

A Rare Association Between SLE and Acute Lymphatic Leukemia

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Abstract:

A 30-year-old housewife with past history of acute lymphoblastic leukemia 12 years back, still in remission, was admitted with polyarthritis of 2 months duration. She was evaluated and found to have SLE with positive ANA and Anti ds DNA which were strongly positive

Case Report

A rare association between SLE and ALL

Introduction

Systemic lupus erythematosus (SLE) is an autoimmune disease in which organs, tissues, and cells undergo damage mediated by tissue-binding autoantibodies and immune complexes. It is associated with an increased risk of lymphoreticular malignancy, especially lymphoma. The association between SLE and acute lymphoblastic leukemia is also described, but rare. We report a lady with past history of ALL now presenting as SLE.

Case summary

30-year-old house wife, was admitted with symmetrical polyarthritis involving small and large joints of 2 months duration. There was no axial joint involvement. She also had associated low-grade fever and breathlessness with wheezing. She was admitted in our hospital 12 years back with history of anemia, generalized lymphadenopathy and hepatosplenomegaly and was diagnosed as ALL-L1 for which she received a full course chemotherapy and cranial irradiation with standard protocol and attained complete remission and also completed the maintenance treatment. During chemotherapy she had pulmonary tuberculosis and fungal pneumonia, both of which got cured with treatment. She had polyarthritis 9 years back, which was treated as rheumatic fever with aspirin and penicillin prophylaxis. She also had bronchial asthma in childhood.

At the time of admission this time she was conscious oriented, pallor was present, pulse-100/mnt, blood pressure -110/70 mm of Hg, temperature-100°F, respiratory rate was 24/mt, no lymphadenopathy. She had symmetrical erythematous swollen joints with tenderness involving knee, ankle, elbow, wrist and small joints of hand with no deformities. Abdomen examination revealed mild splenomegaly.

There was bilateral rhonchi; Cardiovascular and nervous system examination were within normal limits.

Her routine blood investigations showed Hb-10.4g%; total count was 42,200/cmm with 93% polymorphs and 4 % lymphocytes. Platelet count was 69,000/cmm. B.urea -39mg; S.creatinine-0.7mg; Na-132mEq/L; k-3.4 mEq/L, bilirubin-0.8 mg; direct bilirubin 0.3 mg; SGOT-28 IU/L; SGPT-14 IU/L; ALP-206 IU/L; LDH- 897; CRP-192(Normal<6) , ASO titer<200 IU/L; RA factor-Negative ,Urine routine examination showed 2+ albumin and 24 hour urinary protein was 175mg. Her chest X-ray was normal. On further evaluation ANA was positive and anti ds DNA was strongly positive 344 IU/mL (normal<117 IU/mL). Possibility of relapse of ALL was ruled out with a normal bone marrow and peripheral smear, which showed only, marked neutrophilia with shift to left but not excess blasts. A normal echocardiogram ruled out any cardiac abnormality.

She was treated with intravenous steroids, cefotaxim , bronchodilators, indomethacin, hydroxy chloroquine and supportive measures. Patient responded well to the treatment.

Discussion

SLE is a disease of unknown etiology but is obviously due to multiple factors; caused by interactions between susceptibility genes and environmental factors, resulting in abnormal immune responses. It is associated with an increased risk of lymphoreticular malignancy among which lymphomas are the commonest ^{1,2,3,4}. Association with myelomas and leukemias are also described. Usually SLE precedes the onset of lymphoproliferative diseases, but it can occur simultaneously or after the occurrence of malignancy⁵. Two cases have been described of SLE appearing a few years after complete remission of acute lymphoblastic leukemia ⁶.



Various mechanisms have been postulated to explain the association between SLE and ALL. Both these diseases could simply be different expressions of the same immunological disorders, although ALL is not generally associated with autoantibody formation, children with ALL have been found with positive test results for ANA in conjunction with clinical features of SLE⁷. Other hypotheses include a common stimulus, such as a viral infection in a genetically or environmentally susceptible host, facilitating neoplastic process by the autoimmune disorder and suppression of immune surveillance by cytotoxic therapy. Lymphocytes from patients with SLE exhibit increased expression of proto-oncogenes *c-myc* and *c-myb*, transcripts of which are found in large amounts in lymphoid tumors⁸. Activation of these oncogenes can initiate neoplasia. All the previous reports are of cases of SLE developing lymphoreticular malignancy later or SLE coexisting with them. In the present case SLE appeared almost twelve years after chemotherapy it could be even unrelated or more likely that the chemotherapeutic agents or radiotherapy had some way contributed to triggering of the autoimmune phenomena. This could also suggest etiological relation to toxins and radiation in pathogenesis of SLE. Association with other lymphoreticular malignancies like lymphomas(4,5,6,7), myelomas and leukemias are also described. There have been also reports of SLE occurring after thymectomy or thymomectomy or after chemotherapy and radiotherapy for malignant thymoma [8].

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