anti-ds DNA was positive and the ANA profile showed RNP/Sm positive and Sm positive. Chest x-ray showed bilateral infiltrates, chest ultrasound showed bilateral pleural effusion and pericardial effusion, and serum albumin level was low (2 g/dL).

Based on the results of the physical and supporting examination, the patient was diagnosed with SLE, lupus nephritis, Community-Acquired Pneumonia (CAP), bilateral pleural effusion, pericardial effusion, and hypoalbuminemia. The patient was treated with methylprednisolone 1 mg/kgBW per day, hydroxychloroquine 200 mg per day, diuretics, and antibiotics. The patient's symptoms improved and complications resolved with treatment. Patient prognosis varies and depends on the severity of complications and response to treatment. This case shows that SLE can cause a variety of serious complications, including lupus nephritis, CAP, bilateral pleural effusion, pericardial effusion, and hypoalbuminemia. Early diagnosis and treatment of these complications are essential to improve the patient's prognosis.

Table 1. Case presentation.

Parameter	Description
Patient	Male, 20 years old
Disease history	- Diagnosed with SLE 6 months ago - Has received therapy with methylprednisolone 3x4 mg and hydroxychloroquine 1x200 mg for 4 months, but has not taken medication for 2 months - History of mouth ulcers
Main complaint	Pain and stiffness in the joints, fever, cough, cough with phlegm, shortness of breath
Physical examination	Blood pressure: 130/90 mmHg Pulse: 100 beats per minute Temperature: 38.5°C Respiratory frequency: 24 beats per minute Oxygen saturation: 92% Cardiac examination: pericardial rub murmur heard Lung examination: bilateral crackles heard Abdomen: there are ascites. Joint examination: there is tenderness and swelling in the wrist
Supporting investigation	Urinalysis: proteinuria 2+, hematuria 3+, granular casts 5-10/field of view Anti-ds DNA: positive ANA profile: RNP/Sm positive, Sm positive chest X-ray: bilateral infiltrates Chest ultrasound: bilateral pleural effusion, pericardial effusion Levels serum albumin: 2 g/dL
Diagnosis	Systemic lupus erythematosus (SLE) Lupus nephritis community-acquired pneumonia (CAP) bilateral pleural effusion pericardial effusion hypoalbuminemia
Treatment	Methylprednisolone 1 mg/kgBW per day Hydroxychloroquine 200 mg per day Diuretic Antibiotic
Prognosis	Varies, depending on the severity of complications and response to treatment

3. Discussion

Lupus nephritis is a serious complication of systemic lupus erythematosus (SLE) which attacks the kidneys. In the case of the 20-year-old male patient described previously, the findings of 2+ proteinuria, 3+ hematuria, and 5-10 granular casts/field on urinalysis were strong indications of renal glomerular damage due to autoimmune inflammation underlying the SLE. Lupus nephritis can manifest with a variety of symptoms, including: Proteinuria: Protein in the urine indicates glomerular damage that allows protein to leak from the blood into the urine; Hematuria: Blood in the urine indicates inflammation and damage to the glomerulus; Urinary casts: Cylinders are proteins that coagulate in the kidneys and are then excreted in the urine. Different cylinder types can provide information about the type and severity of kidney damage; Decreased kidney function: In severe cases, lupus nephritis can lead to decreased kidney function, characterized by decreased glomerular filtration rate (GFR) and retention of waste products in the blood; High blood pressure: Lupus nephritis can cause hypertension (high blood pressure) because kidney damage can interfere with blood pressure regulation;

Edema: Fluid buildup in the feet, ankles, and face can result from kidney damage leading to water retention. The mechanism behind lupus nephritis involves the complexity of the immune system attacking kidney tissue. In individuals with SLE, the immune system mistakenly identifies kidney tissue as foreign, triggering an inflammatory response that attacks the glomerulus. This inflammation can damage the structure of the glomerulus and interfere with its ability to filter blood properly, resulting in protein, blood, and blood cells leaking into the urine.[8-10]

Lupus nephritis is a serious complication that can be fatal if not treated properly. Progressive kidney damage can lead to kidney failure, requiring dialysis or a kidney transplant. Early diagnosis and effective treatment are essential to prevent further kidney damage and improve the patient's quality of life. Treatment of lupus nephritis generally involves a combination of medications. Corticosteroids are powerful anti-inflammatory drugs that can help control kidney inflammation. Immunosuppressive drugs such as azathioprine or mycophenolate mofetil suppress the immune system and help reduce kidney inflammation. Diuretics help remove excess fluid and salt from the body, which can help lower blood pressure and edema. Treatment of high blood pressure with medications such as ACE inhibitors or angiotensin II receptor blockers (ARBs) is important to protect the kidneys from further damage. This patient's case is an important reminder that lupus nephritis is a serious complication of SLE that needs to be watched out for. Early diagnosis through urinalysis, renal function tests, and renal biopsy (if necessary) is essential for appropriate therapeutic intervention. With effective treatment and ongoing health monitoring, SLE patients with lupus nephritis can live well and productively. Prevention of kidney damage through blood pressure control, a healthy lifestyle, and adherence to treatment are the main keys to managing SLE and its serious complications.[11-13]

Community acquired pneumonia (CAP) is a lung infection that is acquired outside the hospital, different from hospital-acquired pneumonia (HCAP) which usually occurs in patients treated in hospitals or other health facilities. Patients with SLE have a higher risk of developing CAP compared with the general population. SLE is an autoimmune disease that causes the body's immune system to attack the body's own tissues. This weakens the immune system and makes it more susceptible to infections, including CAP. Treatment of SLE often involves taking immunosuppressive drugs that suppress the immune system. These drugs may increase the risk of infections, including CAP. SLE complications such as lupus nephritis and kidney failure can increase the risk of CAP. Smoking, lack of sleep, and malnutrition may increase the risk of CAP in patients with SLE. Symptoms of CAP in patients with SLE: Fever: Fever is a common symptom of CAP and usually occurs suddenly; Cough: The cough in CAP is usually phlegmy and can be green, yellow, or brown; Shortness of breath: Shortness of breath may occur with CAP, especially in patients with underlying lung or heart disease; Chest pain: Chest pain in CAP usually feels like a stabbing pain or chest pain; Fatigue: Fatigue is a common symptom of CAP and can make patients feel weak and tired. The diagnosis of CAP in patients with SLE is based on a combination of clinical symptoms, physical examination, and supporting examinations. Supporting examinations that can be carried out to help diagnose CAP include: Chest X-ray: Chest X-ray can show infiltrates (inflammation) in the lungs; Blood tests: Blood tests may show an increase in the number of white blood cells (leukocytes), which is a sign of infection; Culture blood test: A culture blood test may be performed to identify the bacteria or virus that causes CAP; Sputum test: A sputum test may be performed to identify the bacteria or viruses that cause CAP. Treatment of CAP in patients with SLE generally involves the use of antibiotics. The choice of antibiotic will depend on the bacteria or virus causing CAP. Patients with severe CAP may require hospitalization and administration of supplemental oxygen. CAP is a serious complication that can occur in patients with SLE. The risk factors, symptoms, diagnosis, and treatment of CAP in

patients with SLE differ from those in the general population. Prevention of CAP in patients with SLE can be done in several ways, such as vaccination, washing hands, avoiding contact with sick people, stopping smoking, and maintaining health.[14-17]

Bilateral pleural effusion, a buildup of fluid in the space between the lungs and chest wall on both sides, is a serious complication in systemic lupus erythematosus (SLE) patients. The case described above shows how bilateral pleural effusion can occur in SLE patients and how it can influence the course of the disease and the patient's prognosis. There are several mechanisms that can cause bilateral pleural effusion in SLE patients. Inflammation of the pleura, the membrane lining the lungs and chest wall, is the most common cause of bilateral pleural effusion in SLE patients. This inflammation can be caused by various factors. In SLE, the immune system attacks healthy tissue, including the pleura. This can cause inflammation and fluid leakage into the pleural space. Lung infections, such as pneumonia, can cause pleural inflammation and pleural effusion. SLE patients are more susceptible to infections because of their weakened immune systems. Congestive heart failure, in which the heart cannot pump blood effectively enough, can cause fluid to build up in the lungs and pleural space. SLE patients with lupus nephritis (kidney inflammation) are at greater risk of developing congestive heart failure. High blood pressure in the pulmonary veins, the blood vessels that carry blood from the lungs to the heart, can cause fluid to leak into the pleural space. SLE patients with pulmonary hypertension, a condition characterized by high blood pressure in the pulmonary arteries (blood vessels that carry blood from the heart to the lungs), are more at risk of developing pleural effusion. Symptoms of bilateral pleural effusion in SLE patients can vary, and some patients may experience no symptoms at all. The most common symptoms include: Chest pain: Chest pain is usually felt on the side of the chest affected by the effusion. Pain can feel sharp, stabbing, or dull. Pain usually worsens when coughing, sneezing, or lying down; Shortness of breath: Shortness of breath can occur because fluid in the pleural space presses on the lungs and limits their ability to expand; Dry cough: A dry cough may occur due to irritation of the pleura; Fever: Fever may occur if the pleural effusion is caused by an infection. The diagnosis of bilateral pleural effusion in SLE patients is based on a combination of factors, including: Clinical symptoms: Symptoms such as chest pain, shortness of breath, dry cough, and fever can help guide the diagnosis; Physical examination: A physical examination may show signs of pleural effusion, such as pleural rubbing sounds when listening to the lungs; Imaging: A chest x-ray may show fluid in the pleural space. A chest ultrasound can provide a clearer picture of the amount and location of fluid; Supporting examinations: Supporting examinations such as thoracentesis (pleural fluid sampling) can be performed to help diagnose and determine the cause of pleural effusion. Treatment of bilateral pleural effusion in SLE patients depends on the cause: Pleural Inflammation: Treatment of pleural inflammation in the SLE usually involves anti-inflammatory drugs, such as corticosteroids; Infection: Lung infections are treated with appropriate antibiotics; Congestive Heart Failure: Congestive heart failure is treated with medications that help the heart pump blood more effectively and diuretics to remove excess fluid; High blood pressure in the pulmonary veins: Treatment of high blood pressure in the pulmonary veins depends on the cause.[15-18]

Pericardial effusion is a buildup of fluid in the space between the heart and the pericardium, which is the thin membrane that covers the heart. This condition is one of the complications that can occur in patients with systemic lupus erythematosus (SLE), as experienced by the patient in this case. In SLE patients, pericardial effusion can be caused by several mechanisms. Inflammation of the pericardium, the membrane that covers the heart, can cause pericardial effusion. This inflammation can occur as a result of an autoimmune reaction in SLE patients. Inflammation of the heart muscle (myocardium) can also cause pericardial effusion. Myocarditis in SLE patients can

occur as a result of an autoimmune reaction or a complication of lupus nephritis. A medical emergency situation occurs when fluid buildup in the pericardial space compresses the heart and disrupts blood flow. Pericardial tamponade may occur in SLE patients with severe pericardial effusion. Diagnosis of pericardial effusion in SLE patients can be done through several steps: Physical examination: The doctor will listen to pericardial friction sounds, which are rough rubbing sounds that are heard when the heart beats. This sound can be heard with a stethoscope in patients with pericardial effusion; Supporting Examinations: Several supporting examinations that can be carried out to help diagnose pericardial effusion include: Echocardiography: Ultrasound examination of the heart which can show fluid buildup in the pericardial space, Electrocardiogram (ECG): Examination which records the electrical activity of the heart. ECG can show changes in the ECG pattern typical of patients with pericardial effusion. Blood tests: Blood tests can show signs of inflammation, such as increased levels of C-reactive protein (CRP) or erythrocyte sedimentation rate (ESR). Treatment of pericardial effusion in SLE patients varies depending on the cause and severity of the condition. Nonsteroidal anti-inflammatory drugs (NSAIDs): NSAIDs such as ibuprofen or naproxen can help relieve pericardial inflammation and pain. Corticosteroids: Corticosteroids such as prednisone may be used to control more severe inflammation. Diuretic drugs: Diuretics can be used to help remove excess fluid in the body, including fluid that accumulates in the pericardial space. Pericardiocentesis: A medical procedure performed to remove excess fluid from the pericardial space. Pericardiectomy: A surgical procedure performed to remove pericardial inflammation or damage. Early diagnosis and treatment of pericardial effusion in SLE patients is very important to prevent serious complications, such as pericardial tamponade which can be fatal. If a patient experiences symptoms suggestive of pericardial effusion, such as chest pain, shortness of breath, or dry cough, it is important to seek immediate medical attention. Pericardial effusion is a complication that can occur in SLE patients and can be caused by various factors. Early diagnosis and treatment of pericardial effusion is essential to prevent serious complications and improve the patient's prognosis.[16-19]

Hypoalbuminemia, or low serum albumin levels, is a common complication in systemic lupus erythematosus (SLE) patients. Albumin, an important protein in the blood, plays a crucial role in maintaining body fluid balance and transporting important substances such as hormones, vitamins, and minerals. In the case of a 20-year-old male patient with SLE who experienced hypoalbuminemia (2 g/dL). Lupus nephritis, a renal complication of SLE, is the primary cause of hypoalbuminemia in these patients. Damage to the kidney glomerulus due to autoimmune inflammation in lupus nephritis can cause leakage of albumin into the urine. This is known as proteinuria, which is characterized by high protein levels in the patient's urinalysis (proteinuria 2+). This significant loss of albumin through urine contributes directly to the decrease in serum albumin levels. SLE patients often experience decreased appetite and difficulty eating due to symptoms such as joint pain, fatigue, and mouth ulcers. This can lead to malnutrition, where inadequate protein intake contributes to low albumin levels. Protein-losing enteropathy, a malabsorptive condition characterized by excessive loss of protein through the intestine, may also contribute to hypoalbuminemia in SLE patients. Chronic intestinal inflammation due to SLE, drug side effects, or intestinal infections can cause damage to the intestinal mucosa, so that protein cannot be absorbed properly and is excreted in the feces. Decreased serum albumin levels in SLE patients can cause various serious consequences. Low albumin osmotic pressure due to hypoalbuminemia can cause fluid movement from the intravascular to the interstitial space, resulting in edema (swelling) in the legs, face, and abdominal cavity (ascites) which is observed in this patient. Hypoalbuminemia can cause a decrease in blood pressure (hypotension) due to

reduced effective blood volume. Low levels of albumin can disrupt the function of the liver, kidneys, and immune system. Hypoalbuminemia can increase the risk of infection due to decreased immune system function. Treatment of lupus nephritis with corticosteroids, immunosuppressive drugs, or biologics to reduce kidney inflammation and prevent albumin leakage. Management of malnutrition with nutritional counseling, food supplements, or parenteral nutrition to increase protein intake. Treatment of protein-losing enteropathy involves identifying and treating the underlying cause, such as intestinal inflammation or infection. Intravenous administration of albumin can help increase serum albumin levels and overcome the consequences of hypoalbuminemia such as edema and hypotension. This therapy needs to be considered carefully in patients at risk of complications such as fluid overload or heart failure. Hypoalbuminemia in SLE patients is a serious complication that can be fatal if not treated appropriately. An in-depth understanding of the underlying causes and comprehensive management, including drug therapy and albumin replacement, are essential to improve patient prognosis and their quality of life.[17-20]

4. Conclusion

SLE is a complex disease that can cause a variety of serious complications. Early diagnosis and treatment of these complications are essential to improve the patient's prognosis.

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