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# Assessment of Cardiac Functional Alterations of Ankylosing Spondylitis Patients without Cardiovascular Risk Factors

## *Kardiyovasküler Risk Faktörü Olmayan Ankilozan Spondilit Hastalarında Kardiyak Fonksiyonel Değişikliklerin Araştırılması*

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**ABSTRACT**

***Introduction:*** *The aim of this study is to evaluate cardiac functional alterations of ankylosing spondylitis patients without any cardiovascular risk factors.*

***Patients and Methods:*** *Thirty seven consecutive akylosing spondylitis patients without any cardiovascular risk factors constituted our study patient population (age: 41.4 ± 11.1 years, 28 male). Electrocardiographs (ECG) of all patients were obtained and all patients underwent comprehensive transthoracic echocardiographic examination. QRS durations, p wave dispersion and corrected QT dispersion (QTcd) values were calculated from 12-lead ECG's. Data reflecting left ventricular systolic and diastolic functions were obtained from echocardiographic examinations. Data of patients were compared with the data of 28 age-and gender matched healthy control subjects (age: 40.1 ± 10.5 years, 19 male).*

***Results:*** *There were no significant differences between patients and controls regarding QRS durations, p wave dispersion and QTcd values. There were also no significant differences between patients and controls regarding parameters reflecting left ventricular systolic and diastolic functions. Annular velocities at mitral and tricuspid annulus levels evaluated with pulsed-wave tissue Doppler imaging were also similar as well. Two (7.2%) subjects in the control group and 2 (5.4%) patients in the akylosing spondylitis group had minimal aortic regurgitation (p= 0.51).*

***Conclusion:*** *We could not demonstrate any electrocardiographic or echocardiographic evidence of structural myocardial alterations in a small sample of akylosing spondylitis patients free of cardiovascular risk factors. Effects of frequently encountered co-existent cardiovascular risk factors of ankylosing spondylitis patients might have contributed to the conflicting literature data related with this topic.*

***Key Words:*** *Spondylitis, ankylosing; arrhythmias, cardiac; echocardiography; electrocardiography.*

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**ÖZET**

***Giriş:*** *Bu çalışmada amacımız eş zamanlı kardiyovasküler risk faktörü olmayan ankilozan spondilit hastalarında kardiyak fonksiyonel değişiklikleri araştırmaktır.*

***Hastalar ve Yöntem:*** *Çalışma popülasyonu kardiyovasküler risk faktörü olmayan sıralı 37 ankilozan spondilit hastasından (yaş: 41.4 ± 11.1; 28 erkek) oluşturuldu. Tüm hastaların 12 derivasyonlu elektrokardiyografileri (EKG) çekildi ve tüm hastalara detaylı transtorasik ekokardiyografik çalışma yapıldı. 12 derivasyonlu EKG'lerden QRS süresi, p dalga dispersiyonu ve düzeltilmiş QT dispersiyonu değerleri hesaplandı. Ekokardiyografik çalışmada sol ventrikül sistolik ve diyastolik fonksiyonlarını yansıtan veriler elde edildi. Hastaların verileri yaş ve cinsiyet açısından benzer 28 sağlıklı bireyin (yaş: 40.1 ± 10.5; 19 erkek) verileri ile karşılaştırıldı.*

***Bulgular:*** *Hasta ve kontrol grupları arasında QRS süreleri, p dalga dispersiyonu ve düzeltilmiş QT dispersiyonu değerleri arasında anlamlı farklılık saptanmadı. Hasta ve kontrol gruplarının sol ventrikül sistolik ve diyastolik fonksiyonlarını yansıtan ekokardiyografik parametrelerinin de benzer olduğu görüldü. Doku Doppler ile elde edilen mitral ve triküspid anülüs hızlarında da anlamlı farklılık saptanmadı. Ankilozan spondilit grubunda 2 (%5.4) hastada ve kontrol grubunda 2 (%7.2) bireyde minimal aort yetmezliği olduğu izlendi (p= 0.51).*

***Sonuç:*** *Kardiyovasküler risk faktörü olmayan ankilozan spondilit hastaları kullanılarak yapılan çalışmamızda, ankilozan spondilit hastalarının elektrokardiyografik ve ekokardiyografik bulgularının kontrol grubu ile benzer olduğu görüldü. Ankilozan spondilit hastalarındaki kardiyovasküler patoloji prevalansı hakkındaki çelişkili literatür verilerine eş zamanlı bulunan kardiyovasküler risk faktörlerinin katkısı olabileceği düşünüldü.*

***Anahtar Kelimeler:*** *Spondilit, ankilozan; aritmiler, kalp; ekokardiyografi; elektrokardiyografi.*

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## INTRODUCTION

Ankylosing spondylitis is a chronic and inflammatory condition, affecting the spine, sacroiliac and peripheral joints. Although classically thought of as a spinal and peripheral articular disease, extra-articular organs such as the eyes, lungs, neurological system, and heart may also be affected[1]. Association between ankylosing spondylitis and cardiac disorders have been suggested by many studies. Most frequent cardiovascular disorders reported include aortitis, aortic root abnormalities with aortic regurgitation, conduction system alterations and cardiomyopathies[2]. Cardiac involvement may be a result of sclerosing inflammatory process which primarily involves aortic root and aortic valve cusps[3,4]. Fibrosis may also extend below the level of aortic valve through anterior mitral leaflet and septum causing mitral regurgitation and conduction disorders[3,5]. Myocardial involvement is suggested to be a result of diffuse increase in myocardial connective tissue which may give rise to diastolic, and in advanced cases systolic dysfunction[2,6].

There are conflicting data regarding the prevalence of cardiovascular pathologies in patients with ankylosing spondylitis. It has been estimated that cardiac manifestations in patients with ankylosing spondylitis are found in 2-10% of patients; however, some studies have reported similar rate of cardiovascular abnormalities compared with normal population[7-10]. Effects of co-existing cardiovascular risk factors, such as hypertension, may be a contributing factor for these conflicting data.

The aim of this study is to evaluate independent effects of ankylosing spondylitis on cardiac functions by examining electrocardiographic and echocardiographic data of outpatient ankylosing spondylitis patients without cardiovascular risk factors. The data of ankylosing spondylitis patients are compared with the ones of age-and gender matched healthy controls.

## PATIENTS and METHODS

### Patients

Thirty seven ankylosing spondylitis patients without any cardiovascular risk factors were recruited to this cross sectional study (28 male, 9 female; mean age: 41.3 ± 11.1 years). Patients were consecutively selected among patients of the physical medicine and rehabilitation outpatient clinic of our hospital. Twenty-eight healthy subjects who were recruited from hospital staff constituted our control group (19 male, 9 female; mean age: 40.1 ± 10.5 years). A detailed medical story, physical examination, chest X-ray and 12-lead electrocardiography were obtained from all patients. Erythrocyte sedimentation rates (ESR), serum C-reactive protein (CRP) levels, Bath ankylosing spondylitis disease activity index (BASDAI) scores, disease durations and concurrent medications of ankylosing spondylitis patients as non-steroid anti-inflammatory drugs and anti-TNF agents were recorded. Patients with classical cardiovascular risk factors were excluded from the study. Other exclusion criteria were evidence of ischemia on electrocardiograms and history of coronary artery disease, rheumatic

valvular heart disease and atrial fibrillation. All subjects gave written informed consent and the institutional ethical committee approved the study protocol.

**Cardiovascular Risk Factors**

Based on the criteria used previously in similar studies, diabetes mellitus was diagnosed when patients were taking hypoglycemic medications or when, in the absence of treatment, fasting blood glucose levels were higher than 126 mg/dL in two consecutive determinations[11-13]. Hyperlipidemia was defined as fasting total serum cholesterol more than 240 mg/dL and/or when patients were taking an oral lipid-lowering agent. Subjects currently taking antihypertensive drugs or showing a systolic blood pressure of 140 mmHg or more and/or a diastolic blood pressure of 90 mmHg or more, based on the average of two or more readings taken in the sitting position at different days before investigation were defined as hypertensive. Patients smoking at least one cigarette daily for one year within the last five year were considered smokers.

**Echocardiography**

All echocardiographic examinations were performed by the same operator who was blinded to clinical data by using cardiac ultrasound machine Vivid Four (GE-Vingmed Ultrasound ankylosing spondylitis Horten, Norway) equipped with a multifrequency transducer.

Two-dimensional echocardiography, pulsed and continuous wave Doppler, and color flow Doppler studies were performed using standard techniques on each patient. All patients were in sinus rhythm at the time of examination and all measurements were calculated from three consecutive cycles and average of the three measurements was recorded.

Left ventricular end-diastolic (LVEDD) and end-systolic (LVESD) diameters were determined with M-mode echocardiography under two-dimensional guidance in the parasternal long-axis view, according to the recommendations of the American Society of Echocardiography[14]. Left ventricular ejection fraction (LVEF) was calculated from apical four-chamber view, using Simpson's biplane method. Conventional pulsed Doppler imaging of mitral inflow was recorded in the apical four-chamber view placing the sample volume on the leaflet tips of the open mitral valve. Early (E) and atrial (A) peak velocities of mitral valve, their ratio (E/A), and E velocity deceleration time (DT) were measured. Isovolumic relaxation time (IVRT) was measured from the continuous wave Doppler (CWD) signal of the simultaneous aortic and mitral valve flows from the apical 5-chamber view with the sample volume placed between the left ventricle (LV) outflow tract and mitral valve.

Pulsed wave tissue Doppler imaging (TDI) was performed to assess both LV and RV longitudinal functions. In apical four-chamber view, a 5 mm pulsed Doppler sample volume was placed on the mitral annulus at the septal and lateral sites and on the tricuspid annulus at the place of attachment of the anterior leaflet of the tricuspid valve. To minimize the angle between the beam and the direction of annular motion, care was taken to keep the ultrasound beam perpendicular to the plane of the annulus. Peak systolic (S'), early and late diastolic myocardial velocities (E' and A') were recorded. Left ventricular mean E' value was calculated by using E' velocities obtained from septal and lateral mitral annular sites. Left ventricular mean E' value was used for calculation of left ventricle E/E' ratio.

**Electrocardiographic Measurements**

All standard 12-lead ECG's were obtained using a recorder (Schiller AT-2 plus, Switzerland) set at a 50 mm/s paper speed and 1 mV/cm standardization. A single cardiologist who was blinded to clinical status of patients measured ECG intervals. Three consecutive cycles were measured in each of the standard 12 leads and from three values a mean of the parameter was calculated and included in the analysis.

**Basal measurements:** PR intervals, QRS intervals and RR intervals of all patients were recorded.

**p-wave dispersion (Pwd) measurements:** The beginning of p-wave was defined as the point where the initial deflection of p-wave crossed the isoelectric line and the end of p-wave was defined as the point where the final deflection of the p-wave crossed the isoelectric line. p-maximum was determined as the p-wave duration in any lead with the longest interval and p-minimum was determined as the p-wave duration in any lead with shortest interval. p-wave dispersion was calculated by substracting the minimum p-wave duration ($p_{min}$) from the maximal p-wave duration ($p_{max}$). In all patients derivations were excluded if the beginning or the ending of the p-wave could not be clearly identified. Only recordings with more than eight analyzable leads were included.

**QT and QT dispersion measurements:** The QT interval was measured from the onset of the QRS complex to the end of the T wave, defined as the return to the T-P isoelectrical line. If a U wave was present, the QT interval was measured to the nadir between the T and the U waves. If the T waves were isoelectric or of very

low amplitude, that lead was excluded. Only recordings with more than eight analyzable leads were included. QT maximum was determined as the QT interval in any lead with the longest interval and QT minimum was determined as the QT interval in any lead with shortest interval. Measured QT intervals were corrected for heart rate with Bazett's formula (QTc = QT/√RR). Corrected QTd (QTcd) was defined as the difference between corrected maximum ($QTc_{max}$) and minimum QT ($QTc_{min}$) interval among the 12 ECG leads.

### Statistics

Statistical Package for Social Sciences (SPSS Inc., Chicago, IL, USA) version 11.0 was used for data analysis. Distribution of data was assessed by using one-sample Kolmogorov-Smirnov test. Values displaying normal distribution were expressed as the mean ± SD while values not displaying normal distribution were expressed as median (interquartile range). Significance of difference between groups regarding numeric variables with normal distribution was tested with independent samples Student's t-test. Significance of difference between groups regarding numeric variables without normal distribution was tested with Mann-Whitney U test. A p value less than 0.05 was considered significant.

## RESULTS

There were no significant differences between patients and controls with respect to age and gender (28 male, mean age: 41.3 ± 11.1 years vs. 19 male, mean age: 40.1 ± 10.5 years, respectively; p= 0.33 for gender and p= 0.66 for age). The laboratory and clinical characteristics of ankylosing spondylitis patients is shown on Table 1.

There were no significant differences between ankylosing spondylitis patients and controls regarding M-mode, 2D and Doppler mitral inflow parameters (Table 2). Two patients in the control group (7.2%) and 2 (5.4%) patients in the ankylosing spondylitis group had minimal aortic regurgitation (p= 0.51). Three subjects in the control group (10.7%) and 4 (10.8%) patients in the ankylosing spondylitis group had minimal mitral regurgitation (p= 0.65).

There were also no significant differences regarding tissue Doppler velocities obtained at the level of mitral and tricuspid annulus between ankylosing spondylitis patients and controls (Table 3).

Results of electrocardiographic analysis of patients and controls are shown on Table 4. There were no significant differences between groups regarding PR intervals, QRS intervals and p wave and QTc dispersion values. One

*Table 1. The laboratory and clinical characteristics of ankylosing spondylitis patients*

| | Ankylosing spondylitis patients (n= 37) | Controls (n= 28) | p |
|---|---|---|---|
| **Systolic blood pressure (mmHg)** | 126.3 ± 12.0 | 125.0 ± 10.0 | 0.75 |
| **Diastolic blood pressure (mmHg)** | 78.0 ± 6.2 | 81.7 ± 11.1 | 0.20 |
| **Fasting blood glucose (mg/dL)** | 87.9 ± 9.1 | 86.2 ± 10.1 | 0.53 |
| **Total cholesterol (mg/dL)** | 182.2 ± 25.7 | 183.1 ± 31.1 | 0.92 |
| **Plasma LDL (mg/dL)** | 125.4 ± 19.8 | 123.7 ± 26.1 | 0.80 |
| **Plasma HDL (mg/dL)** | 36.6 ± 6.2 | 39.9 ± 12.5 | 0.24 |
| **Plasma triglyceride (mg/dL)** | 103.0 ± 49.9 | 104.9 ± 45.2 | 0.90 |
| **ESR (mm/hour)** | 27.6 ± 16.5 | - | - |
| **CRP (mg/L)** | 1.4 ± 1.5 | - | - |
| **BASDAI score** | 3.2 ± 2.1 | - | - |
| **Disease duration (years)** | 11.2 ± 10.2 | - | - |
| **Chest expansion (cm)** | 2.9 ± 1.7 | - | - |
| **NSAI therapy** | 29 (78%) | - | - |
| **Anti-TNF-α therapy** | 2 (5%) | - | - |
| **HLA B27** | Positive: 4 (10.8%)<br>Unknown: 33 (89.2%) | - | - |

LDL: Low density lipoprotein, HDL: High density lipoprotein, ESR: Erythrocyte sedimentation rate, CRP: C-reactive protein, BASDAI: Bath ankylosing spondylitis disease activity indeks, NSAI: Non-steroid anti-inflammatory.

*Table 2. Comparison of conventional M-mode, 2D, and Doppler mitral inflow parameters between ankylosing spondylitis patients and controls*

| | Patients (n= 37) | Controls (n= 28) | p |
|---|---|---|---|
| **Aort diameter (mm)** | 29.6 ± 4.9 | 28.8 ± 4.5 | 0.47 |
| **Left atrium diameter (mm)** | 33.2 ± 3.7 | 32.9 ± 3.6 | 0.83 |
| **End-Diastolic parameter (mm)** | 44.9 ± 5.2 | 46.9 ± 5.2 | 0.14 |
| **End-Systolic parameter (mm)** | 28.4 ± 4.3 | 29.3 ± 4.0 | 0.39 |
| **Ejection fraction (%)** | 65.5 ± 5.2 | 66.6 ± 4.0 | 0.32 |
| **Septum thickness (mm)** | 0.94 ± 0.10 | 0.93 ± 0.08 | 0.63 |
| **Posterior wall thickness (mm)** | 0.94 ± 0.08 | 0.90 ± 0.08 | 0.08 |
| **Mitral E velocity (cm/sec)** | 0.72 ± 0.14 | 0.77 ± 0.15 | 0.25 |
| **Mitral A velocity (cm/sec)** | 0.67 ± 0.20 | 0.73 ± 0.26 | 0.35 |
| **IVRT (msec)** | 87.3 ± 12.1 | 85.6 ± 15.0 | 0.61 |
| **E deceleration time (msec)** | 200.3 ± 40.6 | 201.3 ± 33.5 | 0.92 |
| **Mitral E/A ratio** | 1.15 ± 0.34 | 1.16 ± 0.45 | 0.93 |

IVRT: Isovolumic relaxation time.

*Table 3. Comparison of tissue doppler velocities between ankylosing spondylitis patients and controls*

| | Patients (n= 37) | Controls (n= 28) | p |
|---|---|---|---|
| **Septal S' (cm/sec)** | 8.1 ± 2.0 | 7.8 ± 1.8 | 0.44 |
| **Septal E' (cm/sec)** | 10.0 ± 2.9 | 10.9 ± 2.7 | 0.21 |
| **Septal A' (cm/sec)** | 9.3 ± 2.8 | 9.5 ± 2.9 | 0.75 |
| **Lateral S' (cm/sec)** | 10.4 ± 3.2 | 9.4 ± 2.7 | 0.22 |
| **Lateral E' (cm/sec)** | 13.5 ± 3.9 | 15.3 ± 3.6 | 0.07 |
| **Lateral A' (cm/sec)** | 9.9 ± 3.6 | 9.6 ± 2.8 | 0.70 |
| **RV S' (cm/sec)** | 13.2 ± 2.1 | 12.4 ± 1.9 | 0.11 |
| **RV E' (cm/sec)** | 13.4 ± 3.1 | 13.5 ± 3.5 | 0.96 |
| **RV A' (cm/sec)** | 15.3 ± 4.6 | 14.8 ± 4.8 | 0.72 |
| **E/E' ratio** | 6.4 ± 1.8 | 6.1 ± 1.2 | 0.43 |

*Table 4. Comparison of electrocardiographic parameters between ankylosing spondylitis patients and controls*

| | Patients (n= 37) | Controls (n= 28) | p |
|---|---|---|---|
| **QRS duration (msec)** | 98.4 ± 15.4 | 101.6 ± 16.7 | 0.43 |
| **PR interval (msec)** | 151.4 ± 22.5 | 150.8 ± 24.5 | 0.92 |
| **RR interval (msec)** | 788.6 ± 137.5 | 825.8 ± 110.5 | 0.26 |
| **$P_{max}$ (msec)** | 109.6 ± 12.8 | 114.9 ± 9.5 | 0.08 |
| **$P_{min}$ (msec)** | 67.8 ± 12.3 | 73.9 ± 15.2 | 0.08 |
| **$P_{dispersion}$ (msec)** | 41.9 ± 12.6 | 41.0 ± 13.8 | 0.80 |
| **$QTc_{max}$ (msec)** | 496.4 ± 70.9 | 493.0 ± 62.0 | 0.84 |
| **$QTc_{min}$ (msec)** | 439.4 ± 60.0 | 423.1 ± 49.2 | 0.26 |
| **$QTc_{dispersion}$ (msec)** | 57.0 ± 28.2 | 69.9 ± 32.4 | 0.10 |

(2.7%) patient in the ankylosing spondylitis group and 2 (7.1%) patients in the control group had first degree AV block (p= 0.41). Five (13.5%) patients in the ankylosing spondylitis group and 4 (14.3%) patients in the control group had incomplete right bundle branch block (p= 0.60). Two (5.4%) patients in the ankylosing spondylitis group and 1 (3.6%) patient in the control group had left anterior hemi-block (p= 0.60). None of the subjects in the study population had complete right or left bundle branch block. Two (5.4%) patients in the ankylosing spondylitis group displayed ventricular extrasystoles while none of control subjects displayed ventricular extrasystoles on their ECG's (p= 0.32). Neither patients nor controls displayed any supraventricular extrasystoles on their ECG's.

## DISCUSSION

There were no significant differences between ankylosing spondylitis patients without cardiovascular risk factors and control subjects regarding cardiac functions as evaluated by echocardiographic and electrocardiographic parameters in our study. There were also no significant differences regarding rate of aortic or mitral valve regurgitation.

Several studies have shown higher rate of aortic valve regurgitation, conduction disturbances and cardiomyopathies in patients with ankylosing spondylitis compared to the normal population[2,7]. However, symptomatic cardiac involvement is rarely seen in practice and there are conflicting data regarding the prevalence of cardiovascular pathologies in patients with ankylosing spondylitis. The prevalence of aortic root disease and valve disease associated with ankylosing spondylitis has been reported to range from 24% to %100 in postmortem series and 8% to 31% by transthorasic echocardiography[3,15-19]. Aort diameters of ankylosing spondylitis patients were similar with the ones of controls in our study and only two ankylosing spondylitis patients had minimal aortic valve regurgitation (5.4%).

The rate of conduction disorders of ankylosing spondylitis patients as atrioventricular and bundle branch blocks in the literature vary between 3% and 23%[10,20]. (21.6%) Eight patients in the ankylosing spondylitis group displayed first degree atrioventricular block or incomplete right bundle branch block or left anterior hemiblock in our study. However, there were no significant differences regarding rate of atrioventricular and bundle branch blocks between patients and controls and none of the ankylosing spondylitis patients displayed clinically significant conduction disorders. QRS and PR durations of patients and controls were also similar which argues against the suggestion of ankylosing spondylitis related myocardial fibrosis to be the cause of conduction disturbances observed in ankylosing spondylitis patients. The dispersion of ventricular repolarization from the surface electrocardiogram has been proposed as a simple non-invasive marker of susceptibility to ventricular arrhythmias[21-23]. One study reported increased QT dispersion (QTd) in ankylosing spondylitis patients; however another study found similar QTd between ankylosing spondylitis patients and controls[10,24]. p wave dispersion (PWd) has been suggested to have predictive value for atrial fibrillation and two studies evaluating PWd in ankylosing spondylitis patients reported discrepant results[10,25]. We also found similar QTd and PWd values between ankylosing spondylitis patients and controls, which may imply similar arrhythmia tendency of ankylosing spondylitis patients and controls in our study.

Diastolic dysfunction is also a frequently reported abnormality in ankylosing spondylitis patients and myocardial fibrosis related with the disease process has been suggested to be responsible factor[2]. However, effects of other possible factors like hypertension and significant valvular pathologies should be excluded to relate diastolic dysfunction with ankylosing spondylitis. Mitral inflow parameters and tissue Doppler velocities of ankylosing spondylitis patients without any cardiovascular risk factors were similar with the ones of control subjects in our study. E/E' ratio, which is suggested to be a reliable predictor of ventricular compliance, was also similar between our patients and controls[26].

Our results are in agreement with the results of Brunner et al. who examined 100 male subjects with diagnosed ankylosing spondylitis for over 15 years[9]. They evaluated cardiac abnormalities of these patients with resting ECG and echocardiography as in our study and compared their results with results from the literature in the normal population. They found no significant differences regarding the rates of valve regurgitation, conduction blocks and arrhythmia. They reported higher prevalence of diastolic dysfunction in ankylosing spondylitis patients however much of these patients were relatively older and had co-existing hypertension, which are two important risk factors for diastolic dysfunction. They suggested that impact of cardiac abnormalities in patients with ankylosing spondylitis might have been overestimated in the past and they argued against routine cardiologic evaluation with echocardiography in these patients. Hypertensive patients were excluded in our

study and age and gender matched control group was used which excluded effects of age, gender, hypertension and other cardiovascular risk factors on the presence of diastolic dysfunctions and conduction disorders.

The cause of discrepancy regarding the prevalence of cardiovascular pathologies of ankylosing spondylitis patients in the literature is not clear. Effects of coexisting cardiovascular risk factors, effects of anti-inflammatory therapy and heterogeneity regarding disease durations and activities may be contributing factors. Most of our patients were on anti-inflammatory therapy and their ESR and CRP levels were low which may be a reflection of suppressed inflammatory activity. Roldan et al. studied the root and valves in ankylosing spondylitis patients using transesophageal echocardiography and found markedly increased rate of aortic root and valve disease compared with controls[3]. During follow up of 25 patients over 39 months in this study, in up to 24% new aortic root or valve abnormalities developed, in 12% existing valve regurgitation worsened significantly and in 20% abnormalities resolved which demonstrates dynamic nature of ankylosing spondylitis related cardiac inflammatory activity. Therefore, it may be hypothesized that anti-inflammatory medications of patients may have suppressed cardiac inflammation and related cardiac abnormalities in our study. Unfortunately, to our knowledge, no studies have been performed evaluating the usefulness of anti-inflammatory medications in halting the progression of the disease. Therefore, further research is necessary to test our hypothesis.

Our study was a cross sectional study and patients were consecutively selected among patients who had been followed by the diagnosis of ankylosing spondylitis. HLA-B27 status was unknown for majority of patients. This may also be concerned with our observations since presence of HLA-B27 is suggested to be related with ankylosing spondylitis related cardiac complications as severe conduction disturbances and aortic valve regurgitation[27,28].

### Limitations

Small sample size is main limitation of this study. In addition, our study was a cross-sectional study and there was heterogeneity regarding the duration of ankylosing spondylitis between patients. HLA-B27 status was unknown for majority of patients. We were unable to perform Holter examination in our study, which precluded assessment of arrhythmia prevalence and cardiac autonomic functions of ankylosing spondylitis patients. However, we examined QT and p wave dispersions on ECG's which are supposed to reflect tendency for arrhythmia. In our opinion, it would be more interesting if the cohort would be prospectively followed to unmask potential cardiac interrelation.

### Conclusion

There was no electrocardiographic or echocardiographic evidence of structural myocardial alterations in a small sample of ankylosing spodylitis patients without cardiovascular risk factors in our study. Our results are in agreement with the notion that impact of cardiac abnormalities in patients with ankylosing spondylitis might have been overestimated in the past. Further large-scale and prospective studies are needed to reach a conclusion regarding cardiovascular effects of ankylosing spondylitis and the effects of disease related anti-inflammatory medications on possible cardiovascular complications of ankylosing spondylitis.

## CONFLICT of INTEREST

None declared.

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