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Significant Prevalence of Left Ventricular Diastolic Dysfunction in Patients with Sarcoidosis

Zeynep Zeren Uçar[1], Ahmet Erbaycu[1], Adnan Pınar[2], Aydan Cakan[1], Ayşe Özsöz[1], Nursen Postacı[2], Yeşil Murat[2]

[1]İzmir Chest Diseases and Surgery Center, Chest Diseases, İzmir, Turkey
[2]Atatürk Training and Research Hospital, Department of Cardiology, İzmir, Turkey

Abstract

Background: Sarcoid heart disease is a rare concern. The aim of the study was to evaluate the cardiac involvement, determine the prevalence of left ventricular diastolic dysfunction and find out the relation with other findings in patients with pulmonary sarcoidosis.

Methods: Thirty consecutive inpatients with pulmonary sarcoidosis were underwent 2-D, M-mode and doppler echocardiography. All patients were assessed clinically and 12-lead electrocardiography, serum angiotensin converting enzyme levels, thorax computed tomography, pulmonary function tests, arterial blood gas levels, carbon monoxide diffusion capacity, bronchoalveolar lavage were ordered. Patients were divided in to two groups; with diastolic dysfunction (A) or without diastolic dysfunction (B).

Results: Left ventricular diastolic dysfunction was detected in 19 (63.3%) patients. In comparison of the diastolic indexes, it was disclosed that there were significant prolonged isovolumic relaxation time, deceleration rate of early diastolic flow values and, reversal of E/A ratio in group A. There was statistically significant difference for P_aO_2 level between two groups (p=0.029). Low P_aO_2 levels were observed in seven patients (36.8%) with diastolic dysfunction, where P_aO_2 levels were normal in all patients without diastolic dysfunction. Low forced vital capacity (p=0.023) and much more symptoms like dyspnea and nonspecific chest pain (p=0.026) were detected in patients with diastolic dysfunction. There were no differences when the patients in group A and group B were compared regarding the clinical, radiological, laboratory characteristics.

Conclusion: There is a significant prevalence of abnormal left ventricular diastolic dysfunction in patients with sarcoidosis even if there was no clinical suspicion of cardiac involvement.

Key words: Pulmonary sarcoidosis, cardiac functions, diastolic dysfunction, echocardiography

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INTRODUCTION

Sarcoidosis is a multi system non-caseating granulomatous disorder of unknown etiology predominantly affects lungs, skin and eyes. Cardiac involvement was considered an uncommon complication until relatively recent postmortem studies demonstrated cardiac involvement in 10% to 68% of patients with systemic sarcoidosis. The prognosis of sarcoidosis is generally favorable. However, the prognosis is poor in some patients with cardiac involvement. Sarcoid heart disease is a major concern in patients with sarcoidosis because it accounts for 65% of all sudden death which is the first finding in 40% of this patients [1]. Cardiac sarcoidosis patients demonstrate one, three and five-years survival rates of 91%, 50%, and 37%, respectively [2].

Although cardiac sarcoidosis can result in significant morbidity and death, it can also remain clinically silent despite extensive involvement. The aim of the study was to evaluate the cardiac involvement with two-dimensional (2-D), M-mode and doppler echocardiography (ECHO) and especially to determine the prevalence of left ventricular diastolic dysfunction in patients with pulmonary sarcoidosis. Also the relation between clinical, radiological and laboratory characteristics and diastolic dysfunction was evaluated.

METHODS

Patients And Methods

Thirty consecutive inpatients with biopsy specimen-proved pulmonary sarcoidosis were included in the study. ECHO was performed to all patients with or without cardiac symptoms. Patients, after a detailed evaluation and inquiry, were excluded if any of the following causes of ventricular diastolic dysfunction were present: hypertension, ischemic or structural heart disease, cor pulmonale, diabetes mellitus or alcoholism. Patients known to have atrial fibrillation, sinus tachycardia or any disorders (severe mitral and/or aortic regurgitation or stenosis that may affect Doppler derived parameters of diastolic dysfunction) were also excluded.

All patients were assessed clinically and 12-lead electrocardiography (ECG), serum angiotensin converting enzyme (ACE) levels, thorax computed tomography, pulmonary function tests, arterial blood gas levels, carbon monoxide (CO) diffusion capacity, bronchoalveolary lavage were ordered. Echocardiography was performed just before initiation of corticosteroid treatment.

Patients were staged and divided in to two groups; with diastolic dysfunction (group A) and without diastolic dysfunction (group B). Two groups were compared according

Corresponding Author: Dr. Ahmet Erbaycu, İzmir Chest Diseases and Surgery Center, Chest Diseases, İzmir, Turkey Phone: +90 232 330 93 41 Fax: +90 232 458 72 62
E-mail: drerbaycu@yahoo.com

to age, gender, smoking habits, radiological stage, frequency of bilateral hilar lymphadenopathy (BHL), extrapulmonary involvement, serum ACE level (normal range=8-52U/l), lymphocyte percentage of BAL, CO diffusion capacity, abnormality of ECG, heart rate, forced vital capacity (FVC), and $Pa0_2$. Low FVC was defined as low than 80% of predicted value or as lower than normal limit. Low $Pa0_2$ was defined as the value lower than the value calculated by the formula; 104.2-0.27 x age [3].

Echocardiographic Technique

All patients were examined in the left lateral semirecumbent position using a cardiac ultrasonographic system. (VingMed 725/750 CFM System,Horton, Norway). Two-dimensional (2-D) ECHO was performed from all parasternal and apical windows, Pulsed Doppler ECHO was performed from apical four chamber window, as sample volume placed at the tip of the mitral leaflets and M- mode ECHO was performed only from parasternal long axis. In additional, color Doppler echocardiography was performed from parasternal and apical windows. Firstly, all patients scanned with 2-D ECHO, for any wall motion abnormality, granulomatousis, aneurysm, or heterogeneity. Subsequently, we scanned ejection fraction (EF) and fractional shortening (FS) as systolic functions and Isovolumic relaxation time (IVRT), Deceleration rate of early diastolic flow (DT), Peak velocity of early ventricular filling (E), Peak velocity of late ventricular filling (A) as diastolic functions.

Echocardiographic Measurements

All measurements were made over three cardiac cycles. Left ventricular ejection fraction (EF) was calculated using a modification of Simpson's rule Diastolic dysfunction was defined as the presence of at least two abnormal unrelated indexes of diastolic dysfunction. The abnormal range for each indexes were E/A≤1, IVRT>100, DT>220. The normal range for each index of diastolic function was defined as the mean -/+ SDs of the control group.

Statistical Analysis

Group data expressed as mean-/+ SD. Parametric data were compared using the unpaired Student's t test. Fisher's Exact Test was used to analyze categorical variables. Statistical significance was defined as a p value less than 0.05.

RESULTS

Thirty patients (19 women, 11 men) aged between 23 to 69 years (mean age: 48.1-/+14.3 in group A; 42.7-/+14.8 in group B) were included in the prospective study. Doppler ECHO evidence of left ventricular diastolic dysfunction was detected in 19 (63.3%) of 30 patients. In comparison of the diastolic indexes in group A and B, it was disclosed that there was significant prolonged IVRT (p=0.009), DT (p=0.000) and reversal of E/A ratio (p=0.000) in group A (Table I).

There were no difference in comparison of the patients of group A (with diastolic dysfunction) and group B (without diastolic dysfunction) regarding the clinical, radiological, laboratory characteristics such as age, gender, radiological stage, frequency of BHL, extra pulmonary involvement, serum ACE level, percentage of lymphocyte in BAL, CO diffusion capacity, abnormality in heart rate. Statistically significant difference of $Pa0_2$ level was found in comparison of two groups (p=0.029). Low $Pa0_2$ levels were observed in seven patients (36.8%) with diastolic dysfunction. However, $Pa0_2$ levels were normal in all patients without diastolic dysfunction. We observed low forced vital capacity (85.16-/+19.04% vs 101.18-/+14.61%, p=0.023), more symptoms like dyspnea and nonspecific chest pain (63.2% vs 18.2%, p=0.026) in patients with diastolic dysfunction (Table II).

Table 1. Diastolic Indexes In Two Groups

Echocardiographic Parameter	Group-A	Group-B	P
IVRT (msn)	112.63 ±18.51	96.36 ±6.74	0.009
DT (msn)	238.42 ±44.38	188.18 ±20.4	0.000
E (m/sn)	0.605 ±0.217	0.805 ±0.427	0.042
A (m/sn)	0.753 ±0.212	0.664 ±0.356	0.393
E/A	0.825 ±0.251	1.216 ±0.156	0.000

A: Peak velocity of late ventricular filling
DT: Deceleration rate of early diastolic flow
E: Peak velocity of early ventricular filling
E/ A: Peak velocity of early ventricular filling / peak velocity of late ventricular filling)
IVRT: Isovolumic relaxation time

Table 2. Clinical, Radiological and Laboratory Characteristics of Two Groups

Parameter	Group-A	Group-B	P
Patient (%-n)	%63.3 (19)	%36.7 (11)	-
Age (mean-SD)	48.1±14.3	42.7±14.8	0.33
Women (%-n)	%63.2 (12)	%90.9 (10)	0.19
Men (%-n)	%36.8 (7)	%9.1 (1)	
Smoker (%-n)	%26.3 (5)	%0	0.12
Stage I (%-n)	%15.8 (3)	%36.4 (4)	0.37
Stage II (%-n)	%84.2 (16)	%63.6 (7)	
Dyspnea, nonspecific chest pain (%-n)	%63.2 (12)	%18.2 (2)	0.026
Extrapulmonary involvement (%-n)	%31.6 (6)	%54.5 (6)	0.26
Erythema nodosum (%-n)	%26.3 (5)	%9.1 (1)	0.5
Serum ACE level (mean-SD)U/l	63.85±67.75	77.61±72.61	0.6
BAL lymphocyte (%mean)	31.08±15.55	30.33±9.76	0.9
High cardiothoracic ratio (%-n)	%5.2 (1)	%0	-
Bilateral hilar lymphadenopathy (%-n)	%79 (15)	%54.5 (6)	0.24
Low $Pa0_2$ level (%-(n)	%36.8 (7)	%0	0.029
Low FVC level (%-n)	%31.6 (6)	%0	0.061
FVC (%)	%85.16	%101.18	0.023
Mean CO diffusion capacity (%)	71±26.8	80.5±29.3	0.54
Low CO diffusion capacity (%-n)	%58.3 (7)	%50 (2)	0.419
ECG abnormality (%-n)	%57.9 (11)	%27.3 (3)	0.14
Heart rate beats/min	82.58±16.04	84.18±9.65	0.76

ACE: angiotensin converting enzyme
BAL: bronchoalveolar lavage
CO: carbon monoxide
ECG: electrocardiography
FVC: forced vital capacity
$Pa0_2$: parsiyel arterial oxygen pressure

Electrocardiography showed abnormality in 14 patients (46.7%) of which have also 57.9% abnormality in ECHO. In eight patients (42.12%) no ECG abnormality have been detected. ECG findings are summarized in Table III.

Table 3. Electrocardiography In Two Groups

Electrocardiography	Group-A n (%)	Group-B n (%)
Right axis deviation	3 (%15.8)	2 (%18.2)
Right bundle branch block	2 (%10.5)	0
Sol posterior fascicular block	1 (%5.26)	0
Left axis deviation	1 (%5.26)	0
ST-T changes	3 (%15.8)	0
Q wave	3 (%15.8)	0
Sinus bradycardia	1 (%5.26)	0
Intraventriculary conduction delay	0	1 (%5.26)

2-D Echocardiography was normal except in one patient which showed interventricular septum (IVS) thickening and asymmetric hypertrophy. Mean left ventricle (LV) septum and LV posterior wall thickness were in normal ranges for all patients but mean left ventricular posterior wall (LVPW) thickness was significantly higher in patients group A than group B (p=0.000). Left ventricle dimensions were normal in all patients. Left atrial dimensions and EF were also normal in all patients and there was no statistically significant difference between two groups. Pericardial effusion was observed in one patient (Table IV).

Electrocardiographic findings of patients with diastolic dysfunction are summarized in Table V.

Mitral regurgitation was seen in seven patients with Doppler ECHO, only one of which was in group B. However there was no statistically significant difference between group A and B according to having cardiac valve disorder (p=0.11).

Table 4. 2-D Echocardiographic Parameters In Two Groups

Echocardiographic Parameter	Group-A	Group-B	P
IVS (mm)	10.02 ±1.21	9.51 ±0.67	0.207
LVPW (mm)	9.99 ±0.68	8.91 ±0.70	0.000
LVEDD (mm)	47.05 ±3.27	46.09 ±3.56	0.459
LVESD (mm)	31.21 ±3.66	30.82 ±3.31	0.772
EF(%)	62.58 ±5.67	62.36 ±5.14	0.918
LA (mm)	34.97 ±2.64	33.73 ±3.47	0.287
Pericardial effusion (n)	1	0	-

EF: Ejection fraction
IVS: interventricular septum
LA: left atrium
LVEDD: left ventricle end-diastolic diameter
LVESD: left ventricle end-sistolic diameter
LVPW: left ventricular posterior wall

Table 5. Echocardiographic Parameters of Sarcoidosis Patients With Diastolic Dysfunction

	2 Dimensional Echocardiography									
Case	LA (mm)	LVEDD (mm)	LVESD (mm)	EF (%)	IVS (mm)	LVPW (mm)	IVRT (msn)	DT (msn)	E (m/sn)	A (m/sn)
1	38	44	32	57	9	10	100	240	0.3	0.5
2	40	49	35	60	10	10	100	260	0.4	0.6
3	33	47	31	58	9	9	110	230	0.9	1.2
4	34	45	30	60	10	10	100	340	0.6	0.7
5	34	50	31	60	10	10	110	180	0.4	0.6
6	31	47	32	69	10	10	110	180	0.8	0.8
7	35	52	37	55	7	10	110	220	0.8	1
8	40	44	25	70	11	10	160	240	0.5	0.8
9	37	42	26	65	11	10	130	220	0.6	0.67
10	35	45	30	62	10	9	110	280	0.6	0.75
11	36	52	36	60	13	11,5	100	190	0.4	0.7
12	38	43	27	55	10,4	10,4	110	200	0.9	1.2
13	34	54	39	55	9	9	120	200	0.5	0.5
14	33	48	33	59	9	9	160	330	0.3	0.59
15	33	48	32	60	10	10	110	250	0.9	0.5
16	34	48	28	75	11	11	100	270	0.9	0.9
17	33	46	29	60	10	10	100	230	0.4	0.8
18	34	46	30	70	11	11	100	220	0.5	0.6
19	32	44	30	65	10	10	100	250	0.8	0.9
Mean	34.97±2.64	47.05±3.27	31.21±3.66	62.58±5.67	10.02 ±1.21	9.99±0.68	112.63 ±18.51	238.42 ±44.38	0.605±0.217	0.753±0.212

A : Peak velocity of late ventricular filling
DT : Deceleration rate of early diastolic flow
E : Peak velocity of early ventricular filling
EF : Ejection fraction
IVS :interventricular septum
IVRT : Isovolumic relaxation time
LA: left atrium
LVEDD: left ventricle end-diastolic diameter
LVESD: left ventricle end-sistolic diameter
LVPW :left ventricular posterior wall

DISCUSSION

There is a significant prevalence of abnormal left ventricular diastolic dysfunction in patients with sarcoidosis even if there was no clinical suspicion of cardiac involvement. A statistically significant relation is disclosed between P_aO_2, FVC level and diastolic dysfunction.

Clinical manifestation of sarcoid cardiomyopathy is dependent on the site and extent of granulomatous infiltration; complete heart block ventricular arrhythmias, cardiac failure, sudden death, first degree heart block or BBB, supraventricular arrhythmias, mitral regurgitation, and pericarditis. It is imperative to diagnose this disease as early in it is course as possible. However clinical detection of this disease is unreliable pulmonary and cardiac granulomatous infiltration associated with similar symptoms and signs. Cardiac sarcoidosis is considered, uncommon, and early diagnosis may be further hampered by failure of the physician. This is highlighted by one study revealed an ante-mortem diagnosis in any 65% of cases [4]. Matsui et al reported cardiac sarcoidosis in 78.8% of Japanese sarcoidosis cases with autopsy and death due to cardiac sarcoidosis in 58%. Cardiac lesions were; isolated disseminated granuloma type, conglomerated granuloma type and interstitially spreading type which demonstrated extensive tree-like lesions in the myocardium [5]. In an analysis of 90 Turkish sarcoidosis patients, there was no diagnosis of cardiac sarcoidosis, but no specific techniques were used to reveal cardiac involvement [6]. Aytemur et al also reported no cardiac sarcoidosis in a group of 77 patients [7].

A variety of diagnostic tools which detect evidence of early sarcoid cardiomyopathy mostly suffered from either poor sensitivity or specificity. Some workers have demonstrated abnormalities in 14 to 31% of the cases using 2-D ECHO. In this study and in Fahy et al.'s study [1], 2-D ECHO was abnormal in only one patient (3.3% and 2%, respectively). Aagomachelelis et al reported using Doppler ECHO that 50% of cases with systemic sarcoidosis with clinically normal hearts had evidence of left ventricular diastolic dysfunction. However only ten patients and ten control subjects, all of whom were women, were studied [8]. Previous studies have reported left ventricular diastolic dysfunction in 14 to 50% of cases [1,9].

The overall prevalence of diastolic abnormalities in the community, as defined by the European Study Group on Diastolic Heart Failure (i.e. age dependent isovolumic relaxation time (92-105ms) and early (E-wave) and late (A-wave) left ventricular filling (E/A-ratio, 1-0.5)) is 11.1%. Independent predictors of diastolic abnormalities are arterial hypertension, evidence of left ventricular (LV) hypertrophy, and coronary artery disease. Interestingly, in the absence of these predisposing conditions, diastolic abnormalities (4.3%) or diastolic dysfunction (1.1%) are rare, even in subjects older than 50 years of age (4.6%) and (1.2%), respectively [10]. Although the patients with abnormalities those might effect diastolic dysfunctions were excluded, the present study revealed left ventricular diastolic dysfunction in 63% of cases, using Doppler ECHO. Not to study on a control group concurrently, is a limitation of the study.

Yazaki et al reported an increase of left ventricular end diastolic diameter (>55mm measured with M-mode ECHO) with decreased systolic function (ejection fraction <40% derived from ECHO or left ventriculography) [2]. Although this and other studies show such 14% [11] and different percentages of echocardiographic evidence of left ventricular systolic dysfunction attributed to cardiac sarcoidosis, in our study; LVEF, left ventricular atrial dimensions and systolic functions were normal in all patients. Abnormal wall thickness, with thinning of the basal anterior wall and thinning of the lateral wall and anterolateral papillary muscle and left atrial enlargement were reported in the same study [11]. We could only report mean LVPW thickness in group A to be in normal range but higher than group B.

Fahy et al. [1]; detected left ventricular diastolic dysfunction in seven (14%) of 50 patients. They reported that those with diastolic dysfunction had a longer duration of illness (15-/+7 vs 6-/+5 years; p= 0.0004), were significantly older (52-/+11 vs 38-/+9 years; p=0.0009). In our study there was not statistically significant difference between two groups' ages.

In another study, ECHO of 42 patients with sarcoidosis disclosed 13 patients with abnormalities compatible sarcoid heart involvement such as thickening or thinning of the septum (n=8), pericardial effusion (n=4), and increased end diastolic dimension of the left ventricle with decreased systolic function (n=3) [12].

Different from Lewin et al's study, patients included in the present study were recently diagnosed suffering symptoms less than one year and none of them were on steroid therapy manifested as stage I-II. Because of this characteristics we may have observed less systolic dysfunctions, pericardial effusion and left ventricular or atrial dimension abnormality than Lewin's [12] and other series.

Reports of cardiac sarcoidosis mimicking hypertrophic cardiomyopathy (HCM) are rare. Yazaki et al [13]; reported a case with cardiac sarcoidosis mimicking HCM. ECHO revealed in this case that the thickening of the IVS (18mm) was localized to the basal portions and the normal thickness of the posterior wall (10mm) without chamber dilation simulated HCM with asymmetric septal hypertrophy. In our series, only one patient was observed to have IVS thickening and asymmetric hypertrophy.

The possibility of heart disease in sarcoidosis is sometimes caused by cor pulmonale due to advanced pulmonary fibrosis [14]. None of the patients in present study suffered from cor pulmonale in their clinical, ECG, ECHO findings.

Diastolic dysfunction may be due to infiltration of non-caseating granulomas in the myocardial interstitium or coronary microangiopathy with consequent ischemia or effects of confounding factors like hypertension. However, none of our patients experienced anginal-type chest pain and no significant differences in mean age and arterial blood pressure between two groups. The only confounding factors that may effect diastolic dysfunction between two groups were $Pa0_2$ and FVC. PaO_2 and FVC were lower in the group with diastolic dysfunction [1]. Diastolic dysfunction was manifest by a prolonged IVRT in four out of the seven patients in Fahy's study [1] and 10 out of 19 patients in our study. Also there was a reversal of the normal E/A ratio in six out of seven patients in Fahy's study and 15 out of 19 patients in our study. No patients had evidence of a restrictive pattern of left ventricular filling and LVPW and LA size were normal in both studies.

Some studies have shown that mitral regurgitation is due to papillary muscle involvement and corticosteroid treatment is effective in this group [2,9,15]. Mitral regurgitation was seen in six patients (31.5%) in our study those have diastolic dysfunction. These patients were the ones those had not had any diagnosis of heart disease. They were thought to have the abnormality due to sarcoidosis.

Sarcoid involvement of the heart occurs most frequently in the LV free wall (94%), followed by the IVS (68%), the right ventricle (45%), and the atria (17%). In cardiac sarcoidosis, ECG frequently reveals abnormalities such as atrioventricular conduction defects (85%), right BBB (43%), ventricular beats (43%), complex ventricular arrhythmias (22%) and ST-T changes (21%). ECG abnormality is seen in 9 to 50% of patients with systemic sarcoidosis [14,16-19]. Ventricular tachycardia is seen in 40% and RBBB rate in 51% of the patients with autopsy proven cardiac sarcoidosis. However the patients without histopathological evidence of cardiac sarcoidosis but with clinical suspicion have ventricular tachicardia and RBBB in a percentage of 17% and 51%, respectively [14]. In most cases the lesions have been confined to the atrioventricular node and bundle branches. Only a few case of cardiac sarcoidosis claiming sinus node involvement has been published [20]. In present study, 47.6% of the patients had abnormal ECG, however none of these patients had complex ventricular arrhythmia.

In Sarac et al.'s study [19]; echocardiographic changes in patients with active pulmonary sarcoidosis (stage I, II or III) were registered with 10 (38%) out of 26 patients. Out of 10 patients those have abnormal ECHO, four patients (40%) had increased echoes and hypokinesis of the proximal part of interventricle septum, three (30%) had significantly reduced diffusive capacity and pulmonary hypertension. 58.3% of our patients with diastolic dysfunction had have reduced diffusive capacity.

Non-caseating granuloma is rarely found by subendomyocardial biopsy (19.2%) and so histological confirmation of cardiac involvement may be difficult. However, the sensitivity of endomyocardial biopsy in detecting sarcoid granuloma is low (20-30%) and, instead, various kinds of non-granulomatous pathologies are often seen [14]. Serum ACE used for diagnosis and evaluation of the disease activity, but the former marker is influenced by ACE inhibitors [21,22]. Serum ACE levels were high in both group A and B (63.85-/+67.75 vs 77.61-/+72.61) in the present study and there was no statistically significant difference between them.

Findings of enlarged cardiothoracic ratio are not enough to detect cardiac sarcoidosis in the early stage [17]. We did not revealed any relation between diastolic dysfunction, BHL frequency and cardiothoracic ratio.

There is a belief that sarcoidosis patients with cutaneous lesions, especially facial annular lesions, should be carefully examined and monitored for cardiac involvement, even in cases without apparent cardiac symptoms [23,24]. Okamoto [24]; reported five patients with cutaneous sarcoidosis lesions where cardiac involvement was detected with the appearance of mild cardiac symptoms on a careful examination of the heart. Three out of the five patients showed complete atrioventricular heart block while one showed complete right BBB and left bundle anterior branch block. Examining the frequency of cardiac involvement in sarcoidosis patients with EN may prove of great clinical benefit. We reported only one cutaneous sarcoidosis in group A. There was not statistically significant difference in frequency of EN between group A and B (26.3% vs 9.1%, p=0.5). In cardiac sarcoidosis; noted frequencies are 53% in pulmonary involvement, 13% in skin lesions and 33% in ocular lesions [2]. We did not observe difference in group A and B according to extrapulmonary involvement (31.6% vs 54.5%, p=0.26). We noted one cutaneous, one ocular, one liver, one bone, two parotis involvement in patients with diastolic dysfunction. 63.2% of the patients with diastolic dysfunction had dyspnea and chest pain.

Cardiac sarcoidosis can be clinically silent until patients present with cardiac arrest or sudden death. Patients with cardiac sarcoidosis also may present with arrhythmias such as premature ventricular contractions or nonsustained ventricular tachiacardia. The workup for these arrhythmias usually involves echocardiography and stress testing with nuclear imaging. Cardiac magnetic resonance imaging offers a diagnostic advantage over these tests and should be considered in any patient suspected of having cardiac sarcoidosis [25]. When a suspect occurs for cardiac sarcoidosis, treatment have to be started as soon as possible and to be continued along at least one or two years. Cardiac, especially systolic and diastolic, functions will be normal by treatment [26].

In conclusion, there is no relation between age, gender distribution and frequency of diastolic dysfunction. There is a significant prevalence of abnormal left ventricular diastolic dysfunction in patients with sarcoidosis without suspected cardiac involvement. Usefulness of Doppler ECHO as a screening tool in the detection of early sarcoid cardiomyopathy is uncertain and warrants further investigations. However, the higher presence of diastolic dysfunction in patients with sarcoidosis, where those had cardiac abnormalities had been excluded, lead to the need of routine echocardiographic evaluation. Our study is limited by the absence of histopathological proof of cardiac granulomatous infiltration in patients with diastolic dysfunction. We detected statistically significant relation between P_aO_2, FVC level and diastolic dysfunction. These echocardiographic diastolic abnormalities may be due to hypoxia and the other variables those we could not figure out.

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