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Systemic Lupus Erithematosus (SLE) With Tuberculosis Lymphadenitis: A Case Report

Slamet Wahyudi[1,2*], Awalia[3,4]

[1]Study Program of Internal Medicine, Faculty of Medicine, Universitas Airlangga, Surabaya, Indonesia

[2]Department of Internal Medicine, Dr. Soetomo General Academic Hospital, Surabaya, Indonesia

[3]Division of Reumatology, Department of Internal Medicine, Faculty of Medicine, Universitas Airlangga, Surabaya, Indonesia

[4]Division of Reumatology, Department of Internal Medicine, Dr. Soetomo General Academic Hospital, Surabaya, Indonesia

***Corresponding Author:**

Slamet Wahyudi

Study Program of Internal Medicine, Faculty of Medicine, Universitas Airlangga, Jl. Mayjend Prof. Dr. Moestopo No. 6-8, Airlangga, Gubeng, Surabaya, East Java, 60286, Indonesia

Email ID: slametwahyudi1990@gmail.com

Orcid ID: 0009-0009-5784-831X

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ABSTRACT

Background: Systemic lupus erythematosus (SLE) is a chronic autoimmune disease characterized by multisystem involvement and a heightened risk of infection due to immune dysregulation and immunosuppressive therapy. Tuberculosis (TB), particularly extrapulmonary forms like lymphadenitis, poses a significant threat to SLE patients, especially in TB-endemic regions.

Case Presentation: We report a case of a 22-year-old woman with a known history of SLE who presented with fever, persistent cough, and a progressively enlarging left cervical mass. The patient had been on immunosuppressive therapy, including methylprednisolone and cyclosporine. Physical examination and imaging revealed cervical lymphadenopathy, and fine needle aspiration biopsy (FNAB) confirmed TB lymphadenitis through the presence of acid-fast bacilli. GeneXpert testing of sputum further identified rifampicin-sensitive mycobacterium tuberculosis. The patient was treated with standard anti-tubercular therapy (2HRZE/4HR) while SLE management was continued with low-dose corticosteroids, folic acid, and calcium supplementation, guided by a SLEDAI-2K score of 0, indicating no active disease.

Outcome: Over a 6-month treatment period, the patient showed clinical improvement with resolution of the cervical mass, disappearance of respiratory symptoms, and weight gain. Laboratory parameters also improved, and no SLE flare was noted throughout TB treatment.

Conclusion: This case highlights the importance of early recognition and appropriate management of TB lymphadenitis in immunocompromised patients with SLE. FNAB remains a key diagnostic tool, and a multidisciplinary approach is critical to balancing anti-tuberculous therapy and autoimmune disease management without exacerbating either condition..

Keywords: *Systemic lupus erythematosus, Tuberculosis lymphadenitis*

1. INTRODUCTION

Systemic lupus erythematosus (SLE) is a systemic autoimmune disease with multisystemic involvement. Several genetic, immunological, endocrine, and environmental factors play a role in the etiopathogenesis of SLE (Justiz et al., 2023). The overall global incidence of SLE ranges between 1.5 and 11 per 100,000 person-years. In Asia, incidence of SLE varies from 2.8 to 8.6 per 100,000 person-years (Barber et al., 2021).

Infections are one of the most common causes of morbidity and mortality in patients with systemic lupus erythematosus (SLE). Impaired immune defence and use of immunosuppressive agents (ISAs) predispose SLE patients to opportunistic infections. As an immunocompromised population, SLE patients have a higher risk of Tuberculosis (TB) infection than the general population (Xiao et al., 2020). The prevalence of TB in SLE varies between series and regions, ranging from 5 to

30%. Although pulmonary TB is the most common, extrapulmonary TB is also an important clinical problem. Extra-pulmonary TB constitutes around 15-20% of all cases of TB. TB lymphadenopathy is seen in nearly 35% of extra-pulmonary cases of TB (Patel, 2019).

Tuberculosis lymphadenitis (TBLA) can affect all ages, especially those aged 10–30 years, and more often in women. Enlarged lymph nodes are most commonly seen in the neck area and sometimes in the armpit area. Among TB lymphadenopathy cases, cervical lymph nodes are the most common site of involvement with may range from 60% to 90%. TB infection and its treatment (especially, isoniazid) may induce lupus like disease, SLE flares and perpetuate flares. In addition to the SLE influence, the use of glucocorticoids and immunosuppressants increment further the risk of developing TB. When combined with cyclophosphamide, steroids increase the risk of infection even further (Balbi et al, 2018). In the following, we report a case of tuberculosis lymphadenitis with systemic lupus erythematosus in a young woman

CASE REPORT

A 22 years old young woman complaint of fever and a mass in left neck. Fever for 1 week previously, feeling up and down. A mass in the left neck first appeared 5 months ago, felt like it was getting bigger. Throat felt sore in the last 3 days before entering the hospital. The patient had been short of breath in the last 1 day before entering the hospital. Coughing with thick yellowish phlegm for 2 weeks before admission to hospital. Patients also complained of weight loss. Decreased appetite. Night sweats, nausea and vomiting denied. Defecation and urination are within normal limits. There were no complaints of seizures, impaired consciousness, headaches, joint pain, hair loss or chest pain.

The patient was diagnosed with SLE since 6 years, had routine control at the rheumatology clinic of Soetomo Hospital and was on therapy sandimun 2x50 mg, methyl prednisolone 4 mg-0-0, calcium lactate 1x1 tab, folic acid 1x1 tab. There is no history of hypertension, diabetes mellitus, pulmonary tuberculosis before.

On admission, she presented with general condition appeared weak and pale. There was a mass in left neck, soft solid, not warm, skin coloured, painless, size 1x1 cm, firm boundaries. Blood laboratory examination revealed that haemoglobin 6.8 g/dL ; haematocrit 24.6% ; MCV 74.5 fL ; MCH 20.6 pg ; leukocytes 7280/uL ; neutrophils 79.6% ; lymphocyte 9.5% ; platelets 299000/uL ; CRP 7.7 ; HbsAg non-reactive ; anti HIV non-reactive. Radiological examination found there were consolidation patchy pattern in both lungs. Consolidation patchy pattern in both lungs can be found in SLE manifestation dd pneumonia

Based on results of the history, physical examination and additional examination the initial diagnosis in this patient is CAP PSI score 22 class II, systemic lupus erythematous SLEDAI score 0, hypochromic microcytic anaemic, hypoalbumin, neck lymphadenopathy ec susp. lymphadenitis TB. Patient was planned to do a neck lymph fine needle aspiration biopsy. Fifth day of treatment patient getting better, minimal cough, no fever, good appetite. Patient was planned to discharge from hospital and control to outpatient rheumatology clinic.

After being discharged, patient came control with the FNAB result. It showed hypocellular smear reveals a large area of necrotic debris with scattered mononuclear inflammatory cells and erythrocytes. ZN stain: positive acid-fast bacilli examination. Its conclusion was tuberculosis chronic inflammation with positive acid-fast bacilli. The patient was planned to get methyl prednisolone 4 mg-0-0. Folic acid 1x1 tab po. Calcium lactate 1x500 mg po. First line oral anti tuberculosis agents 2HRZE/4H3R3 for 6 months, with a calculated dose of drugs according to the patient's weight of 45 kg, 3 tabs 4FDC (Rifampicin 150 mg, Isoniazid 75 mg, Pyrazinamide 400 mg, Ethambutol HCl 275 mg)

After six months therapy, the cervical mass disappeared, disappearance of respiratory symptoms, and weight gain. Laboratory parameters also improved, and no SLE flare was noted throughout TB treatment.

2. DISCUSSION

Systemic lupus erythematosus (SLE) is a complex autoimmune disease with variable clinical features. SLE manifestations are associated with multiple autoantibodies, ensuing immune complex formation and deposition, and other immune processes. This complex clinical presentation and pathogenesis makes SLE a difficult disease to grasp and define (EULAR, 2019).

The diagnosis of SLE is made based on current symptoms and supporting examinations. The latest diagnostic criteria of SLE used by experts are European League Against Rheumatism (EULAR) / American College of Rheumatology (ACR) 2019. This diagnostic criterion has a sensitivity of 96.1% and a specificity of 93.4%. This criterion uses an ANA test $\geq$ 1:80 or other equivalent tests as a requirement criterion to move towards the next diagnostic criteria. If positive ANA test then the 2019 EULAR/ACR score assessment is continued to assess clinical scores and immunology. Patients are diagnosed with SLE if a score of $\geq$ 10 is obtained at least one clinical criteria (Aringer et al., 2019).

The patient was diagnosed with SLE 6 years ago and has had routine check-ups at the rheumatology clinic.

Tuberculosis (TB) is a communicable disease that is a major cause of ill health and one of the leading causes of death worldwide. TB is caused by the bacillus Mycobacterium tuberculosis, which is spread when people who are sick with TB

expel bacteria into the air (e.g. by coughing). The two types of clinical manifestation of tuberculosis (TB) are pulmonary TB (PTB) and extrapulmonary TB (EPTB) (Lee, 2015). Tuberculosis lymphadenitis is among the most frequent presentations of extrapulmonary tuberculosis (EPTB). In the United States, among 253,299 cases 18.7% were EPTB, lymphatic cases were the highest (40.4%) (Peto et al., 2009).

Autoimmune diseases including SLE, especially where immunosuppressive drugs are required based on disease activity, can increase the risk of TB. In SLE patients there is an abnormality of the immune system and the patient is receiving an immunosuppressant agent as therapy. This condition causes the patient to have a high risk of infection, one of which is TB infection. This has also become the major risk factor that accounts for the high prevalence of TB infection in SLE (Agrawal and Prabu, 2012).

A Spanish study reported, a six-fold higher incidence of TB in the SLE group as compared to the general population (Erdozain et al., 2006). Similarly, a study from Hong Kong reported, a 5 to 15-fold higher risk (Mok et al., 2005). In SLE patients, EPTB is more common than pulmonary TB (Hou et al., 2008). In a previous study on 452 SLE patients on immunosuppressive therapy, 42 were diagnosed to have tuberculosis infection, of which 23.8% had pulmonary TB while 73.8% had extra-pulmonary disease (Zhang et al., 2008). A cohort study on the incidence of TB in SLE patients in Indonesia showed on 1278 SLE patients, 131 were diagnosed to have tuberculosis infection, of which 113 patients (81.9%) had pulmonary involvement and 61 (44.2%) had extrapulmonary involvement. The most frequent form of extra-pulmonary TB was observed to be lymphadenitis (15.2%) (Hamijoyo et al., 2022).

The general symptoms between tuberculosis lymphadenitis and pulmonary tuberculosis are the same. The symptoms that can always be found include sub febrile, decreased appetite, weight loss, weakness, and night sweats. The physical appearance of superficial TB lymphadenitis is classified into 5 stages by Jones and Campbell, namely: (a) Stage I: Enlarged lymph nodes with a spongy consistency, mobile/easy to move, separated from other nodules, this indicates a nonspecific hyperplastic reaction; (b) Stage 2: larger than stage 1 with a spongy consistency, adherent to the surrounding tissue/confluent; (c) Stage 3: central tenderness due to abscess formation; (d) Stage 4: collar stud abscess formation/redness over the abscessed skin; (e) Stage 5: formation of sinuses that drain purulent secretions (Sari and Kusmiati, 2019).

Fine needle aspiration biopsy (FNAB) is a simple, safe, and inexpensive procedure for diagnosing TB lymphadenitis. On preparations from fine needle aspiration or biopsy can reveal caseous granulomatous inflammation with datia langhans cells (PNPK-TB, 2020). In addition, it has high sensitivity (78.95%) and specificity (90.32%) values (Qasmi et al., 2012). The diagnosis can also be confirmed through examination molecular fast test.

The patient complained of fever for 1 week, shortness of breath in the last 1 day. Coughing with thick yellowish phlegm for 2 weeks, decreased appetite. as well as weight loss. Found a lump on the left side of the neck which felt like it was getting bigger for the past 5 months. Physical examination revealed mass in left neck, soft solid, not warm, skin coloured, painless, size 1x1 cm, firm boundaries. From the FNAB results of the lump in the neck, the picture was consistent with TB lymphadenitis.

Assessment of SLE disease activity is necessary to determine the appropriateness of a therapy plan for each individual. The Systemic Lupus Erythematous Disease Activity Index (SLEDAI) was first introduced in 1985 as a global index that assesses SLE disease activity in the last 10 days. The SLEDAI consists of 24 clinical and laboratory variables covering nine organ systems. SLEDAI-2K scores are grouped into no disease activity (score 0), mild disease activity (score 1-5), moderate disease activity (score 6-10), severe disease activity (score 11-19), very severe disease activity (score $\geq$20).

SLEDAI-2K score	Descriptor	Definition
8	**Seizure**	Recent onset, exclude metabolic, infectious or drug causes.
8	**Psychosis**	Altered ability to function in normal activity due to severe disturbance in the perception of reality.
8	**Organic brain syndrome**	Altered mental function with impaired orientation, memory or other intellectual function.
8	**Visual disturbance**	Retinal changes.
8	**Cranial nerve disorder**	New onset of sensory or motor neuropathy involving cranial nerves.
8	**Lupus headache**	Severe, persistent headache which may be migrainous, but must be nonresponsive to narcotic analgesia.
8	**Cerebrovascular accident**	New onset of cerebrovascular accident(s). Exclude arteriosclerosis.
8	**Vasculitis**	Ulceration, gangrene, tender finger nodules, periungual infarction, splinter haemorrhages, or biopsy or angiogram proof of vasculitis.
4	**Arthritis**	≥2 joints with pain and signs of inflammation (i.e. tenderness, swelling or effusion).
4	**Myositis**	Proximal muscle aching/weakness, associated with elevated creatine phosphokinase/aldolase or electromyogram changes or biopsy showing myositis.
4	**Urinary casts**	Heme granular or red blood cell casts.
4	**Haematuria**	>5 red blood cells/high power field. Exclude stone, infection or other cause.
4	**Proteinuria**	>0.5 gram/24 hours.
4	**Pyuria**	>5 white blood cells/high power field. Exclude infection.
2	**Rash**	Inflammatory type rash.
2	**Alopecia**	Abnormal, patchy or diffuse loss of hair.
2	**Mucosal ulcers**	Oral or nasal ulcerations.
2	**Pleurisy**	Pleuritic chest pain with pleural rub or effusion, or pleural thickening.
2	**Pericarditis**	Pericardial pain with at least 1 of the following: rub, effusion, or electrocardiogram or echocardiogram confirmation.
2	**Low complement**	Decrease in CH50, C3 or C4.
2	**Increased DNA binding**	Increased DNA binding by Farr assay.
1	**Fever**	>38°C. Exclude infectious cause.
1	**Thrombocytopenia**	<100 000 platelets / x10^9/L, exclude drug causes.
1	**Leukopenia**	<3000 white blood cells / x10^9/L, exclude drug causes.

C3 = Complement protein 3, C4 = Complement protein 4, CH50 = 50% haemolytic complement activity, DNA = deoxyribonuclease, SLEDAI-2K = SLE disease activity index 2000
Summarized from *Gladman DD, Ibanez D, Urowitz MB. Systemic lupus erythematosus disease activity index 2000. J Rheumatol. 2002;29:288-91* (99).

Figure 1. Systemic Lupus Erythematous Disease Activity Index 2000 (SLEDAI-2K) scoring

In this patient, she did not have complaints of seizures, impaired consciousness, headaches, joint pain, hair loss or chest pain. The patient had a fever, but from physical examination the temperature was not more than 38°C. No abnormalities were found in laboratory results. The total SLEDAI score in this patient was 0 and was included in the no disease activity group.

The treatment of TB in patients with SLE follows the recommendations as for other patients with this disease (Prabu and Agrawal, 2010). In general, the management of extra-pulmonary TB is divided into medical therapy and surgical therapy. Medical therapy uses first-line anti-TB drugs as the main therapy (Lee, 2015). Treatment for TB lymphadenopathy is the same as treatment for pulmonary TB namely 2RHZE/4RH but the duration varies from 6 to 12 months depending on clinical conditions. Surgical excision was considered in lymphadenopathy which gives symptomatic clinical symptoms and cases drug resistance (PNPK-TB, 2020).

There are no specific guidelines for the treatment of TB in patients with SLE or vice versa. Biological treatments should be suspended for at least six months when possible. Adjuvant corticosteroid therapy is not usually indicated in active TB except

pericardial and intracranial TB, where corticosteroids show lower mortality (Silva et al., 2021). Patients with active MTB should have a complete course of anti- tuberculosis therapy before starting a biologic (anti-TNF alpha agent). The goals of treatment are to ensure cure without relapse, prevent death, stop transmission, and prevent the emergence of drug resistance.

The patient was given TB therapy according to the guidelines, administering 2HRZE/4HR for 6 months. Considering the results of the SLEDAI-2K score in patients in the no disease activity group, the patient's SLE therapy was continued with methyl prednisolone 4 mg-0-0, calcium lactate 1x1 tab, folic acid 1x1 tab.

3. SUMMARY

Reported, A 22 year old female patient with SLE accompanied by TB lymphadenitis. This patient was given anti tuberculous therapy (ATT) according to the guidelines in the form of 2HRZE/4HR for 6 months. For SLE therapy, patient continued to be given methyl prednisolone 4 mg-0-0, calcium lactate 1x1 tab, folic acid 1x1 tab based on SLEDAI-2K scoring. The results of the sixth month evaluation showed that the lump in the neck had disappeared, the cough had improved, and the gained weight.

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