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Original Research Article

Evaluating of Cardiac Alterations in Systemic Lupus Erythematous using at a Tertiary Care Centre

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Abstract:

Background: Systemic lupus erythematosus (SLE) is a chronic autoimmune inflammatory rheumatologic disease that can affect several organs such as skin, joints, and kidneys. One of the organs reported to be involved in SLE is the heart. It is a multiorgan autoimmune disease associated with high cardiovascular morbidity and mortality that primarily affects young women. Cardiac diseases in lupus may involve the endocardium, myocardium, and pericardium and may be responsible for fatal outcome. Some cardiovascular abnormalities are seen with positive anti Ro/SS-A, anti La/SS-B, anticardiolipin (aCL), and anti-double- stranded DNA (antids DNA). Moreover, endothelial dysfunction was reported in early SLE cases without CVDs which was mostly not related to a CL antibody, disease activity, or disease duration but rather related to renal disease, diastolic hypertension, and diabetes in SLE. The aim of this study is to assess the cardiac function in systemic lupus erythematosus by 2D echocardiography.

Material and Method: This was a cross-sectional, observational type of study done in one year duration, Study was done in General medicine OPD, Rheumatology OPD and ward of Department of Medicine, GMCH, Bettiah. **Conclusion:** SLE patients had an increased prevalence of subclinical systolic and diastolic LV and RV dysfunction. This result advocates for regular follow-up and early screening of SLE patients. Accordingly, treatment focused on improving diastolic heart function may have a role in enhancing QoL and improving the prognosis of SLE patients.

Keywords: Systemic lupus erythematosus, 2D echocardiography, Heart, Palpitation, Chest pain.

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Introduction

Systemic lupus erythematosus (SLE) is a chronic autoimmune inflammatory rheumatologic disease that can affect several organs such as skin, joints, and kidneys. One of the organs reported to be involved in SLE is the heart. It is a multiorgan autoimmune disease associated with high cardiovascular morbidity and mortality that primarily affects young women. [1] SLE-specific factors that play anadditional role include disease activity, persistence of systemic and local inflammation, immune abnormalities, anaccelerated atherosclerotic process, antiphospholipid antibodies (anti-cardiolipin and antiβ2 glycoprotein I) and potential adverse effects of therapies, especially glucocorticoids (GCs) and nonsteroidal anti- inflammatory drugs. [2-6] The mechanisms by which SLE might directly induce changes in the left ventricular structure are many fold and include underlying inflammatory processes leadingto subclinical vasculitis, myocarditis or vascular stiffening and pre-clinical coronary artery disease [7] Many diseases may affect the complex pumping system of the heart, and can be caused by a variety of factors including genetic defects, aging, and environmental stimuli. Left ventricular (LV)

myocardial infarction (MI) due to coronary heart disease and diastolic dysfunction due to hypertension and LV hypertrophy are two major forms of heart disease. The cardiovascular involvement in SLE and the subsequent cardiovascular disease (CVD) predispose to a significant morbidity and can raise the mortality risk [8], which occurs more often late in the disease in the absence of active SLE states. [9] Cardiovascular events are proportionally higher in SLE compared to general populations of comparable age and sex. [10] The endothelial damage in SLE is believed to bedue to several factors which predispose to premature atherosclerosis with subsequent cardiac events. Older age, smoking status, high C-reactive protein (CRP), and antiphospholipid antibodies (aPLs) were among the factors associated with vascular events. [11] Reduced renal function, high C3, and cumulative steroid use were among SLE-related factors to coronary artery calcification [12], although the use of corticosteroid was not found to be consistently associated with CVDs. From another perspective, atherosclerosis and cardiac diseases in SLE are thought to be attributed to chronic inflammation. [13] Cardiac diseases inlupus

may involve the endocardium, myocardium, and pericardium and may be responsible for fatal outcome. [14] Some cardiovascular abnormalities are seen with positive anti Ro/SS-A, anti La/SS-B, anticardiolipin (aCL), and anti-double- stranded DNA (antids DNA). ^[15] Moreover, endothelial dysfunction was reported in early SLE cases without CVDs which was mostly not related to aCL antibodies, disease activity, or disease duration but rather related to renal disease, diastolic hypertension, and diabetes in SLE. [16,17] Cardiac magnetic resonance imaging is regarded as the non- invasive investigation of choice for the diagnosis of myocarditis, including lupus myocarditis. [18,19,20] It is however an expensive tool especially in resource-limited settings. Echocardiography on the other hand is cost effective and can be utilized at the bedside, even in the unstable, ventilated patient. Routine echocardiographic evaluation of left ventricular (LV) wall motion is usually subjective because it depends on the visual determination of endocardial excursion as well as wall thickening. It is frequently used to support a diagnosis of lupusmyocarditis. [21,22] The aim of this study is to assess the cardiac function in systemic lupus erythematosus by 2D echocardiography.

Material and Method

This study was a cross-sectional, observational type, done Study duration is One year. in General medicine OPD, Rheumatology OPD and ward of Department of Medicine, GMCH Bettiah. Total number of cases and control were 45 each.

Inclusion Criteria

• Age: 18–80 years

- Both sexes.
- Patients satisfying Systemic lupus international collaborating clinics classification criteria 2012.
- Newly diagnosed SLE patients and who are willing togive written informed consent to participate in the study.

Exclusion Criteria

- History of cardiomyopathy.
- History of acute coronary syndrome. •
- Diabetes mellitus. •
- Chronic Kidney disease.

Patients satisfying inclusion and exclusion criteria were subjected to detailed history and physical examination with special emphasis on the examination of cardiovascular system. All patients were evaluated for other cardiovascularrisk factors (smoking, obesity, dyslipidemia, diabetes mellitus and arterial hyper-tension). Echocardiography weredone in all patients. Ejection fraction < 55% were considered abnormal. [23] Pulmonary artery hypertension is considered when systolic pulmonary artery pressure > 36 mmHg. [24]

Result

Total of 45 cases and control were studied in this study inone year of duration Various ECG changes among which 80% patients showed sinus tachycardia followed by 26.67% patients showed LVH strain pattern followed by 11.11% patients showed T wave inversion and 6.67% patients showed low voltagecomplex (table 1).

Table	Sable 1: Distribution according to ECG changes in SLE patient				
	ECG Changes	No. of Patients	Percentage		
	LVH Strain Pattern	12	26.67		
	Sinus Tachycardia	36	80.00		
	T Wave Inversion	5	11.11		
	Low Voltage Complex	3	6.67		

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2-D ECHO	Case Group		Control Group		P-	
	No. of Patients	Percentage	No. of Patients	Percentage	value	
Pericardial effusion	10	22.22	1	2.22	0.003	
MVPS	3	6.67	0	0.00	0.07	
Mitral Regurgitation	25	55.56	15	33.33	0.03	
Aortic Regurgitation	5	11.11	7	15.56	0.53	
Tricuspid Regurgitation	23	51.11	15	33.33	0.08	
Pulmonary Hypertension	5	11.11	1	2.22	0.09	
LV systolic dysfunction	1	2.22	0	0.00	0.3	
LV diastolic dysfunction	10	22.22	9	20.00	0.7	
Regional Hypokinesia	1	2.22	0	0.00	0.3	
Global Hypokinesia	2	4.44	2	4.44	1	
LVH	15	33.33	2	4.44	0.0005	
PR	3	6.67	0	0.00	0.07	
AS	1	2.22	0	0.00	0.3	
Normal	14	31.11	27	60.00	0.006	

Table 2: Distribution according to 2-D ECHO in SLE patients

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2-D ECHO parameters in case group and control group were calculated in table 2. Tricuspid Regurgitation was seen in 51.1% patients of case group whereas 33.33% patients of control group. Mitral Regurgitation was seen in 55.56% patients of case group whereas 33.33% patients of control group. LVH changes was seen in 33.33% patients of control group. 31.11% patients showed no change in case group whereas 60% patients of control group was normal. There was significant difference in between these groups in pericardial effusion, Mitral Regurgitation and LVH parameter as p-value was <0.05 whereas MVPS, AorticRegurgitation, Tricuspid Regurgitation, Pulmonary Hypertension, LV systolic dysfunction, LV diastolic dysfunction, Regional Hypokinesia, Global Hypokinesia,PR, AS showed non-significant difference as p-value was >0.05.

2-D ECHO	Chestpain		Palpitation		Dyspnoea	%
	(N=39)	%	(N=28)	%	(N=17)	
Pericardial Effusion	10	25.64	6	21.43	7	41.18
MVPS	3	7.69	2	7.14	2	11.76
Mitral Regurgitation	23	58.97	16	57.14	10	58.82
Aortic Regurgitation	5	12.82	2	7.14	2	11.76
Tricuspid Regurgitation	21	53.85	16	57.14	9	52.94
Pulmonary Hypertension	5	12.82	2	7.14	4	23.53
LV Systolic Dysfunction	1	2.56	1	3.57	1	5.88
LV Diastolic Dysfunction	10	25.64	5	17.86	4	23.53
Regional Hypokinesia	1	2.56	1	3.57	1	5.88
Global Hypokinesia	2	5.13	2	7.14	1	5.88
LVH	15	38.46	8	28.57	7	41.18
PR	3	7.69	1	3.57	1	5.88
AS	1	2.56	1	3.57	0	0.00

Table 3: Association of 2D-Echo finding with Chest Pain, Palpitation and Dyspnoea in SLE patie

we calculated 2-D ECHO parameter in chest pain, palpitation and dyspnoea in patients of case group (table 3).In chest pain cases mitral regurgitation in 58.97% patients, tricuspid regurgitation in 53.85% patients and LVH changeswas seen in 38.46% patients. In palpitation cases mitral regurgitation in 57.14% patients, tricuspid regurgitation in 57.14% patients and LVH changes was seen in 28.57% patients. In dyspnoea mitral regurgitation in 58.82% patients, tricuspid regurgitation in 52.94% patients and LVH changes was seen in 41.88% patients.

Discussion

The cardiac representation as initial manifestations in lupus is uncommon, and cardiac diseases tend to be clinically silent for long periods. [25,26] Yet, the cardiac complications of lupus are potentially serious. Understanding the pathogenesis of cardiovascular complications is very important and incompletely justified. [27] Cardiac involvement in SLE is prevalent in more than 50% of cases and includes myocarditis, pericarditis, valvular heart disease, coronary arterial disease and conduction abnormalities. [28,29] Because the symptoms of myocardial involvement are usually clinically silent compared with other cardiac involvement, its prevalence in 7– 10% might have been underestimated. [30]

We calculated 2-D ECHO parameter in chest pain patients of case group. Mitral Regurgitation was seen in 58.97% patients. Tricuspid Regurgitation was seen in 53.85% patients. LVH changes was seen in 38.46% patients. Inpalpitation patients of case group 2-D ECHO parameter i.e Mitral Regurgitation was seen in 57.14% patients. Tricuspid Regurgitation was seen in 57.14% patients. LVH changes was seen in 28.57% patients. In dyspnoea patients 2-D ECHO parameter i.e Mitral Regurgitation was seen in 58.82% patients. Tricuspid Regurgitation was seen in 52.94% patients. LVH changes was seen in 41.88% patients. We also found 2-D ECHO parameter in APLP positive of SLE case group. LV Diastolic Dysfunction was seen in 80% patients. PHT (Pulmonary Hypertension) was seen in 40% patients. We found ECG changes and 2-D ECHO changes in our study. Sinus Tachycardia was seen in 80% patients followed by 26.67% of LVH Strain pattern followed by 11.11% patients of T-Wave inversion and 6.67% showed low voltage complex. Tricuspid Regurgitation was seen in 51.1% patients of case group and 33.33% patients of control group. Mitral Regurgitation was seen in 55.56% patients of case group and 33.33% patients of control group. LVH changes was seen in 33.33% patients of case groupand 4.44% patients of control group. 31.11% patients showed no change in case group and 60% patients of control group was normal. There was significant difference in between these group in pericardial effusion, Mitral Regurgitation and LVH parameter as p-value was <0.05. MVPS, Aortic Regurgitation, Tricuspid Regurgitation, Pulmonary Hypertension, LV systolic dysfunction, LV diastolic dysfunction, Regional Hypokinesia, GlobalHypokinesia, PR, AS showed non-significant difference as

p-value was >0.05. Mohamed A A A et al conclude that the frequency of occurrence of echocardiographic abnormalities in this study tends to be similar to other previous observations with the mitral and tricuspid valve involvement being the most frequent, while myocardial dysfunction was less. A study conducted by Ashamallah G A et al found that EF% was preserved in 13 patients (65%) and reduced EF % in 7 patients (35%). Intotal, 15 patients (75%) had free wall motion, while 3 (15%) had regional hypokinesia and 2 (10%) had global hypokinesia. Pericardial effusion was found in seven cases (35%). Toit Du Rcompared the Initial echocardiographic characteristics in patients with control patients. They found that the majority of our patients (63%) presented with severeleft ventricular dysfunction (LVEF \leq 35%). Regional wall ordeformation, measured with STE, represents shortening of longitudinal myocardial fibers during systole, again an earlier, more sensitive marker of left ventricular dysfunction compared to LVEF. The midmyocardial and epicardial function may therefore remain relatively unaffected, with circumferential strain and twist showing compensation in order to preserve left ventricular systolic function.

Conclusion

This study highlighted different echocardiographic features in SLE patients and identified their clinical predictors. The clinical impact of such a study was to identify SLE patients at risk of developing serious cardiac complications via the clinical predictors aiming to improve the prognosis of SLE cardiac diseases. SLE patients had an increased prevalence of subclinical systolic and diastolic LV and RV dysfunction. This result advocates for regular follow-up andearly screening of SLE patients. Accordingly, treatment focused on improving diastolic heart function may have a role in enhancing QoL and improving the prognosis of SLE patients. Future research is also needed to define the significance of echocardiographic evidence of subclinical left ventricular dysfunction in asymptomatic SLE patientsin comparison to clinically evident lupus myocarditis. Such research could aid in determining optimal cut-off values forglobal longitudinal strain supporting a diagnosis of clinical lupus myocarditis.

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