

Generalized Lymphadenopathy as the Initial Presentation of a Young Woman with Systemic Lupus Erythematosus

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Background: Generalized lymphadenopathy (LAP) refers to abnormal enlargement of more than two non-contiguous lymph node regions. There are various causes of LAP, including malignancy, infection, autoimmune disorders, medications, and iatrogenic causes. Obtaining a thorough history and physical examination is paramount in identifying the underlying etiology. Most of the time further investigation with laboratory and radiographic studies should be performed to identify the cause. Patients with high-risk features should undergo a biopsy for the diagnosis of malignancy.

Case presentation: A 36-year-old lady with a remote history of COVID-19 presented with complaints of orthopnea, cough, arthralgia, and left-sided abdominal pain for the past two months. She denied associated B symptoms. On physical examination, her vitals were unremarkable; however, she had round, coin-shaped lesions over her MCPs and abdomen, alopecia, and generalized LAP. The initial workup was remarkable for anemia and leukopenia. CT scan of the abdomen and chest revealed bilateral inguinal, iliac, axillary, mediastinal, hilar, and retroperitoneal lymphadenopathy with bilateral pleural effusions. Due to malignancy suspicion a bone marrow biopsy was pursued, which demonstrated leukopenia, no circulating blasts, and moderate normocytic anemia. Then the patient underwent an excisional biopsy of 3 lymph nodes that showed reactive follicular hyperplasia without malignancy. Infectious workup including blood cultures, respiratory cultures, HIV, hepatitis panel, Bartonella, Brucella, and tuberculosis was negative. Due to the evidence of leukopenia, anemia, serositis, lymphadenopathies, and polyarthritis, a diagnosis of systemic lupus erythematosus (SLE) was entertained. Further workup revealed ANA by immunofluorescence of 1:1280, anti-dsDNA 38, ESR 75 mm/hr, anti-U1RNP 40, C-reactive protein 1.4, complement C3 48, and complement C4 less than 5. Based on these findings, a diagnosis of SLE was made. The patient was discharged on prednisone 40 mg and hydroxychloroquine 200 mg daily and had remarkable improvement.

Conclusions: The prevalence of LAP in SLE ranges anywhere between 30-59% depending on the study. Although it is a common manifestation, it is not considered a criterion for the diagnosis of SLE. The presence of LAP typically correlates with a higher level of disease activity. This case highlights the importance of considering connective tissue diseases in the differential of patients presenting with LAP.