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Problems with Diagnosis and Management of Pneumonia in SLE Patients: Case Reports

I Nyoman Dharma Putra Wijaya, Putu Andrika
Internal Medicine Specialist Education Study Program, Faculty of Medicine, Udayana University, Department/KSM of Internal Medicine, Faculty of Medicine, Udayana University/RSUP Prof. Dr. IGNG Ngoerah, Denpasar, Indonesia
dharma.wijaya83@gmail.com

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Abstract
Systemic lupus erythematosus (SLE) is a chronic disease with a wide range of manifestations including pulmonary disorders. We reported two cases of patients with SLE and pneumonia. The first patient was a 23-year-old woman who had been diagnosed with SLE and then came with complaints of cough and shortness of breath in the last two weeks. Physical examination found crackles in both lungs. Laboratory examination revealed severe anemia, thrombocytopenia, hyponatremia and hypoalbumin. Thoracic x-ray revealed pneumonia. Patient was diagnosed with pneumonia, relapsed mild SLE, lupus nephritis and severe anemia. She was treated with Levofloxacin 1x750mg, Ceftriaxone 1x2 gram, Methylprednisolone 1x62.5mg and Hydroxychloroquine 1x200mg. The second patient was a 20-year-old woman with complaints of fever, cough, and shortness of breath since two weeks ago. Physical examination showed an increase in RR and temperature. Laboratory examination showed leukopenia, mild anemia, thrombocytopenia and elevated procalcitonin and ESR and hypoalbumin. X-ray examination showed pneumonia. Echocardiography revealed a mild circular pericardial effusion. She was diagnosed with relapsed severe SLE with mild anemia, severe thrombocytopenia and lupus nephritis, pneumonia, hypoalbuminemia, and pericardial effusion. She was treated with Ceftazidime 3x2 grams, Levofloxacin 1x750mg, Hydroxychloroquine 1x200mg, Methylprednisolone 2x62.5mg, TC transfusion 5 bags/day, albumin 20% 100ml and fluconazole 1x200mg was added on fourth day of care. Both patients died on the 10th day. Pulmonary involvement can be primary (caused by SLE itself) or secondary (caused by infection or drug toxicity), acute or chronic. The course, treatment and prognosis vary greatly depending on the specific pattern of the disease.

Keywords: Systemic Lupus Erythematosus; Pneumonia; Acute Pneumonitis; Autoimmune Disease;

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Introduction

Systemic lupus erythematosus (SLE) is a chronic autoimmune disease that has a highly varied clinical phenotype. SLE is more common in women than men with a ratio of approximately six women to one man (Rees, Doherty, Grainge, Lanyon, & Zhang, 2017). The incidence of SLE was also associated with the ethnicity of the patients where the black ethnicity had the highest incidence and prevalence while the white ethnicity had the lowest incidence and prevalence. In addition, the incidence and prevalence of SLE is also related to the location of the country where countries located in the equatorial region have the highest incidence due to ultraviolet radiation that can trigger SLE (Grant, 2016).

The pathogenesis of SLE is multifactorial and not yet fully understood. The pathogenesis of SLE is related to the interaction between non-Mendelian genetic predispositions, hormonal and environmental factors that then lead to changes in innate and adaptive immunity. Specifically, the pathogenesis of SLE is characterized by disruption of apoptotic cell destruction through phagocytosis and autoreactivity of B cells and T cells resulting in abnormal production of autoantibodies, and the formation of immune complexes by nuclear and cytosolic antigens. This immune complex can activate the classical pathways of the complementary system, resulting in inflammation and organ damage (Murphy, Lisnevskaia, & Isenberg, 2013)

The clinical manifestations of SLE vary where abnormalities in the skin are one of the most commonly found clinical manifestations. As many as 70% of patients have experienced skin abnormalities related to SLE throughout the course of the disease. Other commonly found auscultative manifestations are malar rash (40%), alopecia (24%) and oral ulcers (19%) (Patel & Werth, 2002), (Blake & Daniel, 2019). SLE can have a wide range of manifestations including disorders of the pulmonary, pleural and vascular parenchyma. Manifestations of pulmonary disorders in SLE patients can include pulmonary hemorrhage, pulmonary hypertension, acute lupus pneumonitis, chronic interstitial pneumonitis, pulmonary shrinkage syndrome, pulmonary vasculitis, pulmonary embolism, bronchitis obliterans, pulmonary nodules, opportunistic lung infections and pleuritis. In addition, SLE therapy can also result in an increased incidence of lung infections (Cuchacovich & Gedalia, 2009), (Singh & Singh, 2020)

The exact prevalence of SLE-related lung disease still varies with a fairly wide range in some studies. Most studies report that 20-90% of patients with SLE will develop lung disorders (Aguilera-Pickens & Abud-Mendoza, 2018). However, the latest study shows a range between 50-70%. More than 50% of SLE patients will experience pleuropulmonary manifestations at least once in their lifetime (Aguilera-Pickens & Abud-Mendoza, 2018). As many as 60% of patients reported dyspneuation at least once in the course of their illness and abnormal lung function tests were found in 30-40% of cases (Fenlon, Doran, Sant, & Breatnach, 1996). Sebuah Cohort Latin American GLADEL (*Latin American Lupus Study Group*) reported at least one pleuropulmonary manifestation in 421 of 1,480 patients (28.4%) (Haye Salinas et al., 2017).

Pulmonary manifestations in SLE are associated with increased mortality and depend on the type and extensibility of pulmonary involvement present. Clinical manifestations of lung in SLE patients should be evaluated routinely such as shortness of breath, chest pain, decreased exercise tolerance and hemoptysis. Chronic pulmonary symptoms in SLE patients have a negative effect on the patient's physical status and quality of life (Fidler, Keen, Touma, & Mittoo, 2016)

Case Illustration

Case 1

A 23-year-old woman came with a major complaint of coughing for two weeks. Coughing is felt continuously without phlegm. The cough was said to have improved after taking cough medicine. Patients also complain of shortness of breath every time they cough and take a deep breath. Tightness is said to not improve with a change of position or rest. Previous fever complaints were denied by the patient. The patient also complained of weakness all over the body since one week and aggravated two days before being admitted to the hospital. The patient was unable to stand because of weakness in both legs. The patient can still eat and drink well but must be helped by his mother. Pain complaints during bowel movements and bowel movements are denied by the patient. Patients deny complaints of nausea, vomiting, and diarrhea.

The patient has a history of SLE since 2017 and is routinely treated with Hydroxychloroquine, Methyl prednisone, Mycophenolate mofetil and Telmisartan. The patient has not been vaccinated against COVID. No family of patients experienced similar complaints. The patient is not currently employed and has no history of smoking or drinking alcohol. On the examination of vital signs, it was found that he was in moderate pain with blood pressure of 110/60 mmHg, pulse 100 times per minute, RR 26 per minute, temperature 36.2^{Oc} Peripheral saturation of 99% on room air. On physical examination, anemic conjunctiva was found, bronchiolar breath sounds with Ronchi were obtained in ICS 4-5 of the second lung without wheezing. Other physical examinations within normal limits.

On a complete blood examination, severe anemia (Hb 5.5 mg/dL) normochromic normocytositers with thrombocytopenia (PLT 64x10^3/uL) and increased NLR (27.48). Examination of liver and kidney function within normal limits. In the electrolyte examination, hyponatremia (Na 127 mmol/L) and hypoalbumin (1.97 g/dL) were found. Blood gas analysis examination within normal limits. In the urinalysis examination, it was found that protein +3, blood +2, leukocytes were negative, and bacteria were 626.5. Negative antigen swab examination at two examinations. On thoracic examination, cardiomegaly was found with consolidation of the upper to lower zones of the right lung which impressed pneumonia. ECG examination with the impression of sinus rhythm with a pulse of 109 times per minute.

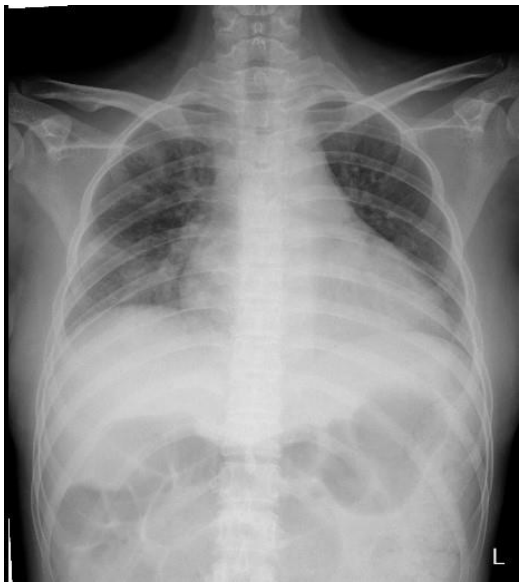

Figure 1. Thoracic photograph in case 1

Patients were diagnosed with pneumonia with suspicion of bacterial infection, SLE mild flare up (MEX SLEDAI 4) with lupus nephritis, normochromic severe anemia normocyte and thrombocytopenia, chronic inflammatory hypoalbuminemia ec susp susp, chronic hyponatremia asymptomatic hypothmolar euvolemic susp SIADH, orbs cardiomegaly ec susp cardiomyopathy SLE and discarded COVID 19.

The patient was then treated with IVFD NaCl 0.9% 16 drops per minute, diet 1,900 kcal per day, PRC transfusion with a target of Hb 10 gr/dL, N-acetylcysteine 200mg every 8 hours, drip human albumin 20% 1 colf per day, levofloxacin 750mg once per day, Ceftriaxone 2gram once per day, methylprednisolone 62.5mg once per day, hydroxychloroquine 200mg once per day, and Telmisartan 80mg once per day.

Case 2

A woman, 20 years old, came with complaints of fever that she felt nine days before entering the hospital. The fever was felt to disappear. The patient also complained of coughing, coughing was said to be non-phlegmatic accompanied by shortness of breath, shortness of breath was felt to not improve with a change of position or rest. The patient also complained of weakness throughout the body since 2 weeks ago, weakness was felt to not improve with rest.

Hair loss complaints since one year ago. Patients also complained of pain in the joints, especially in the knees, complaints of redness on the face were denied, there were bruises on both hands without any previous history of trauma. The patient also said that both of his legs were swollen. Complaints of canker sores in the mouth are denied. The new patient was suspected of suffering from SLE when he was admitted to the hospital this time. The patient had received metillprednisolone treatment and had done an ANA IF examination. The patient was admitted to the hospital at Ganesha Hospital two weeks ago and given cephaxor and ceftazidime.

No family of patients had similar complaints or other autoimmune diseases. The patient is currently not working, does not drink alcohol and smokes. In the examination of vital signs, the patient was in a state of moderate pain with blood pressure of 130/80 mmHg, pulse 92 times per minute, RR 25 times per minute, temperature 38^{oC} and peripheral oxygen saturation is 97% in room air. On physical examination, it was found that the anemic conjunctiva with pitting edema on both legs. Other physical examinations within normal limits. Complete blood tests showed leukopenia (WBC $0.58x10^3$/uL), mild anemia (Hb 9,2 g/dL), trombositopenia (PLT $5x10^3$/uL). There was an increase in procalcitonin (0.25 ng/mL) and LED (41.0 mm/hour) and hypoalbumin (2.39 g/dL). Complete fecal examination, urinalysis, liver function, kidney function, electrolytes and blood gas analysis within normal limits.

Examination of peripheral blood swabs found erythrocytes with a large population of normochromic cells, anisopoikilocytosis (ovalocytes +, sigar cells +), polychromatic cells (-), normoblasts (-), leukocytes with the impression of greatly decreased number, differential count neutropenia, toxic granules (-), vacuolization (-), young cells not found, platelets with the impression of decreased number, giant platelets (-), clumping (-) so that it was concluded normochromic anemia normochromic anemia, leukopenia and thrombocytopenia (pansitopenia). Blood culture tests showed no germ growth.

X-ray examination found consolidation in the lower zone of the right lung with the impression of pneumonia. The patient then underwent a second lung examination, nine days later with a picture of *perivascular haziness of* the left right lung, bronchoscular discharge increased and a cardio myographic impression with pulmonary edema and bilateral pleural effusion was obtained. The ECG examination obtained sinus rhythm with a pulse of 70x/minute and T inversion on V1-V4. The patient was consulted to the cardiology department and then an echocardiography was performed where LV *increasing remodeling*, EF 67%, and pericardium *mild circumferential effusion* with a diameter of 0.4-0.5 cm were found.

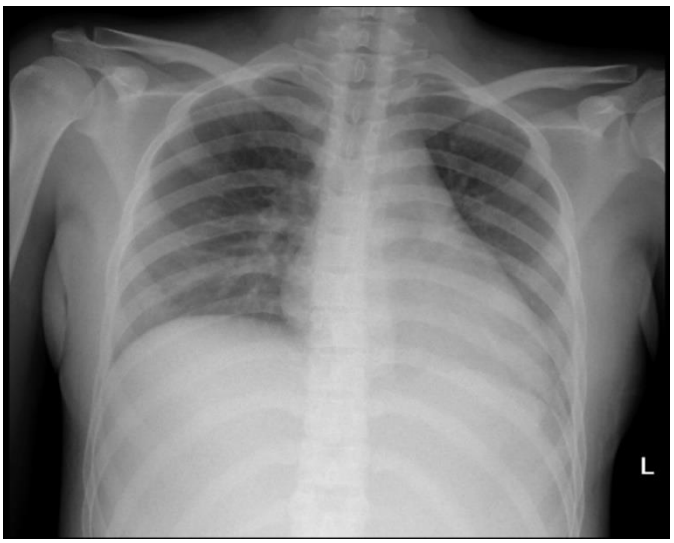

Figure 2. Initial thoracic photograph in case 2

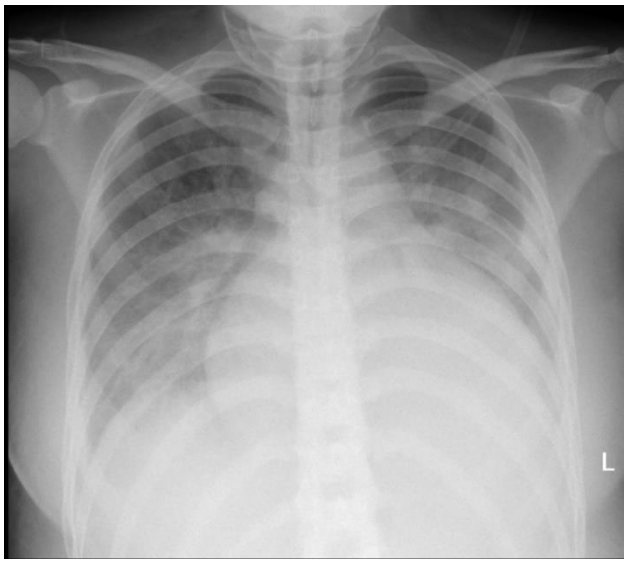

Figure 3. Thoracic photograph of re-evaluation in case 2

Patients were then diagnosed with severe activity degree SLE with mild normochromic normocyte anemia with suspicion of hemolytic autoimmune anemia (AIHA), and suspicion of *lupus nephritis*, community pneumonia, hypoalbuminemia, *incomplete RBBB*, high risk of VTE and pericardium effusion.

The patient was treated with IVFD NaCl 0.9% 20 tpm, O2 cover 8 lpm, diet high in calories and protein 1900kcal/day, Ceftazidime 3x2 grams IV, Levofloxacin 1x750mg IV, paracetamol 3x500mg PO, hydroxychloroquine 1x200mg PO, methylprednisolone 2x62.5mg IV, TC transfusion 5 bags/day up to platelets ≥20,000, and drip albumin 20% 100ml IV. Fluconazole therapy 1x200mg is added on day 4 of treatment. The patient then experienced a deterioration during treatment and died on the 10th day of treatment.

Case 3

A 33-year-old woman came with complaints of shortness of breath. Complaints of shortness of breath were felt since 1 week before entering the hospital. Complaints of tightness are felt not to improve with rest and are felt to be aggravated when the patient coughs and is moderately active. Complaints of shortness of breath are not accompanied by the sound of wheezing. The patient also complained of a cough that was felt continuously since 1 week ago with thick yellowish phlegm accompanied by a cold and a stuffy nose. Complaints accompanied by a fever that disappeared arose from 1 week which was felt to improve after taking paracetamol, but then the fever reappeared.

From the anamnesis, it was found that the patient had a history of kidney disease and routinely underwent dialysis twice per week. History of SLE since 1 year ago with steroid treatment and immunosuppressants. In the vital sign examination, the patient was in a moderately sick state with blood pressure of 100/80 mmHg, pulse 89 times per

minute, RR 21 times per minute, temperature of 38^{oC} and peripheral oxygen saturation of 97% in room air.

On physical examination, a Ronchi was found in the second basal of the pulmonary field with the impression of a right pleural effusion. Other physical examinations within normal limits. Laboratory examination showed leukocytosis (12.29x 103/μL), hemoglobin and platelets within normal limits. The results of the evaluation of liver and kidney function and blood gas analysis were within normal limits. The results of the pleural fluid analysis showed an exudate impression. In the examination of the pulmonary thoracic photo, a sheath was found in the right lung with a sign of pneumonia and effusion of the right pleura, and a sheath was also found in the heart with suspicion of effusion in the pericardium.

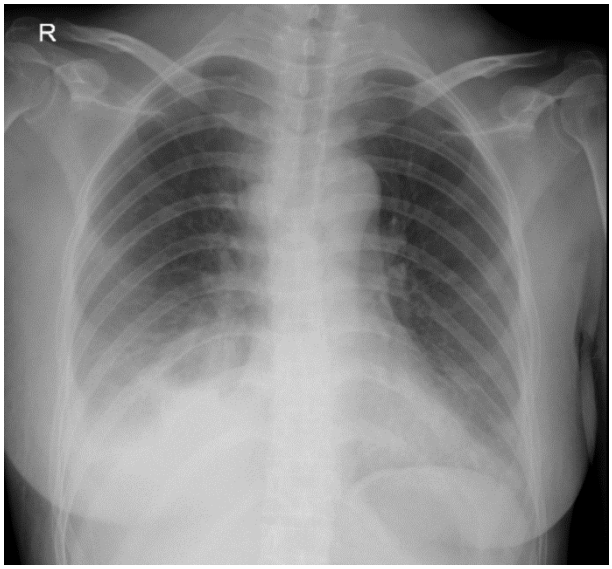

Figure 4. Thoracic photograph in case 3

Paien was diagnosed with *non-severe PSI class III community pneumonia*, mild degree SLE, grade V CKD with suspicion of lupus nefritts, chronic heart failure with paracetamol therapy 500 mg every 8 hours, ceftriaxone 2 grams every 24 hours, azithromycin 500 mg every 24 hours, acetylcysteine 200 mg every 8 hours, hydroxychloroquine 200 mg every 24 hours, Methylprednisolone 31.25 mg every 12 hours. The patient underwent hospitalization for 10 days with improved condition and continued with outpatient treatment.

Discussion

Respiratory system involvement in patients with SLE can be divided into pleural diseases such as pleuritis with or without pleural effusion, parenchymal, vascular and respiratory muscle. Parenchymal involvement is acute lupus pneumonitis, diffuse alveolar hemorrhage, and interstitial lung disease. Meanwhile, those included in vascular diseases are pulmonary embolism and pulmonary hypertension.

Respiratory muscle disease is a syndrome of pulmonary shrinkage, inflammatory myopathy and drug toxicity (Hannah & D'Cruz, 2019). Pleural disease is an intrathoracic manifestation of SLE that is most often found with an incidence of pleuritis or pleural fibrosis of 50-83% at autopsy and an incidence of pleural effusion at radiological examination of 16-50%. Pleural involvement can be one of the manifestations of SLE and is associated with pericarditis (Crestani, 2005), (Lee & Strek, 2021). In this case, both patients were diagnosed with SLE and pneumonia which provides an overview of parenchymal involvement. One of the most common parenchymal involvements found in patients with autoimmune rheumatic disease is interstitial lung disease (*interstitial lung disease*/ILD). The incidence of ILD in patients with SLE is about 1-15% (Aguilera-Pickens & Abud-Mendoza, 2018).

Although rarely severe, the clinical picture of the disease is usually slow and stable. Two-thirds of patients with SLE had an asymptomatic picture and the remaining one-third provided a consistent ILS picture on chest CT-scan. The disease is more common in patients with long-term SLE (>10 years) and patients with older age at the time of initial presentation (>50 years) (Mathai & Danoff, 2016). Patients with SLE-related ILD had clinical presentation of examinerial dyspneu and non-productive cough with ronchi on physical examination. Clubbing and peripheral cyanosis are rarely found in patients with ILD.

Patients with long-term disease (>10 years), Raynaud's phenomenon, seropositivity of anti-U1 RNP antibodies, sclerodactyly and nail capillary abnormalities associated with the radiographic picture associated with ILD (Eisenberg H, Dubois EL, Sherwin RP, Balchum OJ, 1973) at (Amarnani, Yeoh, Denneny, & Wincup, 2021). Pneumonitis can be a precursor to chronic ILD in some patients. Lupus pneumonitis is a fatal syndrome characterized by an acute onset of fever, pleuritis chest pain and tachypneux with a mortality of up to 50%. The disease can be accompanied by ronchy on physical examination and hemoptysis (Mittoo & Fell, 2014).

The diagnosis of SLE-ILD depends on the clinical picture, thoracic, histopathology and physiological abnormalities of the lungs. Lung biopsy examination is the gold standard in diagnosing ILD, while in both cases the patient is in an unstable condition so it is not possible to do so. The most common histological picture found in SLE-ILD is non-specific interstitial pneumonia. Therapy from SLE-ILD depends on the opinion and experience of the clinician. Corticosteroids (prednisone 1 mg/kgBB, maximum up to 60mg or equivalent) and cyclophosfamid (1-2mg/kg orally depending on kidney function and the patient's age) can be the initial therapy for severe ILD. Mild to moderate ILD can be treated with moderate doses of croticosteroids and azathioprine or mycophenolate mofetil (Mittoo & Fell, 2014). SLE patients have a high risk of infection due to both common and opportunistic pathogens. The types of infections that are often found in patients with SLE are lung, urinary tract and skin infections. Bacteria are the most common pathogen, followed by viruses and fungi (Navarra & Leynes, 2010), (Dorgham & Anwar, 2021). The EuroLupus cohort found that 36% of patients with SLE developed

infections and about 30% of deaths were caused by infections in *follow-up* in five years (Cervera, Ricard, 2003), (Zen et al., 2023).

Several causes of increased risk of infection have been studied. One theory is that there is a non-Mendelian genetic predisposition because the risk of severe infection increases as SLE progresses. Immune system dysfunction both adaptive and *innate*, complement deficiency, IgG deficiency, functional asplenia, cytokine production disorders, chemopathic disorders and phagocytosis are the main disorders associated with increased risk. In addition, SLE patients also experience structural disorders in the airways such as respiratory muscle weakness, parenchymal disease, bronchiectasis and atelectasis with local mucociliary clearance disorders (Dorfmüller, P., Humbert, M., Perros, F., Sanchez, O., Simonneau, G., Müller, K. M., & Capron, 2007)

In addition to internal factors, an increased risk of infection is also suspected due to the use of immunosuppressants such as CYC, azathioprine, RTX and belimumab. Corticosteroids can also provide immunosuppressive effects, especially if used for a long period of time (>3 weeks), in high doses and used in combination with other immunosuppressants (Navarra & Leynes, 2010), (Dorgham & Anwar, 2021). In this case, in all three cases, they received corticosteroid treatment and immunosuppressants since being diagnosed with SLE.

Various pathogens can result in infection in SLE patients. However, *Streptococcus pneumoniae* is the most common pathogen that causes respiratory infections. Followed by Salmonella sp. related to bacteremia, especially in patients with functional asplenia. Among the fungal pathogens, *Pneumocystis jiroveci*, *Cryptococcus neoformans*, *Candida albicans*, Aspergillus sp is the most identified type in SLE patients. The viral infections that have been reported are cytomegalovirus and varicella zoster (Navarra & Leynes, 2010), (Dorgham & Anwar, 2021). In this case report, the blood cultures in the first and second cases showed no germ growth while in the third case bacteria were found *Streptococcus viridans* which is the normal flora of the airways.

Diagnosis of infection in SLE patients is particularly challenging because the infection usually presents an atypical picture due to immunosuppression. In addition, lung infections can also provide a picture similar to *lupus flare*. In this context, infection should be excluded first in SLE patients with respiratory complaints with or without infilitrate images before increasing the dose of immunosuppressant drug therapy. Bronchoscopy with BALF (*Bronchoalveolar Lavage Fluid*) can help isolate pathogens and initiate targeted therapy. Short-term reduction of immunosuppressive therapy doses may be needed in patients with severe cases during immunosuppressant therapy to improve immune response (Lai, Sun, Lin, Yang, & Tsai, 2021). In the first case, patients were treated with the antibiotic levofloxacin 750mg once per day and Seftriaxone 2g once per day. In contrast to the second case where the patient was treated with the antibiotic Seftazidime 2 grams every 8 hours and Levofloxacin 750mg once per day, then on the 4th day of treatment was added fluconazole 200mg once per day because there was no improvement in clinical condition.

As for the third case, get the antibiotic ceftriaxone 2 grams once per day and azitromcisin 500 mg once per day for 7 days. In all three cases, patients continued to receive immunosuppressive therapies such as hydroxychloroquine and methylprednisolone during antibiotic administration. In addition to pneumonia due to infection, pulmonary parenchymal disorders can also be caused by acute pneumonitis with a prevalence of 2-9%. The GLADEL cohort reported the prevalence of acute pneumonitis in SLE patients was 2.3% and was associated with non-ischemic heart disease (Haye Salinas et al., 2017). Acute pneumonitis is mainly found in young patients who have just been diagnosed with SLE. However, pneumonitis can be an early manifestation of undiagnosed SLE in 50% of patients (Torre & Harvey, 2011). In this case report, the first and second cases of the patients were women in their early 20s who were newly diagnosed with SLE while in the third case were 33-year-old women who were diagnosed with SLE at the age of 32.

Symptoms of acute pneumonitis are non-specific such as cough with or without hemoptysis, dyspneux and fever. In severe cases, hypoxemia and acute respiratory failure can occur. In some cases, acute pneumonitis can be an early manifestation of SLE (Cheema & Quismorio Jr, 2000), (Shin et al., 2022). The radiographic characteristics found are uni/bilateral alveolar infiltration, usually with a predominance in the pulmonary basal area with or without pleural effusion. In this case report, the thoracic x-ray images of the two patients showed consolidation in the basal of the right lung. Wan, et al. reported five serial cases in which acute pneumonitis was an early manifestation of SLE. They reported that 100% of patients had an opacity picture *ground glass* and bilateral consolidation on CT-scan of the lungs (Wan, Tea, & Jobli, 2016). In the first and second cases, it is unstable so that it is not possible to carry out a pulmonary CT-scan examination, while in the third case it shows a response with the administration of antibiotics so that a pulmonary CT-scan examination is not carried out.

The main differential diagnosis of acute pneumonitis is an infection, so it is recommended to give empirical antibiotics and carry out culture examinations. Antibiotics can be stopped when the infection has been excluded (Crestani, 2005), (Lee & Strek, 2021). *Bronchoalveolar lavage* (BAL) can be performed for the exclusion of infections characterized by increased cellularity of already activated polymorphonuklear cells. Extraction of biopsy specimens may also be performed in some cases where there is doubt for the exclusion of alternative etiology. Biopsy findings are non-specific such as alveolar wall damage and necrosis, inflammatory cell infiltration, edema, bleeding and hyaline membrane (Torre & Harvey, 2011).

Other differential diagnoses are organopneumonia, malignancy, diffuse alveolar hemorrhage, pulmonary edema, and drug toxicity. Keep in mind that in diagnosing acute pneumonitis, it is almost always simultaneous with the presence of *Flares* and involvement of other organs such as serositis and impaired kidney function. In the vast majority of cases, acute pneumonitis is accompanied by the presence of anti-SSA antibodies (82%). The diagnosis can be established if a combination of pneumonitis with multi-organ involvement and positive anti-SSA is found (Chen, Tseng, Yang, Tsao, &

Lin, 2014). In this case report, the first and second cases of the patient experienced *Flares* with the involvement of the lungs, hypo albumin and lupus nephritis. In these three cases, no anti-SSA examination was carried out on the three patients. Guidelines regarding the treatment of acute pneumonitis in SLE patients do not yet exist so the current therapeutic approach is based only on case reports. Administration of broad-spectrum antibiotics is recommended until the infection can be excluded. The main management of acute pneumonitis is the administration of prednisone 1-1.5 mg/kgBB/day (Crestani, 2005), (Lee & Strek, 2021).

If there is no response within 72 hours, feeding is recommended to administer glucorticoid pulsation doses intravenously (methylprednisolone 1g/day for 3 days) followed by prenidsone 1-2mg/kg/day and then done *tapering-off* dosage according to clinical improvement. In addition, other immunosuppressive agents such as cyclophosphamide, rituximab (RTX) and intravenous immunoglobulins can be given in severe cases. Patients with severe tachypneux or hypoxemia and/or suspected alveolar hemorrhage should be admitted to the intensive care unit and given a dose of methylprednisolone pulsation as initial therapy (Pego-Reigosa, J. M., Medeiros, D. A., & Isenberg, 2009). In all three case reports, patients received Hydroxychloroquine and Methylprednisolone from the first day of treatment.

Acute pneumonitis in SLE patients has a poor prognosis with a mortality rate of above 50% where the last series of cases reported a mortality rate of 40% (Wan et al., 2016). Factors associated with poor outcomes are co-infection, aspiration, diaphragm dysfunction, kidney or heart failure, drug and oxygen toxicity (Kakati et al., 2007). The predominance of lymphocytes in BAL is associated with better outcomes while the predominance of eosinophils or neutrophils is associated with higher mortality rates. As many as 50% of patients who recovered, experienced impaired respiratory function and persistent infilitrate with the risk of progression to chronic interstitial pneumonitis (Mittoo & Fell, 2014). In this case report, the first and second cases of the patient died after treatment for approximately ten days, while the third patient recovered after undergoing treatment for ten days.

Conclusion

Systemic lupus erythematosus (SLE) is a chronic autoimmune disease that has a highly varied clinical phenotype with a wide range of manifestations including disorders of the pulmonary, pleural and vascular parenchyma. We reported two cases of patients with SLE and pneumonia. Both patients have been given immunosuppressants and antibiotics according to the protocol but both patients have not shown an adequate response to the therapy given. This can be caused by several factors, including difficulties in establishing the cause of infection as the basis for proper antibiotic administration where infection can also cause reactivation of SLE disease in patients and vice versa, SLE patients who receive immunosuppressants have a high risk of developing new infections during treatment. In accordance with the literature, it is stated that the incidence of

pneumonia in SLE patients has a poor prognosis with a high mortality rate, so it is necessary to establish and handle pneumonia in each SLE patient early and precisely.

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