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Case Report

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CARDIAC INVOLVEMENT IN SYSTEMIC LUPUS ERYTHEMATOSUS: A CASE REPORT

Madiha Nooreen*[1], Saima Aziz[2], Ayesha Habeeb[1] and Shafia Fatima[1]

*[1]Pharm. D, Department of Pharmacy Prcatice, Deccan School of Pharmacy.
[2]Assistant Professor, Department of Physiology, Ayan Medical College.

***Corresponding Author: Madiha Nooreen**

Pharm. D, Department of Pharmacy Prcatice, Deccan School of Pharmacy.

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ABSTRACT

Systemic lupus erythematosus (SLE) is an autoimmune disease which can affect many organs. Cardiac involvement in the systemic lupus erythematosus is one of the dominant complications, significantly responsible for morbidity and mortality of the patients. Lupus myocarditis rarely presents as an initial complication of SLE. The patient may present with wide array of symptoms ranging from being asymptomatic and self limiting disease to acute cardiac failure that can lead to death. The information available on clinical manifestations and outcomes of myocarditis in SLE is scarce. The authors present the case of 27 year old woman diagnosed with SLE and later developed myocarditis.

KEYWORDS: Lupus Erythematosus, Systemic, Lupus Myocarditis, Myocarditis, Pleural Effusion, Autoimmune Disease.

INTRODUCTION

'Systemic lupus erythematosus (SLE) is an autoimmune disease in which the immune system makes antibodies to cells in the body leading to widespread inflammation and tissue damage'.[1] It has been reported that cardiac manifestations of SLE have an incidence rate of more than 50%.[2,3] It can affect all cardiac structures, including pericardium, myocardium, endocardium, valves and coronary arteries. Most commonly affected structure is pericardium, however, myocardium is involved in cardiac manifestations in about 10% of cases.[3] Post mortem studies from 1950s and 1960s, showed the presence of subclinical myocardial involvement in 57% of total studies.[4]

Lupus myocarditis(LM) can be because of the drugs, comorbidities and the autoimmunity. LM can be asymptomatic or life threatening leading to sudden death. The disease can progress to cause arrhythmias, conduction abnormalities, dilated cardiomyopathy or heart failure which may vary in severity, thus requires urgent clinical attention.

Myocardial necrosis serum markers, imaging studies and endomyocardial biopsy can be used to diagnose the myocardial involvement. Treatment is usually to provide supportive therapy, but the severe cases requires mechanical hemodynamic support and even transplantation of heart.[3]

The authors present the case of systemic lupus erythematosus that was complicated due to the development of myocarditis. Since, lupus myocarditis rarely presents as initial presentation of SLE and thus, the literature on clinical manifestations and outcomes of LM is inadequate.

CASE REPORT

A 27 year old female patient was presented to the general ward of tertiary care hospital with complaints of intermittent fever (102 F) associated with chills, body pain, cough and stabbing pain around sternal border since 3-4 days which aggravated on lying down. Chest was productive expectorant on lying but normal at exertion. She also had generalized weakness, pallor, pedal edema and decreased movement of finger joints along with reduced skin pinch ability and sclerodactyly. She was taking ayurvedic medication since 1 year and had complaints of increased hair loss.

On taking personal history, it was revealed that she is non-alcoholic, non-smoker, had decreased appetite and disturbed sleep since a year. She had alternating events of diarrhea and constipation, about 2-3 episodes since 24 hours.

Physical examination showed the patient to be conscious, alert and cooperative, pallor, pedal edema, shortness of breath (NYHA grade III), photosensitivity, skin pigmentation on wrist and hands and blood pressure of 90/60 mmHg. Cardiac auscultation revealed a regular

heart beat with no murmur and a heart rate of 84 beats/min. Respiratory auscultation revealed symmetrical breath sounds, bibasilar inspiratory rales with a respiratory rate of 20 breaths/min. Her abdomen was soft, regular with normal bowel sounds.

Initial laboratory investigations revealed neutrophilic leukocytosis (12,200 / cumm), normocytic hypochromic red blood cells (3.9 millions / cumm), adequate platelets (2.87 lakhs / cumm), hemoglobin 9 mg/dl, positive antinuclear antibodies with speckled pattern (32.426 ng/L), positive SS-A/ Ro52KD, U1 SnRNP. Troponin I was reported to be highly sensitive (96 ng/L) and ESR levels were raised at about 120mm at 1 hour and 135 mm at 2nd hour. Other lab investigations liver biomarkers, thyroid profile, renal function tests and C reactive protein are within normal range.

Chest X ray showed fibrotic patches. ECG showed sinus rhythm with heart rate 84 beats/min but on day 2, sinus tachycardia with pulse rate of 110/min due to atrial fibrillation was reported. 2D Echocardiography revealed mild mitral regurgitation (Grade 1) along with mild tricuspid regurgitation, mild pulmonary hypertension (RVSP; 45 mmHg) and global hypokinesia of LV. No features suggested pulmonary embolism or clot and left ventricular function with ejection fraction of 53%.

Based on the above findings, clinical suspicion of systemic lupus erythematosus along with lupus myocarditis was made.

Initially, the patient was started on IV paracetamol 1gm, IV pantoprazole 40 mg OD, syrup ambroxol (secretolytic agent) 5ml orally BD, tab. Paracetamol 650 mg TD and tepid sponging, inj. Ampicillin and sulbactam 1.5mg IV TID (1/0.5 gm). On day 2, she was given FDC tablets of aspirin (75 mg), clopidogrel (75 mg), rosuvastatin (10 mg) once daily and acetaminophen (325 mg), tramadol (37.5 mg) twice daily along with tab. Atorvastatin 10 mg OD and tab. Metoprolol 12.5 mg BD. On day 3, she was started on tab. methotrexate 7.5 mg once weekly and tab. Hydroxychloroquine 200 mg BD.

On second day hospital admission, an episode of tachyarrhythmia due to atrial fibrillation with ventricular rate of 110/min, which resolved after administration of metoprolol. On day 3, clinical improvement was reported with no signs of orthopnea, fever, BP 110/80 mmHg, HR of 82 beats/min with murmur on pulmonary auscultation, reduced edema.

In view of definite diagnosis made, improvement of constitutional symptoms and relative's request, she was discharged on day 5. On discharge she was advised to continue DMARD's, dual antiplatelet therapy, prescribed antibiotics and proton pump inhibitor. Patient was under follow up and after 1 month of treatment, her lesions has regressed.

DISCUSSION

SLE is an autoimmune disease typified by extensive inflammation of blood vessels and connective tissue. It can affect people of all ages from infants to geriatrics, and women, particularly of childbearing age are more commonly affected than males (4-12 females for every male). The etiological factors are indefinite but genetic, environmental and hormonal factors are known to play prominent roles. The disease has periods of remission and relapse. It includes wide assortment of signs and symptoms that represent the disease clinically like malar rash, photosensitivity, oral ulcers, arthritis, lung problems, heart problems, kidney problems, seizures and psychosis, blood cell abnormalities. Patient may complain of fatigue, pain or swelling in joints, skin rashes and fevers.[5] Cardiac manifestations may differ in different cases, approximately 10% of cases in clinical studies has reported myocarditis in SLE.[1]

In our case the diagnosis of SLE has been made based on 1997 revised American College of Rheumatology (ACR) criteria for classification of SLE such as photosensitivity, nonerosive arthritis, positive ANA and pleuritis.

Myocarditis can manifest as dyspnea, tachycardia, arrhythmias and can advance to ventricular dysfunction, cardiomyopathy and heart failure. Myocarditis diagnosis can be made based on clinical condition which is well supported by laboratory investigations like leukocytosis, elevated ESR levels, and raised troponin T and I along with echocardiography. ECG and cardiac markers may appear to be normal. Endomyocardial biopsy is considered as standard test to diagnose lupus myocarditis but being an invasive procedure its diagnostic yield is approximately 10-20% with low specificity and sensitivity. Thus, echocardiography along with index of suspicion is utilitarian in diagnosing myocarditis associated with SLE.[6] Myocarditis associated echocardiographic consequences are as follows: reduced ejection fraction, increased chamber size, prolonged isovolumic relaxation time, decreased diastolic descent rate of anterior mitral leaflet, decreased ratio of mean systolic velocity to mean diastolic velocity in left ventricular posterior wall, decreased declaration of early diastolic flow velocity, reduced e/a ratio and atrial ejection force.[6,7] Some cases has demonstrated the relationship between anti-SSA/Ro antibodies and myocarditis.

The present case is of a woman who didn't have any risk factors for atherosclerosis. The subject had mildly enlarged LA (40 mm), global hypokinesia of LV with reduced ejection fraction of 53%, mild pulmonary hypertension (RVSP; 45 mmHg) and mild mitral regurgitation (Grade 1) along with mild tricuspid regurgitation.

Acute lupus myocarditis demands urgent clinical attention. The treatment of SLE should be individualized and tailored according to the disease severity. In lupus

myocarditis, immunosuppressants are used to improve the systolic function. In severe form of disease, intravenous pulses of methylprednisolone administered for three days, thereafter prednisolone (1 mg/kg/d) is given intravenously or orally with gradual tapering of dose.[8] Cyclophosphamide, azathioprine, mycophenolate mofetil, should also be added.[9] Certain cases has also concluded propitious role of gamma globulin in steroid refractory LM.[10] Angiotensin converting enzyme inhibitors, diuretics and β-adrenergic blocking agents, should be added as supportive therapy for LV dysfunction.[11] Echocardiographic findings gets improved during follow up period in most of the patients. However, majority of patients reveals improvement in myocardial damage during the 6 month period post immunomodulatory therapy.[12] Clinically overt myocarditis has shown higher mortality rate than subclinical form of disease. Myocarditis in SLE indicates poor disease activity.[13] LM can show spontaneous recovery in short period of time without administration of immunosuppressant therapy but on long run uncommon dilated cardiomyopathy (DCM) with left ventricular dysfunction appears to be serious complication of myocarditis which may even lead to death. Heart transplantation is considered as the final resort for chronic myocarditis with recurrent heart failure.[14]

This case suggests lupus related myocarditis, mild form of disease can be managed with the supportive therapy for LV function i.e., β-adrenergic blocking agent and the prognosis of the subject is consistent with the studies that has encouraged the use of antimalarial drugs in appropriate patients as they have steroid sparing effects.[15]

CONCLUSION

Myocarditis is the most evident feature of cardiac involvement in SLE. The disease can progress to ventricular dysfunction, dilated cardiomyopathy and heart failure. It may appear as the first manifestation of disease or can occur during follow up. Echocardiographic results may help in definite diagnosis of LM. Treatment with immunosuppressive agents helps to improve the cardiac outcomes in LM, however, mild form of disease shows positive prognosis even without the administration of potent drugs like cyclophosphamide. Patients with dormant disease are less likely to experience exacerbations when they are maintained on the hydroxychloroquine.

CONFLICT OF INTEREST

Authors have no conflict of interest.

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