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Case Report **OPEN ACCESS**

Generalized lymphadenopathy as the first clinical manifestations of systemic lupus erythematosus–A case report

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ABSTRACT

Generalized lymphadenopathy is a non-specific symptom of many diseases including infections, inflammations, malignancies, or autoimmune diseases. The presence of lymphadenopathy in systemic lupus erythematosus (SLE) is often associated with active disease and it's rarely present without SLE symptoms. We presented a case of 24 years old postnatal female who experienced of prolonged fever, weight loss, constitutional symptoms, and generalized lymphadenopathy for two months. In view of no suggestive symptoms of infections, connective tissue disease and other malignancies, our major concern was lymphoma. However, over time, diagnosis of SLE was made based on late manifestations of musculoskeletal, hematological, neuropsychiatric, and renal symptoms of lupus supported by positive autoimmune investigations. This case highlighted how SLE first manifests as generalized lymphadenopathy followed by late manifestations of other symptoms. The vague symptoms of SLE make the diagnosis challenging.

Keywords: lymphadenopathy, systemic lupus erythematosus, SLE

INTRODUCTION

Systemic lupus erythematosus (SLE) is a chronic autoimmune disease of unknown cause that affects any organ of the body. It has various clinical manifestations from mild constitutional symptoms to multiple systemic organ involvements. Prevalence of SLE was higher in women of childbearing age with a female-to-male ratio of 9 of 1 [1]. This risk was decreased in menopausal age. The incidence was lower in men but if it occurred, it tended to be more severe [1]. Diagnosis of SLE is made based on the European Alliance Association for Rheumatology/American College of Rheumatology (EULAR/ACR) criteria 2019 [2]. It requires the presence of positive anti-nuclear antibodies (ANA) as an entry criterion with a total score of 10 or more from additional criteria. The additional criteria consist of seven clinical criteria (i.e., constitutional symptoms, hematological, mucocutaneous, neuropsychiatric, renal, musculoskeletal, serosal) and three immunological criteria (i.e., antiphospholipid antibodies, complement proteins, and SLE-specified antibodies) [2]. Other than the above entry criteria, generalized lymphadenopathy can also be found in patients with SLE but it is unspecific and rarely occurs without SLE symptoms. It is common in other diseases such as TB infections, chronic inflammations and malignancy. As compared to SLE, its prevalence was higher in lymphoma [3].

Lymphoma is a group of lymphocyte malignancy with more than 90 subtypes. It can be classified as hodgkin lymphoma and non-hodgkin lymphoma [4]. Hodgkin lymphoma is a type of lymphocyte malignancy characterized by the presence of reed-sternberg cells and typically occurs among young age patients with a range of age 20 to 34 years old at time of diagnosis. Hodgkin lymphoma does not contain reed-sternberg cells and commonly occurs among older patients with a median age of 67 years old [4, 5]. Clinical presentations can be varied but common symptoms are painless lymphadenopathy. In hodgkin lymphoma, lymphadenopathy typically affects any subdiaphragmatic lymph nodes, while non-hodgkin lymphoma can be found anywhere in the body specifically from gastrointestinal tracts, skin or central nervous systems. Both types may have constitutional symptoms such as prolonged fever, unexplained weight loss and night sweats especially those in advanced disease [5]. An open biopsy is the preferred method compared to fine needle aspiration because it provides a higher yield and offers adequate material for microscopic evaluation, which is essential for diagnosing and determining the histological subtype of lymphoma [5]. This paper reported a case of a postnatal lady with generalized lymphadenopathy, fever, unexplained weight loss and constitutional symptoms, without any SLE symptoms at initial visits. Due to unclear symptoms of SLE, we misdiagnosed it as lymphoma. The clinical suspicions of SLE were made after the SLE symptoms began to appear two weeks after her first visits which led to a delay in diagnosis and treatment. This case report was done to highlight the issue of diagnostic challenges in patients with vague symptoms of SLE.

CASE REPORT

A 24-year-old Malay female who is a postnatal mother of three children with no known medical illness. She just delivered a baby boy via spontaneous vaginal delivery three months prior to her visit to our primary care clinic. Her antenatal period was uneventful. The baby was delivered at term, with a good Apgar score and is currently healthy. She presented with two months history of low-grade fever, fatigue, tiredness, and unexplained weight loss (20 kg in 2 months). Additional symptoms of palpable multiple neck lumps, which varied in size, occurred simultaneously during the fever episode. She had her first visit to the hospital a week after her symptoms and was discharged with antibiotics, but the symptoms persisted. She denied any chest infection symptoms, no skin rashes, no joint pain, and no evidence of other connective tissue disease symptoms. Upon review in our clinics, she clinically looks slightly lethargic but well-hydrated. The vital signs were normal, with a blood pressure of 106/78, pulse rate of 98, oxygen saturation of 98% under room air, temperature of 37.2 and respiratory rate of 18. There was a presence of multiple matted cervical lymph nodes bilaterally starting from submental and extending to the posterior triangle, non-tender and not inflamed. Both axillary and inguinal lymph nodes were also palpable. Abdominal palpations revealed liver enlargement about two-finger breaths and the enlargement was confirmed by a bedside ultrasound scan measuring 19cm of craniocaudal length. Other systems were unremarkable. Blood investigations showed normochromic normocytic anemia with hemoglobin of 9.4 and elevated lactate dehydrogenase (LDH) 641 U/L. Other blood investigations and chest radiographs were normal. High index suspicion towards lymphoma was made in view of constitutional symptoms with generalized lymphadenopathy without any evidence of tuberculosis or connective tissue disease. The patient was then referred to the department of surgical for an excisional biopsy.

Two weeks after her first visit, she was electively admitted under Surgical ward for excisional biopsy, however the procedure was abandoned due to resolutions of the size of the lymph nodes. Furthermore, she started to develop bilateral hand myositis, which is suggestive of rare musculoskeletal manifestations of lupus. She complained of bilateral arm swelling associated with limited movement of her elbow, hands and wrist due to pain. Physical examinations showed evidence of inflammations of both arms, no evidence of joint inflammations and radiographic findings were normal. A diagnosis of bilateral arm myositis was made after being reviewed by the orthopedics team. Upon further questioning, noticed she developed oral ulcers, alopecia, and new onset of neuropsychiatric manifestations in the ward. Forgetfulness, and behavioral changes followed by visual and auditory hallucinations raised further clinical suspicions towards SLE. She was transferred to the medical ward. Initial computed tomography brain showed no brain abnormalities, but the magnetic resonance imaging (MRI) brain showed generalized cerebral atrophy with focal bilateral white matter changes and internal cerebral artery vasculitis suggestive of central nerve system of SLE features.

Blood investigations (**Table 1** and **Table 2**) taken showed worsening anemia with hemoglobin reduction from 9.4 to 6.8. The presence of positive direct coombs tests specifically anti-IgG 2, suggestive of autoimmune hemolytic anemia (AIHA), indicates hematological manifestations of SLE. Urine analysis was suggestive of persistent proteinuria as manifestation of lupus nephritis. She also developed persistent liver transaminitis inward autoimmune hepatitis workups done were negative. Thus, the diagnosis of lupus hepatitis was made based on clinical diagnosis. After one month from the initial presentations, the final diagnosis of SLE with multiorgan involvement was made based on the presence of

Table 1. Improvements in blood parameters after completed intravenous cyclophosphamide

Test	FRC	ATW	OA	OT	CT	NR
Full blood count						
WCC	4.9	4.7	7.63	5.95	2.76	4.0-11.0 (10^9/l)
Hb	9.4	8.1	6.8	9.9	11.6	12.0-16.0 (g/dl)
Hct	29.7	24.7	21.3	31.8	37.9	37-47 (%)
Platelet	250	219	520	318	229	150-400 (10^9/l)
CRP	11	-	39	-	-	< 5 (mg/l)
Renal profile						
Urea	1.8	2.8	4.4	3.3	3.0	2.5-7.8 (mmol/l)
Na	138	134	140	135	142	135-145 (mmol/l)
K	3.7	4.1	4.2	4.0	3.1	3.5-5.1 (mmol/l)
Creatinine	50	63	47	54	57	45-90 (umol/l)
Uric acid	327	-	-	-	-	140-360 (umol/l)
LDH	641	507	-	-	-	135-225 (u/l)
Liver function test						
AST	21	326	200	80	25	10-40 (u/l)
ALT	26	232	197	140	18	10-40 (u/l)
ALP	75	191	115	144	61	44-147 (u/l)
Viral screening						
Hepatitis B			Non-reactive			
Hepatitis C			Non-reactive			
Thyroid function test						
TSH	1.38					0.4-4.2 (mg/dl)
Ft4	16.8					12-22 (pmol/l)

Note. FRC: First review in clinic; ATW: After two weeks; OA: On admission; OT: On treatment; CT: Completed treatment; NR: Normal range; WCC: White cell count; Hb: Hemoglobin; Hct: Hematocrit; CRP: C-reactive protein; AST: Aspartate aminotransferase; ALT: Alanine aminotransferase; ALP: Alkaline phosphatase; TSH: Thyroid stimulating hormone; & Ft4: Free thyroxine

Table 2. Findings of autoimmune investigations and full blood count

Blood investigations	Autoimmune investigations done in ward
ANA	Positive titer 1:320
Anti-DsDNA	< 50
Complement	C3: 0.27 & C4: 0.05
Rheumatoid factor	Negative
ASMA	Negative
AMA	Negative
Anticardiolipin antibody	Negative
Urine protein	3+
Urine PCR	144
IFOBT	Positive
Direct coombs test	Positive, anti-IgG 2+, anti C3d: 0, & suggest: ğresence of AIHA as haematological symptoms
Full blood picture	Presences of atypical lymphocytes to exclude viral infections. Unable to exclude bone marrow pathology or hematological malignancy

Note. Anti-DsDNA: Anti-double-stranded DNA; C3: Complement components 3; C4: Complement components 4; RF: Rheumatoid factor; ASMA: Anti-smooth muscle antibody; IFOBT: Immunochemical fecal occult blood test; PCR: Protein-to-creatinine ratio; & IgG: Immunoglobulin G

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

↓

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.

I.

Figure 1. 2019 EULAR/ACR classification criteria for SLE (Reproduced from [2019 European League Against Rheumatism/American College of Rheumatology, Classification Criteria for Systemic Lupus Erythematosus, Aringer et al.,79,1151-59,2019] with permission from BMJ Publishing Group ltd)

hematological, lupus nephritis, musculoskeletal, and neuropsychiatric involvement with positive ANA with low complement levels.

She was started on IV methylprednisolone 500 mg for five days, followed by IV hydrocortisone 100 mg and subsequently changed to prednisolone prior to discharge. In view of severe disease with multiorgan involvement, she was also treated with six cycles of cyclophosphamide. The latest medication for discharge was prednisolone, hydroxychloroquine and azathioprine. She showed obvious improvement after the medications.

DISCUSSION

Diagnosis of SLE based on specific criteria from EULAR/ACR 2019. It requires the presence of positive ANA plus a total score of 10 or more from additional criteria [2]. The additional criteria consist of seven clinical criteria and three immunological criteria (**Figure 1**). Our patient was diagnosed with SLE because she fulfilled the entry criterion of SLE. She had positive ANA titer, low complements level, persistent proteinuria that suggest lupus nephritis, AIHA as hematological symptoms of SLE and having evidence of neuropsychiatric lupus based on MRI. However, the diagnosis made was delayed and treatment was started only after one month of her first visit due to initial misdiagnosis.

The delay in diagnosis was because of our low suspicion towards SLE. Our patient was presented with isolated symptoms of lymphadenopathy without any clue that indicates SLE at her first visit. The main complaint of prolonged fever, malaise and unexplained weight loss with matted lymphadenopathy at the cervical, axillary, and inguinal regions raised more suspicions of lymphoma than SLE. The reason why our suspicions towards lymphoma were stronger than in SLE is because generalized lymphadenopathy is more common in

lymphoma. It was detectable in more than two thirds of patients with lymphoma, especially in classic hodgkin lymphoma. Neck is the most common site of involvement accounting for 60% to 80% of total prevalence. Enlargement of axillary lymph nodes was found in 30% of patients while inguinal lymphadenopathy accounted for about 10% of patients [4]. The presence of B symptoms specifically refers to fever, night sweats, or weight loss accounted for 20% of patients with stage I and II of lymphoma [4]. Other additional findings such as hepatomegaly and raised LDH levels might also increase higher suspicions towards lymphoma. In one study, 6 of 421 consecutive patients who were diagnosed with hodgkin lymphoma were presented with liver abnormalities, including cholestasis and moderate hepatomegaly [6]. The combination of all our patient symptoms strongly suggests a higher suspicion towards lymphoma than in SLE.

As compared to lymphoma, the presence of lymphadenopathy in SLE is rare but if present, always concomitant with SLE symptoms such as fever, serositis, and mucocutaneous [7]. In one study done in Spain involving 103 SLE patients, 27% were found to have lymphadenopathy. 60% of them were accompanied by fever, 86% of them had dermopathy and 45% of them had serositis symptoms [7]. One case study done in America also reported lymphadenopathy as the first manifestations of SLE. Smith et al reported on his paper in 2013, regarding a case with initial presentations of generalized lymphadenopathy without any SLE symptoms. Multiple work up done and his clinical suspicious at that time was lymphoproliferative malignancy. However, from the biopsy taken, the findings didn't fit any of hodgkin or non-hodgkin lymphoma classifications. Diagnosis of SLE was only made after 6 months of the initial manifestations as she slowly developed typical SLE symptoms [8]. Subsequently the patient responded well after the SLE treatments. This case shared the same initial presentations as in my case report. Both of our cases had initial isolated lymphadenopathy as our first clinical presentations whereas the true SLE symptoms developed after a few weeks.

Our patient is unique because despite being presented with lymphadenopathy as the initial manifestation, she also had multiple atypical and unspecific symptoms of SLE, which made the diagnosis of SLE even harder. Two weeks after her first presentation, she was diagnosed with bilateral arm myositis in ward. The prevalence of myositis in SLE was rare. Based on one study done in Texas, among 1718 SLE patients, only 104 (6.3%) had myositis symptoms, while 90% had arthritis symptoms [9]. Due to the low prevalence of myositis in lupus, it is not classified as a diagnostic criterion for SLE. This rare myositis presentation of lupus is also a big challenge for inexperienced doctors because the symptoms are often misdiagnosed as soft tissue injury. Without the presence of other SLE symptoms, the misdiagnosis may lead to another delay in the diagnosis and treatment of SLE.

Our patient also had clinical symptoms of lupus hepatitis. She presented with fatigue, malaise, weight loss, and the presence of hepatomegaly. Her liver enzyme derangement is within the fifth to tenth upper limit normal, and all the viral hepatitis and autoimmune hepatitis workups were negative. After exclusions of other causes, we came out with a clinical diagnosis of lupus hepatitis. The liver derangement showed significant improvement after corticosteroid and cyclophosphamide therapy. Based on the literature, the prevalence of liver involvement in SLE accounted for up to 50% and is usually related to SLE itself or due to its treatment. Only about 3% to 8% of them were related to lupus hepatitis [10]. Clinical manifestations of lupus hepatitis include fatigue, anorexia, malaise, and nausea. Hepatomegaly and splenomegaly might be present in some patients [10, 11]. The typical liver derangement in lupus hepatitis is usually mild to moderate, with the elevation of liver enzyme about five to ten times from upper limit normal. The presence of ribosomal P autoantibodies may differentiate lupus hepatitis from other causes of liver involvement, but some patients with autoimmune hepatitis were also positive in this autoantibody [10, 12]. In our patient, we only took anti-mitochondrial antibody (AMA) to exclude autoimmune hepatitis. Histopathology in lupus hepatitis was essentially nonspecific and diverse. Because of that, the diagnosis of lupus hepatitis is usually made based on exclusions criteria after ruling out other causes of hepatitis, such as viral hepatitis, drug-induced hepatitis, metabolic disease or autoimmune hepatitis. Lupus hepatitis, even uncommon, must be considered in all patients with SLE with liver derangement.

The effects of pregnancy on SLE have been debated in the literature, but a majority of studies reported that the disease activity was increased during pregnancy. They believed that high estrogen levels in pregnancy promote physiological and immunological changes associated with increased lupus activity [13]. Patient with underlying SLE at time of preconception has a higher risk of flare with increment rates of 25-65% [14]. However, the flares were not severe. Cutaneous reactions, arthritis, and hematological symptoms are the most commonly reported in most studies [13]. Even if the flares were mild, the pregnancy complications can lead to life-threatening events. Maternal complications include high risk of hypertension, preeclampsia and renal complications while neonatal complications may include intrauterine growth restrictions, preterm birth, miscarriages as well as neonatal lupus and neonatal heart block [15, 16].

On the other hand, the incidence of new onset of SLE in pregnancy or postpartum patients is rare. Based on one study done in Hong Kong, the prevalence of new onset of SLE in pregnancy accounted for 0.014/1,000 person per year. This study stated that, among 742 SLE patients in this cohort sample, 15 patients were diagnosed during pregnancy with 13% of them diagnosed during the first trimester, 47% in the second trimester and 47% in the third trimester [17]. Common initial presentation of SLE includes renal disease, thrombocytopenia and central nerve system involvement [17]. There was also a review reported on prevalence of new onset of SLE among port partum patients. Among 16 cases of SLE patients, 13 of them were diagnosed after delivery with the onset of symptoms starting between day one after delivery up till one year after delivery [18]. The mean age was 28 years old and pre-eclampsia was found in 11 (65%) of patients. Hemophagocytic syndrome, acute fatty liver, HELLP (hemolysis, elevated liver enzymes, and low platelet count) syndrome, and myocardial infarction were the other severe complications [18]. Our patient had her first onset of SLE symptoms after one month postpartum, whereas she was healthy throughout her pregnancy. The baby was unaffected. She was able to deliver a vigorous baby boy via spontaneous delivery without any complications. She never had any episode of anemia, thrombocytopenia, renal disease or preeclampsia during her antenatal period. Whether or not her pregnancy was a risk factor for her disease remains unknown.

CONCLUSION

Atypical presentation of SLE was a big diagnostic challenge especially in primary care doctors. Generalized lymphadenopathy can be represented in many diseases rather than in SLE. Infections, inflammations, and malignancies were common diseases related to lymphadenopathy as compared to SLE itself. Although it can be presented in SLE, the prevalence is low and usually accompanied by SLE symptoms. Without apparent symptoms, the diagnosis of SLE might be delayed or missed. Early recognition and proper comprehensive evaluation should be made. High index suspicions towards SLE should be made in all patients with lymphadenopathy especially among females, within reproductive age to avoid misdiagnosis or delay in the treatment.

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