SILENT CARDIAC INVOLVEMENT IN RHEUMATIC DISEASE

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SUMMARY: Silent cardiac involvement in rheumatic disease was studied in 61 patients with various rheumatic diseases and 15 healthy controls by noninvasive methods, including electrocardiogram, M-mode and 2 dimensional echocardiography, and systolic time intervals. Patients with Rheumatoid Arthritis and Ankylosing Spondiylitis showed prolongation of QTc and QRS, respectively. Systolic time intervals were equal in both groups. A significant increase in left ventricular mass was found in patients with rheumatic diseases. Therefore we can conclude that these results support the silent cardiac involvement in rheumatic diseases.

Key Words : Rheumatic disease, silent cardiac involvement.

INTRODUCTION

The rheumatic diseases have many similar clinical features and are often classified together. Systemic inflammatory manifestations and acute or chronic arthritis occur in most patients. The etiology and pathogenesis of the rheumatic diseases have not been established yet. They all involve cardiovascular system to varying degrees. Pericarditis, myocarditis, endocarditis and conduction defects in are seen in patients with rheumatic diseases (16). Use of echocardiography has enabled much better diagnosis for cardiac involvement.

The purpose of this study is to investigate the extent of cardiovascular findings in some rheumatic diseases, rheumatoid arthritis (RA), systemic lupus erythematosus (SLE), ankylosing spondiylitis (AS) and Behçet disease (BD), by noninvasive methods. Echocardiography, electrocardiography (EKG), and systolic time intervals (STI) were performed to identify the degree of cardiac involvement.

PATIENTS AND METHODS

Between 1988 and 1992, 61 patients (41 females, 20 males) and 15 normal controls (9 females, 6 males) were included in the study group. Out of 61 patients, 24 patients had RA, 10 patients had SLE, 15 patients had BD and 10 patients had AS. All of the patients fulfilled the American Rheumatism Association (ARA / criteria for RA, SLE, BD an AS (1-3,17).

The mean age for the patients was 35 years (range 19-55) it was 31 years (range 21-37) for the normals. After the physi-

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cal examinations ad the histories, the study group were checked for hematocrit (Hct), white blood count (WBC), serum glucose, urea, creatinine, ALT, AST, uric acid, alkaline phosphatase, ANA and RF, M-mode and 2 dimensional echocardiograms were recorded in the left lateral decubitus position. Ejection fraction (EF), cardiac output (CO), cardiac index (CI), fractional shortening (FS) and left ventricular mass (LVM) were measured by echocardiography. Echocardiographic dimensions were adjusted for body surface area according to the American Society of Echocardiography (15). Twelve-lead ECG was used and PR, QRS and QTc intervals were recorded. STI measurement for left ventricular performance was assessed by Hawlett Packard 8890-A phonocardiogram. The pre ejection period (PEP) the left ventricular ejection time (LVET) the total electromechanical interval (QS_2) and PEP/LVET ratio were calculated according to Weissler regression index (20).

Left ventricular mass (LVM) was calculated by using the formula :

LWM-[(Left ventricul end diastolic dimension + posterior wall thickness + interventricular septum thickness)^3-(Left ventricul end diastolic dimension)^3]x1.05.

Cardiac involvement was diagnosed with either ECG abnormality or echocardiographic abnormality.

Student's t tests were used for statistical analysis.

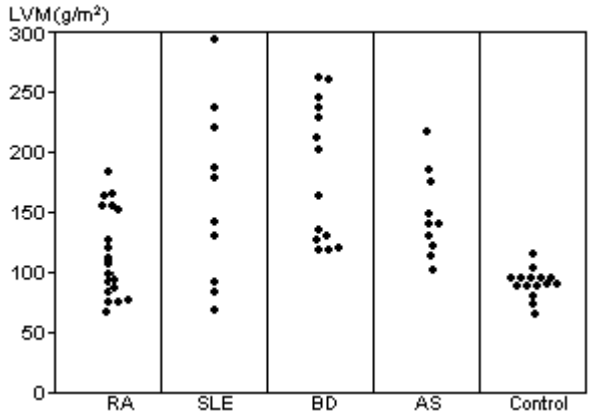
RESULTS

In 61 patients with rheumatic diseases arthritis (RA, SLE, BD and AS) cardiac involvement were compared with 15 normal controls. In RA group, the mean, QTc

Table 1: Comparison of clinical features in the rheumatic diseases and the controls.

Age (years)	RA 32.2±10 (19-59)	SLE 29.7±8.6 (20-46)	BD 30.7±7.2 (20-47)	AS 29.7±6.2 (20-43)	Control 31.1±5.7 (21-37)	Significant p<0.05
Sex (male/female)	-/24	-/12	11/4	9/1	6/9	
The mean duration of disease (year)	5(1-9)	4(1-10)	6(1-2)	8(1-7)		
Heart rate (beats/min)	82.7±9.6	90.6±11.3	80.0±18.1	81.4±15.4	83.2±8.2	p<0.05
Systolic blood pressure (mmHg)	128.7±20.2	126.7±21.9	118.0±16.6	117.5±13.6	120.3±9.7	
Diastolic blood pressure (mmHg)	78.9±12.1	80.0±12.2	77.7±14.0	74.0±9.7	76.7±4.9	p>0.05
ECG findings PR (msn)	148.3±32..2	140.0±21.1	136.0±25.3	136.0±20.6	142.7±18.3	p<0.02
QRS (msn)	76.7±16.3	80.0±0.0	82.2±10.3	104.0±20.6	77.3±28.1	p<0.02
QTc (msn)	411.7±30.8	393.2±14.4	397.1±175.8	382.4±32.6	391.4±18.8	p<0.03
Systolic time intervals PEP (msn)	137.3±24.1	146.0±17.1	159.9±25.7	150.0±15.4	143.8±17.8	p<0.05
LVETI (msn)	443.5±66.3	428.7±25.8	42.4±49.6	421.8±20.4	415.8±37.5	p>0.05
QS_2 I (msn)	570.2±44.4	574.8±23.6	583.5±65.2	571.8±21.7	563.4±39.2	p>0.05
PEP/LVET (msn)	0.369±0.109	0.389±0.077	0.443±0.074	0.215±0.077	0.390±0.082	p>0.05
Echocardiographic measurements Pericardial effusion	2/24(8.3%)	3/12(25%)				
EF	0.58±0.19	0.66±0.07	0.59±0.14	0.57±0.12	0.65±0.05	p>0.05
CO (L/min)	4.17±1.8	5.7±1.6	4.7±2.3	4.9±1.3	4.2±1.3	p>0.05
CI (L/min/m^2)	2.5±1.2	3.7±1.3	2.7±1.3	2.8±0.6	2.3±0.6	p>0.05
PS (%)	34.2±12.7	36.7±5.0	33.7±9.8	33.0±8.5	36.0±2.9	p>0.05
LVM (g/m^2)	125.5±31.0	164.4±82.5	196.9±66.8	149.7±39.1	96.5±13.6	p>0.001

Figure 1: LVM measurements in each group.



interval was significantly longer than both the other groups and the controls ($p<0.03$). On the other hand, in AS group, the mean QRS interval was significantly longer than both the other groups and the controls ($p<0.05$). Systolic time intervals, blood pressure and heart rate measurements were similar in each group.

Echocardiographic signs of pericardial effusion were found in two patients (%8.3) with RA and three patients (%25) with SLE. Echocardiographic measurements (EF, CO, CI, FS) were similar in each group, except LVM. The mean LVM was significantly increased in all groups (RA, SLE, BD and AS) compared to normals. In BD, it was even more significant than it was in the other rheumatic diseases (Table 1).

LVM values were shown in Figure 1.

QTc longer than 420 msn was found in 8 of 24 RA (33%) patient QRS longer than 120 msn was found in 6 of 10 AS (60%) patients.

DISCUSSION

Cardiac involvement in patients with rheumatic diseases has been emphasized since 1881 (4). Cardiac manifestations have been investigated in clinically asymptomatic patients by postmortem and pathological studies. With the use of modern noninvasive techniques, cardiac involvement in rheumatic diseases has become more frequent in recent studies. Especially echocardiography has a unique role in analyzing morphological and functional changes of the heart in patients with these diseases.

Cardiac disease in RA is seen as pericarditis (11-50%), myocarditis (19%), endocardial inflammation, conduction defects and coronary arthritis (11,13,14,19). In one study, increased LVM were observed in 182 RA patients, whereas STI, EF, blood pressure and ECG findings were in normal limits (19). In our study, we demonstrated prolongation of QTc interval increased LVM and 8.3% pericardial effusion in patients with RA. Left ventricular dysfunction is accompanied by vagal autonomic neuropathy of RA, slight anemia, chemical mediators of inflammation, NSAIDs and endogenous catecholamines (12,21). These findings were indicator of cardiac involvement in clinically asymptomatic patients with RA in our study.

Pericarditis (80%) is the most common cardiac manifestation in SLE, and the others are myocarditis (8-18%), endocarditis (16-44%) and conduction defects (34-70%) (5,6,16). We detected pericarditis (25 %) and an increased in LVM in patients with SLE. It has been suggested that an altered hemodynamic state is caused by the disease itself or its treatment (NSAIDs) and cardiac involvement may be found in clinically asymptomatic patients with SLE.

Cardiac involvement in BD is 7-29%, and pericarditis, myocarditis and arrhythmias have been reported in literature (8,9). We found increased LVM in BD. We

suggested that these findings might be related to myocardial involvement of BD.

Ankylosing spondiylitis is a common disease and the degree of cardiac involvement in AS has been described as aortic insufficiency (20%), aortitis, conduction disturbance (5-33%), cardiomyopathy and altered diastolic function. In our study although 10 patients with AS had no cardiac abnormality (7,10,18) on clinical examination, we investigated prolongation of QRS interval and increased LVM in patients with AS.

In conclusion, by noninvasive techniques, we demonstrated cardiac involvement in patients with RA, SLE, BD and AS, although they were all clinically asymptomatic cardiac wise. This is a preliminary study and there is also a need for a multicentre, prospective clinicopathological study for cardiac involvement in rheumatic diseases. Asymptomatic patients with rheumatic disease show definite cardiac abnormalities by ECG, STI and echocardiographic evaluation. Follow up studies could help one understand the natural history and importance of these abnormalities.

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