Cardiac Involvement in Patients with Systemic Lupus Erythematosus

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Abstract

Background: The aim this study was to analyze the patterns of involvement of the heart in our patients with SLE by means of clinical evaluation, electrocardiography, and echocardiography.

Methods: Forty six consecutive patients (40 females and 6 males) with systemic lupus erythematosus (SLE) were studied for cardiac manifestations of the disease. Clinical history, physical examination, electrocardiography, chest X ray ,echocardiography study and standard laboratory tests were performed .Thirty three patients (72%) have evidence of cardiac involvement .

Results: Thirty one patients (67%) were found to have abnormal echocardiography findings. Reduction in shortening fraction which reflects reduction in left ventricular function was the commonest abnormality. It was present in 15 patients (33%). Endocardial involvement in form of valvular dysfunction with or with out thickening was found in 10 patients (22%).

Conclusion: The heart is a common target for systemic lupus erythematosus. Therefore, echocardiography should be performed periodically in SLE patients.

Key words: lupus erythematosus, heart, echocardiography.

Introduction

Systemic lupus erythematosus (SLE)is a multisystem connective tissue disease characterized by the presence of numerous autoantibodies ,circulating immune complexes, and wide spread of immunologically determined tissue damage.[1] The earliest description of SLE were as cutaneous disease only.[2] Osler in1895 and 1904 noted endocarditis and pericarditis as part of recurrent manifestation of an exudative erythema group of diseases. Libman and Sacks in 1924 brought attention to the cardiac manifestation of lupus with their description of four cases of nonbacterial "verrucous" endocarditis. [2] Gross in 1932 and 1940 pointed out the significance of pericardial and myocardial lesions associated with this syndrome.[2]

Cardiac manifestations are common in lupus patients and include pericarditis , myocarditis ,endocarditis and coronary artery disease.[3] The most characteristic finding is endocardial involvement as described by Libman and Sacks as bland verrucous endocarditis affecting predominantly aterioventricular valves of the left side.[3] They occur in the forward direction of blood flow and in contrast to rheumatic heart diseases were situated away from the line of closure to involve the most distal part of chordae tendinea, papillary muscle and mural endocardium.[4]

The type of lesion is regurgitant lesions rather than stenotic and affect mitral valve then the aortic valve in

frequency of involvement.[5] They are present in 43% of SLE patients at autopsy 2, however the prevalence in living patients has not been established, since cardiac involvement is usually clinically silent.

The aim this study was to analyze the patterns of involvement of the heart in our patients with SLE by means of clinicalevaluation, electrocardiography ,and echocardiography.

Methods

Forty six consecutive patients with SLE were included in this study. They attend the medical city teaching hospital during the period from June1993 to June 1994; all patients met the 1982 revised diagnostic criteria of the American Rheumatism association for the classification of SLE.[6]
Patients who did not met four of the revised criteria, those who had history of rheumatic heart disease and those who had unsatisfactory echocardiography where excluded from the study.
A through clinical history was obtained, and detailed clinical examination of cardiovascular system was performed for all patients. Twelve lead electrocardiography (ECG) and chest X ray was taken for all patients. M mode and 2D echocardiography were carried out for each patient, and the image were analyzed according to the requirement of the American society of echocardiography.
The following tests were carried out for each patient: urinalysis, complete blood picture and erythocytes sedimentation rate, LE cell preparation, antinuclear antibody (ANA), antidouble stranded DNA, rheumatoid factor, combs test, VDRL, serum protein electrophoresis, blood urea and serum creatinine.All tests were performed using the standard methods.
Statistical analysis done, using Chi-square test. A P value of < 0.05 was considered to be significant.

Results

The clinical characteristics of SLE patients are summarized in Table 1. Females were predominating (87%) with mean age of 24.9+11.4.Maler rash was the commonest manifestation (60.8%).

Table 1. Basic characteristics of the study participants (N 46).

Clinical features		Number (%)
Age (years) mean ± SD		24.9+11.4
Sex	Male	6(13%)
	Female	40(87%)
Duration (months)		38.2 months(2-144 m)
Malar rash		28(60.8)
Discoid		11(24)
Photosensitivity		27(58.7)
Oral ulcer		24(52)
Arthritis		42(91)
Serositis		16(34.8)
Renal disorder		30(65)
Hematologic disorder		26(56.5)
Immunologic disorder		44(95)
ANA positive		41(89)
VDRL positive		12(26)

The abnormal physical findings on physical examination are shown in table 2; unexplained sinus tachycardia was the most common finding 22%,

while cardiac murmurs were found in 19% and 69.7 % have no physical signs. Cardiomegally was seen on Chest X ray in 20%.

Table 2. Physical signs in the patients

Sign	Number (%)
Tachycardia	10(21.7)
Cardiac murmur	9(19.5)
Irregular pulse	6(13)
S 3 and or S4	6(13)
Elevated JVP	5(11)
Displaced apex	5(11)
Pericardial rub	2(4.3)
No abnormal cardiac signs	32(69.7)

Elctrocardiographic finding were summarized in (table 3).Normal ECG was seen in 65.2%, and the other finding were non specific changes. Echocardiographic study was abnormal in 31 patients (67%) (Table 4).The commonest abnormality was decrease in shortening fraction in 43%, pericardial effusion and or thickening in 32%.Mitral valve was found to be abnormal 22% (10 patients), either in form of incompetence or stenosis and or thickening, while aortic valve incompetence was found in three patients (6.5%) only. We found that reduction in shortening fraction which is an important criterion for left ventricular function was significantly associated with symptoms suggestive of heart failure, reflecting myocardial involvement (P value < 0.05).

We did not find statistically relation between the cardiac involvement in SLE patients and the duration of the illnesses (table 5).

Table 3. Elctrocardiographic finding.

Abnormality	Number (%)
ST ,T changes	10(22)
Abnormal rhythm	9(19)
Ectopic beats	3(6.5)
Conduction defect	2(4.4)
LV hypertrophy	1(2.2)
Low voltage ECG	3(6.5)
Normal ECG	30(65.2)

Table 4.Echocardiographic finding in 46 patients with SLE.

Finding	Number (%)
Normal echocardiography	15(32.6)
Abnormal echocardiographic finding	
Decrease shortening fraction	20(43)
Pericardial effusion and or thickening	15(32.6)
Mitral valve involvement	10(22)
LA enlargement	10(22)
Increase LV dimension	6(13)
Increase LV wall thickness	6(13)
Aortic valve involvement	3(6.5)

Table 5. Correlation between duration of disease in months with the cardiac involvement.

Duration	Number of patient with cardiac involvement	Number of patients without cardiac involvement	Total
<12 months	6	3	9
12 -35 months	11	3	14
>36	16	7	23
Total	33	13	46

P> 0.05

Discussion

This study confirms the frequent involvement of the heart in SLE. There was a clinical evidence of cardiac abnormality in 72% of our patient, an incidence higher than that of 58% reported by Hejtmancik et al who used ECG examination and chest X ray .[7] This can be explained by the fact that we used echo study which can detect asymptomatic cases of pericardial effusion and myocardial dysfunction.

The manifestations of the disease, the cumulative incidence of clinical features are similar to the current description of the disease in other series.[8-11] The cardiovascular symptoms was reported in 21 patients (46%) while abnormal cardiac finding was detected in 14 patients (30%).

There are no certain ECG changes specific for SLE .The most common abnormality detected was ST,T changes which was found in 21% of the patients ,either in form ST elevation due to pericarditis or T inversion with or with out ST depression due to myocarditis or coronary artery disease. Clinical evidence of coronary artery disease was found in two patients who had typical ischemic chest pain and required nitrate therapy. Patients with SLE have endothelial dysfunction that remain significant even after adjustment of other risk factors of coronary artery disease [12].Low HDL cholesterol and diabetes mellitus have a significant influence on myocardial perfusion, found in asymptomatic patients with SLE which was manifested as abnormal myocardial scintigraphy 13 .Myocardial perfusion defect in SLE patients are strongly and independently predictive of coronary artery disease [14]

Low voltage ECG was found in three patients, all had significant pericardial effusion by echocardiography.

Echocardiographic examination detected abnormal finding in 31 patients (67%), the commonest was reduction in shortening fraction, which was found in 20 patients (47%), and seven of them were asymptomatic. There is significant association between symptoms suggestive of heart failure and reduction in shortening fraction measured by Echo. SLE can alter left ventricular structure and function in the absence of valvular and clinical coronary artery disease [15]. Pericardial involvement in the form of effusion and or thickening was detected in 33% an incidence which is comparable to that of 37% reported by Leung at el, who studied 75 patients with SLE by echocardiography9 , and higher than of 25 % reported by Doherty et al.[2] Echocardiographic studies show pericardial abnormalities in between 11% and 54% of SLE patients in recent studies.[16]

Endocardial involvement in the form of valvular dysfunction, thickening, or vegetation was found in 10 patients (22%), seven of them had pure mitral valve disease, and three

patients had both mitral and aortic valve lesions. These figures are comparable to that of 24% and5% for mitral and aortic valve involvement respectively reported by Doherty et al. [2] Mitral valve involvement in form of incompetence was found in six patients, four of them mitral valve thickening. Mitral valve stenosis was reported in two patients and mitral thickening with out functional abnormality in two patients.

Anatomical and functional valvular abnormalities were observed in 40–50% of cases with the transthoracic echocardiography (TTE) and in 50–60% with transesophageal echocardiography (TEE).[17] Therefore, TEE is more sensitive than TTE in revealing valve abnormalities.

Libman Sacks endocarditis was detected in one patient only (2%), a finding which is characteristic of the disease, and it was found in 43% of SLE patients at autopsy. [2] This incidence is much lower than that 12% reported by Leung et al [9] , and 9.3 reported by Galve et al .[18] This might be explained at least partially by the relatively low resolution of our echo machine. The mitral valve thickening which was detected in six patients may be due to small vegetation which can not be differentiated by echo machine.

Aortic valve was affected in three patients, all were in form of aortic regurgitation. No one of our patients found to have tricuspid or pulmonary valve involvement. In contrast to Doherty [2] study who reported 5% and 3% for tricuspid and pulmonary valve respectively.

The pathogenesis of the valve lesions in SLE is generally assumed to be due to the formation of fibrin platelet thrombi on the valve with subsequent organization. The deposition of thrombotic material on damaged valve might lead to valvular thickening , commissural fusion ,and ultimately to stenosis or regurgitation .[2]

In this study we did not find significant association between the duration of the disease and heart involvement, a finding which is consistent with other series, who conclude that clinically significant SLE valvular heart disease, is not necessarily the result of long standing disease. [5]

Study limitations

Numbers of relevant studies in patients with SLE have evidenced an association of antiphospholipid antibodies with cardiopathy, particularly in the case of Libman-Sacks endocarditis, valvar dysfunction, and valvar thickening and this was not studied in this study .[19]

Conclusion

The heart is a common target for systemic lupus erythematosus, hence special attention should be offered for examination and follow up of the cardiovascular system, including echocardiography which can detect abnormalities and can be repeated non-invasively.

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