CASE REPORT

AMIODARONE INDUCED SYSTEMIC LUPUS ERYTHEMATOSUS – A CASE REPORT

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Abstract : *Drug inducted lupus (DIL) is an uncommon entity. Various drugs like hydralazine, procainamide, isoniazid, D-penicillamin, practalol, methyldopa, alpha-interferon, quinidine, phenytoin, oral contraceptives and ethosuximide etc. have been reported to induce lupus. But there are very few reports in the world literature regarding amiodarone induced systemic lupus erythematous (SLE). A case of hypertension and atrial fibrillation in a 70 year old male patient treated with digitalis, amiloride and amiodarone (200 mg twice daily) for the last two years in being reported for its rarity.*

INTRODUCTION

Amadarone induced SLE is uncommon in India. On extensive review of Indian medical literature, only one case of amadarone induced SLE could be found on repeated attempts and that too in a male patient. Probably, this could be the second case of amedorone-induced SLE from this part of India. Hence the case report.

CASE REPORT

A 70 years old male patient presented with complaints of intermittent fever (39°C), dyspnoea on exertion, non productive cough and pleuritic chest pain since 3 weeks. He also had history of weight loss, loss of appetite, weakness and malaise for more than 2 months. He was a known case of hypertension and atrial fibrillation treated with amiodarone given 200 mg two times daily, amiloride and digitalis for the past two years. There was no history of Raynand's phenomenon, oral ulcers and photosensitivity. Physical examination revealed pulse rate 80/min, regular of normal and equal volume in all the four limbs, BP 150/92 mmHg in the supine position, temperature 39°C, respiration rate 24/min. Malar rash and mild anaemia was also noticed. On auscultation of cardivascular system, an aortic systolic murmur was heard (Grade II/VI). The examination of other systems was not contributory. Investigations revealed Hb 9.3 g/dl, normocytic-normochromic, anaemia, TLC 3900/mm^3, DLC-P-$_{68}$, L_{24}, M_4, E_4, ESR 110 mm/first hour, platelet count 190000/ml, MP slide –ve and urine C/E NAD. B.urea, S.creatinine, fasting blood sugar, serum bilirubin were within normal limits. Widal test, ECG, coagulation profile, Mantoux test, Coomb's test, C reative protein, VDRL, ELIZA test for HIV and M.Tuberculosis as well as T_3, T_4, TSH were non contributory. X-ray chest revealed small bilateral pleural effusion without fibrosis or cardiomegaly. The pleural fluid on aspiration was exudative with lymphocytic predominance without cytological evidence of malignancy. Cultures of pleural fluid for bacteria, including for M.tuberculosis, blood and urine cultures were negative. 2-D echocardiogram showed mild aortic stenosis. The histopathological examination of biopsy specimens of the skin, including immunoflurescence stain, muscle and temporal artery did not show any abnormality. In view of the sympatomatology and laboratory findings, the patient was subjected to total collagen profile. The results showed: rheumatoid factor +ve 1:320, antinuclear factor (ANF)+ve 1: 640, circulating immune complexes (IgG-C1q)+ve but other autoantibody tests were negative.

A diagnosis of amadarone-induced lupus was considered. The amadarone was stopped and the patient started improving progressively. No corticosteroids were given. After one year, the patient was symptom free without any clinical, analytical or radiological findings of lupus. The patient on further follow up after one year and five months, remained free of symptoms while ESR, TLC, DLC and radiological profile were normal. The titre of ANA decreased but remained weakly positive at 1:40. The patient is now on digitalis, amiloride and is totally asymptomatic.

DISCUSSION

A syndrome resembling SLE may be induced by various drugs like hydralazine, procainamide, isoniazid, D-penicillamin, practolol, methyldopa, alpha-interferon, quinidine, phenytoin, oral contraceptive and ethosuxamine[1]. Only few cases have been reported in the world literature. The first case was reported by Susano R et al in 1992[2] and the second case by Sheikh Zadeh A et al in 2002[3]. In the west, it is estimated that 3-7% of all patients of SLE might have drug induced lupus (DIL)[4,5]. Clinically it is difficult to differentiate between SLE and DIL, but there are certain distinguishing features between them: the patient with DIL is usually older; the prevalence of male and female is equal; and the common presenting symptoms are usually mild with the patient usually complaining of malaise, fever, arthralgia with or without arthritis whereas involvement of skin, renal and central nervous system is rare. Pleuropericardial disease is frequent and as in classic SLE, anaemia and leucopenia may be present. Serum complement levels are usually normal. ANF is positive, anti- dS DNA and anti - SM antibodies are negative while antihistone-antibodies are positive in majority of the patients[6]. Amiodarone induced side effects range from 40-93% and are in the form of nausea, vomiting, hepatitis, alveolitis, photosensitivity, pulmonary fibrosis, microdeposits in cornea, bluish skin, peripheral neuropathy, bradycardia, Q-T prolongation and thyroid function abnormalities[4].

The exact pathogenesis of DIL is not known but could be cross reactivity between the drug and nucleic acid, hapten complex formation between drug and nucleic acid, or structural damage to the chromosomal DNA, action of drug as an adjuvant or immunostimulant, which in concert with appropriate immune response genes, triggers polyclonal B/T cell activation, and interference with the compliment pathway[5]. This case, presenting with fever, malaise arthralgia, circulating immune complexes, and autoantibodies strongly suggests an immunological underlying condition. Moreover, according to 1982, American Rheumatism Association revised criteria for the classification of SLE[7], this patient meets four SLE criteria, i.e. malar rash, serositis, leucopenia and lymphopenia as well as positive ANF.

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