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The Cardiac MRI Assessment of Myocardial Fibrosis in Ankylosing Spondylitis and Its Correlation with Transthoracic Echocardiography (TTE) and Clinical Activation Indices

Ankilozan Spondilitte Kardiyak Manyetik Rezonans Görüntüleme (MRG) ile Miyokardiyal Fibrozisin Değerlendirilmesi ve Bulguların Transtorasik Ekokardiyografi (TTE) ile Klinik Aktivasyon İndeksleriyle Korelasyonu

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ABSTRACT

Objectives: This study investigates cardiac involvement in ankylosing spondylitis (AS) using cardiac magnetic resonance imaging (MRI). The findings are correlated with transthoracic echocardiography (TTE) and clinical and laboratory data.

Methods: The study included patients diagnosed with HLA-B27 positive AS for at least 10 years, without any known cardiac complications or risk factors, but with diastolic dysfunction detected via TTE. The average patient age was 40 years. Contrast-enhanced cardiac MRI was performed to assess cardiac morphology and myocardial pathologies. Disease activity was evaluated using the Bath Ankylosing Spondylitis Activity Index (BASDAI) and the Bath Ankylosing Spondylitis Functional Index (BASFI). Laboratory markers, including C-reactive protein (CRP) and erythrocyte sedimentation rate (ESR), were measured to determine disease activity.

Results: Patients with increased clinical activation indices and prolonged E-wave deceleration time on tissue Doppler imaging exhibited myocardial pathology on cardiac MRI. MRI findings included intramural myocardial edema, late gadolinium enhancement, and myocardial thinning in the left ventricular wall. No statistically significant association was found between cardiac MRI findings and laboratory markers.

Conclusion: AS patients with diastolic dysfunction may benefit from MRI to localize underlying myocardial pathology. Abnormal MRI findings were associated with higher disease activation indices, suggesting a role for inflammation in the pathogenesis of myocardial involvement in AS.

Keywords: Activation indices; Ankylosing spondylitis; Cardiac MRI; Diastolic dysfunction; Myocardial fibrosis; Transthoracic echocardiography.

ÖZET

Amaç: Bu çalışma, ankilozan spondilit (AS) hastalarında kardiyak manyetik rezonans görüntüleme (MRG) kullanılarak kardiyak tutulumu araştırmakta ve elde edilen bulgular transtorasik ekokardiyografi (TTE) ile klinik ve laboratuvar verilerle karşılaştırılmaktadır.

Yöntem: Çalışmaya, en az 10 yıldır HLA-B27 pozitif AS tanısı almış, bilinen kardiyak hastalığı veya risk faktörü bulunmayan ancak TTE ile diyastolik disfonksiyon saptanan hastalar dahil edilmiştir. Hastaların ortalama yaşı 40'tır. Kardiyak morfoloji ve miyokardiyal patolojileri değerlendirmek amacıyla kontrastlı kardiyak MRG yapılmıştır. Hastalık aktivitesi, Bath Ankilozan Spondilit Aktivite İndeksi (BASDAI) ve Bath Ankilozan Spondilit Fonksiyonel İndeksi (BASFI) ile değerlendirilmiştir. Hastalık aktivitesini belirlemek için C-reaktif protein (CRP) ve eritrosit sedimantasyon hızı (ESR) gibi laboratuvar belirteçleri ölçülmüştür.

Bulgular: Klinik aktivasyon indeksleri yüksek olan ve doku Doppler görüntülemede E-dalgası deselerasyon süresi uzamış hastalarda kardiyak MRG'de miyokardiyal patoloji saptanmıştır. MRG bulguları arasında intramural miyokardiyal ödem, geç gadolinyum tutulumu ve sol ventrikül duvarında miyokard incelmesi yer almıştır. Kardiyak MRG bulguları ile laboratuvar belirteçleri arasında istatistiksel olarak anlamlı bir ilişki bulunmamıştır.

Sonuç: Diyastolik disfonksiyonu olan AS hastaları, altta yatan miyokardiyal patolojinin lokalizasyonunu saptamak açısından MRG'den fayda görebilir. Anormal MRG bulguları, daha yüksek hastalık aktivite indeksleri ile ilişkilendirilmiştir; bu da miyokard tutulumu patogenezinde inflamasyonun rol oynayabileceğini düşündürmektedir.

Anahtar sözcükler: Aktivasyon indeksleri; Ankilozan spondilit; Diyastolik disfonksiyon; Kardiyak MRG; Miyokardiyal fibrozis; Transtorasik ekokardiyografi.

Ankylosing spondylitis (AS) is a systemic chronic inflammatory disease that affects multiple organs. It has a prevalence of approximately 1%, with a male-to-female ratio of 3:1 to 4:1. The sacroiliac and spinal joints are primarily involved, leading to characteristic inflammation of tendons and ligaments at their attachment sites (enthesitis).[1–2] Peripheral joint involvement is less common. Beyond musculoskeletal symptoms, cardiovascular involvement is reported in 2%–10% of cases, with cardiac-related mortality rates ranging from 20%–40%.[1,3]

Cardiac involvement in AS presents in four forms: valvular, myocardial, pericardial, and coronary artery involvement.[1] The most frequently observed pathology is aortic valve disease, which can result in aortic regurgitation, ascending aortitis and, less commonly, mitral valve dysfunction and heart failure.[3] Myocardial involvement in AS is primarily characterized by myocardial fibrosis, which results from arteritis affecting the AV-node artery and inflammation extending along the interventricular septum and aortomitral complex. These pathological changes may contribute to conduction abnormalities, including first-degree atrioventricular (AV) block, right or left bundle branch block and, less frequently, pericarditis.[4]

Studies have demonstrated a 1.4-fold increased prevalence of coronary artery disease (CAD) in AS patients compared to non-AS individuals. Chronic endothelial inflammation is believed to accelerate atherosclerosis in AS patients.[5] Various imaging modalities, including transthoracic echocardiography (TTE), tissue Doppler imaging, gated Tc-99 and T-201 SPECT scans, PET scans, and serum collagen biomarker measurements, have been used to assess cardiac involvement in AS.[6,7] More recently, cardiac MRI has been employed to visualize myocardial inflammation and fibrosis in autoimmune diseases such as rheumatoid arthritis (RA) and systemic lupus erythematosus (SLE).[8,9]

This study aimed to assess myocardial involvement using cardiac MRI in a cohort of long-standing AS patients with diastolic dysfunction but no overt cardiac symptoms.

Methods

Patient Selection

This prospective study was approved by the institutional ethics committee (protocol nos. 4.34.59-20/5536). Informed consent was obtained from all participants. Our study complied with the Declaration of Helsinki and the Good Clinical Practice Guidelines. The study enrolled 14 patients aged 40±10 years, all of whom had been diagnosed with HLA-B27 positive AS for at least 10 years and exhibited diastolic dysfunction on echocardiography. Within one month of the TTE diagnosis, patients underwent contrast-enhanced cardiac MRI.

Exclusion Criteria

Patients with known CAD (angina or TTE evidence of myocardial ischemia), previous heart disease, significant valvular disease, atrial fibrillation, or risk factors for CAD

(diabetes mellitus, hyperlipidemia, hypertension, smoking) were excluded. Additional exclusion criteria included MRI contraindications (pacemakers, defibrillators, contrast agent hypersensitivity, severe renal dysfunction, claustrophobia, or chronic obstructive pulmonary disease).

Rheumatological and Cardiac Assessments

Blood tests were conducted to assess human leukocyte antigen B27 (HLA-B27), CRP, and ESR. Disease activity was measured using the BASDAI and BASFI scores.

Echocardiography Protocol

TTE assessments were conducted using a Vivid 4 (GE-Vingmed Ultrasound, Horten, Norway) with a multifrequency transducer. Measurements included mitral valve early (E) and atrial (A) peak velocities, E/A ratio, E-wave deceleration time (DT), and isovolumetric relaxation time (IVRT). Tissue Doppler imaging was used to measure left ventricular and right ventricular longitudinal function, including peak systolic (S'), early (E'), and late (A') diastolic myocardial velocities. The left ventricular E/E' ratio was calculated.

Cardiac MRI Protocol

Cardiac MRI was performed using a 1.5-T MR system (Avanto, Siemens Medical Systems, Erlangen, Germany). All sequences were acquired during a 10–15 second breathhold with electrocardiographic triggering. The cardiac MRI protocol included a functional left ventricular study utilizing True FISP (True Fast Imaging with Steady-State Precession) cine imaging (TR:1.4 ms, TE:1.2 ms, slice thickness:8 mm) in 2-chamber, 4-chamber, and short-axis views. Additionally, HASTE (Half-Fourier Single-Shot Turbo Spin Echo) imaging (TR:700 ms, TE:43 ms, slice thickness:8 mm) was performed. T2 black-blood weighted imaging (TSE-STIR or triple inversion; alternative TSE-SSFP) was acquired in three short-axis slices (basal, midventricular, and apical) and long-axis slices (TR:2 RR intervals, TE:65 ms, slice thickness:8 mm).

For late gadolinium enhancement imaging, datasets were obtained 10–20 minutes after administering 0.2 mmol/kg of a gadolinium-based contrast agent (gadopentetate dimeglumine, Gd-DTPA2) using a three-dimensional (3D) inversion recovery TurboFLASH (Fast Low Angle Shot) sequence (TR:8 ms, TE:4 ms, flip angle:25°, slice thickness:6 mm).

Cardiac chamber dimensions and myocardial thickness were evaluated using True FISP and HASTE sequences. Myocardial edema presence and localization (subendocardial, intramural, or subepicardial) were assessed on T2 TIRM sequences. Myocardial contractility was analyzed through True FISP cine imaging. Lastly, myocardial fibrosis presence and localization were examined using late contrast FLASH sequences.

Statistical Analysis

During the research application phase, a statistical comparison was conducted between TTE tissue Doppler findings and cardiac MRI results. The relationship between myocardial contrast enhancement on cardiac MRI and the presence of diastolic dysfunction parameters on TTE was analyzed using the SPSS v.18.0 statistical software.

First, the Shapiro-Wilk test was applied to assess whether the data followed a normal distribution. The results indicated that the data did not conform to a normal distribution. Consequently, the non-parametric Kruskal-Wallis test was used to determine whether there were significant differences in TTE tissue Doppler findings between patient groups with and without myocardial contrast enhancement on cardiac MRI. The significance threshold was set at 0.05.

To further analyze the relationship between BASDAI and BASFI scores and myocardial contrast agent uptake, a regression analysis was performed. The overall regression model was statistically significant ($F=53.531$, $p<0.01$), explaining 89% of the variance in myocardial contrast agent involvement ($R^2=0.890$).

A second regression analysis was conducted to assess the additional impact of inflammatory markers (CRP and ESR) on myocardial contrast agent uptake. The significance level for this regression analysis was also set at 0.05.

Results

The mean age of the 14 patients included in the study was 44.5 years (range:30–50 years), and the mean duration of the disease was 15 years (range:10–20 years). In all patients, the transthoracic TTE tissue Doppler test was consistent with diastolic dysfunction; one or more findings were observed, including a decreased E/A and E'/A' ratio, changes in E, E', A, A' wave rates, and an increased isovolumetric relaxation time (Table 1).

Table 1. Baseline clinical and imaging characteristics of patients

	E	A	E/A	E' Septal	A' Septal	E'/A' Septal	E' Lateral	A' Lateral	E'/A' Lateral	IVRT	E DEC TIME	BASDAI	BASFI	MRI	CRP	SED
1.	0.5	0.9	0.5	8.5	10.2	0.83	6.2	8.7	0.71	80	222	6.4	5.1	+	0.8	10
2.	0.88	0.66	1.3	15.41	9.5	1.62	14.07	8.5	1.65	71	202	3.2	0	-	0.8	18
3.	0.7	0.6	1.16	13.9	13.06	1.06	16.6	15.6	1.06	103	207	3.7	1.3	-	1.1	11
4.	0.5	0.7	0.71	6.8	13.23	0.51	6.5	9	0.72	88	316	3.6	6.5	+	3.9	52
5.	0.7	0.6	1.16	10.3	14.91	0.69	13.4	5.2	2.57	88	191	2.4	0	-	1.14	18
6.	0.7	0.5	1.4	9.7	8.2	1.18	10.7	5.8	1.84	87	198	5.8	7.6	+	6	30
7.	0.7	1	0.7	5	12.9	0.38	6.5	8.5	0.76	87	181	1	1.3	-	1.38	34
8.	0.5	0.6	0.83	9.2	9.7	0.94	9.7	12.2	0.79	95	220.6	2.4	2.9	-	0.53	20
9.	0.63	0.53	1.13	11.5	5.6	2.05	12.5	7.2	1.73	95	236	1.3	0	-	0.64	28
10.	0.58	0.44	1.31	10.38	8.4	1.23	18.09	7.5	2.52	108	310	6.2	7.3	+	0.58	33
11.	0.54	0.93	0.58	6.2	9.8	0.63	7.5	10.8	0.69	96	126	1	1.3	-	1.3	34
12.	0.6	0.9	0.66	8.8	10.2	0.86	9.5	9.8	0.96	80	176	1.3	2.1	-	0.6	34
13.	0.7	0.6	1.16	9.8	9.2	1.06	11.2	8.4	1.3	72	180	2.4	2.3	-	0.8	22
14.	0.7	0.5	1.4	9.3	11.8	0.78	9.4	9.7	0.96	85	170	1.2	1.5	-	1.2	15

E early peak velocity of mitral valve, A atrial peak velocity of mitral valve, E/A ratio of E and A values, E'early diastolic myocardial velocity, A late diastolic myocardial velocity, E'/ A'ratio of E' and A', IVRT: isovolumetric relaxation time, E DEC TIME: E velocity deceleration time, BASDAI: Bath Ankylosing Spondylitis Disease Activity Index, BASFI: Bath Ankylosing Spondylitis Functional Index, MR: Magnetic resonance imaging, CRP: C-reactive protein; ESR: Erythrocyte sedimentation rate .

In the contrast-enhanced cardiac MRI performed following the transthoracic TTE, cardiac pathology was identified in 4 out of the 14 patients. In one patient, intramural edema extending from the interventricular septum to the apex with contrast enhancement was observed (Figs. 1, 2). In another patient, intramural nodular contrast enhancement was found in the lateral wall of the left ventricle (Fig. 3). In two

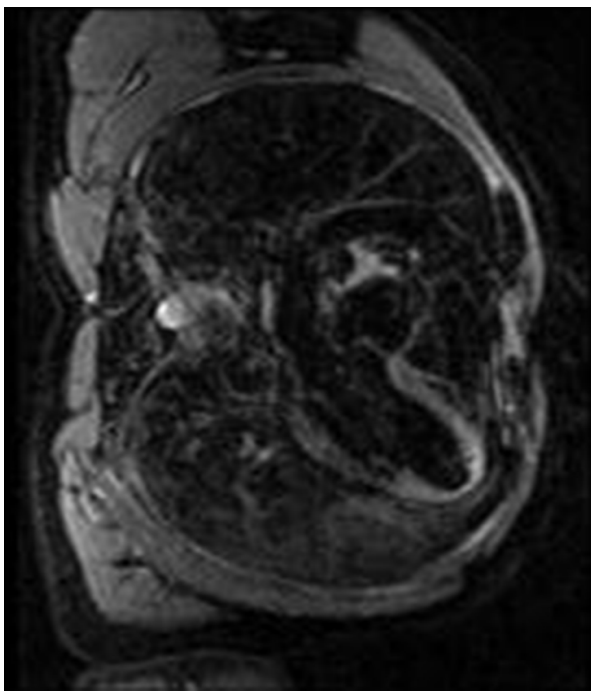

Figure 1. The True-FISP short-axis view showing a pattern of intramyocardial edema in the interventricular septum of the left ventricle.

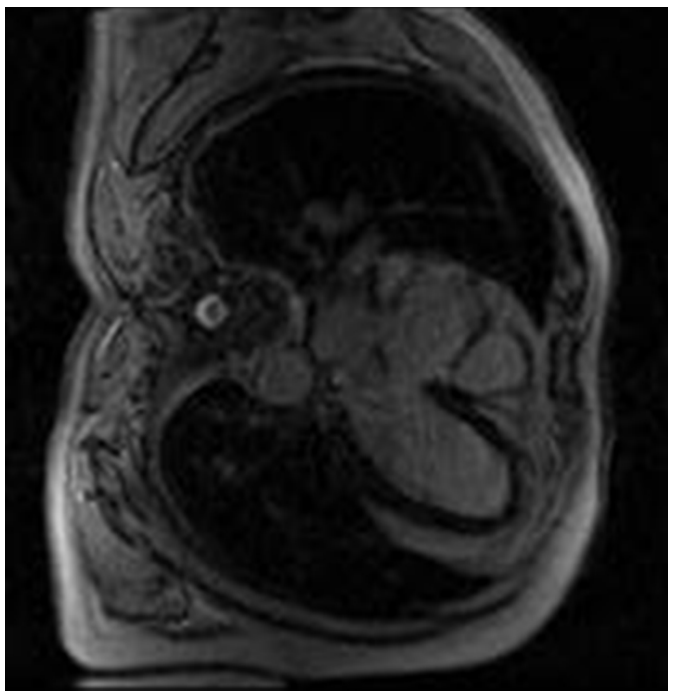

Figure 2. The delayed-enhanced four-chamber view showing intramyocardial late enhancement in the inferoseptal and apical walls of the left ventricle.

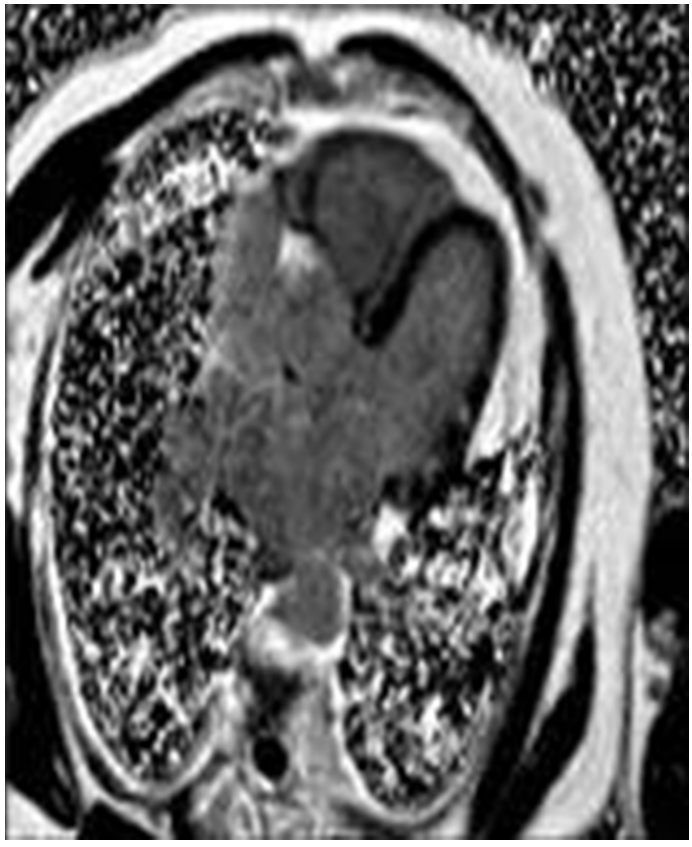

Figure 3. The delayed-enhanced four-chamber view showing multiple nodular areas of contrast enhancement in the mid-lateral segment of the left ventricle.

patients, contrast enhancement was observed in the cardiac apex, and in one of these patients, thinning of the anterior wall of the left ventricle was also noted.

When the transthoracic TTE findings were compared with myocardial contrast enhancement observed in the cardiac MRI using the Kruskal-Wallis test, no statistically significant differences were found between myocardial contrast involvement and either the E/A ratio or the IVRT time on TTE ($p>0.05$). However, a statistically significant correlation was found with E deceleration time ($p<0.05$) (Table 1).

When examining the activation index, the mean BASDAI value was 2.99 (range:1–6.4), and the mean BASFI value was 2.8 (range:0–7.6) (Table 1). When independent variables were analyzed individually, both BASDAI (β:0.331, $p<0.05$) and BASFI (β:0.673, $p<0.01$) were found to have a positive and significant effect on MR contrast agent involvement. However, when the overall regression model was examined, it was not statistically significant ($F=2.675$, $p>0.05$).

In the regression analysis evaluating the effect of CRP and ESR on MR contrast agent involvement, no statistical evidence supported an association between CRP or ESR and MR contrast enhancement.

Discussion

This study utilized cardiac MRI to assess myocardial involvement in ankylosing spondylitis (AS) patients who exhibited no cardiac symptoms but had transthoracic TTE findings consistent with diastolic dysfunction.

Myocardial involvement in AS was first identified in an autopsy study by Bulkey and Roberts,[10] which revealed intimal proliferation in the aortic root as a contributor to cardiac mortality and endarteritis. Additionally, the study demonstrated fibrosis development along the myocardial transmission system.[10] Myocardial fibrosis in AS is characterized by fibrosis in the perivascular area and interstitium without myocardial destruction. This fibrosis leads to reduced cardiac compliance and diastolic dysfunction.[11] When fibrosis affects the myocardial transmission system, it can result in conduction disorders such as first-degree atrioventricular block, high-degree atrioventricular block, right and left bundle branch blocks, arrhythmias, and extrasystoles.[4,12]

Another mechanism affecting heart rate in AS patients was identified by Toussirot et al.,[13] who demonstrated autonomic nervous system involvement. Their study found that patients with elevated inflammatory markers (CRP and ESR) and high BASDAI scores exhibited increased heart rates and decreased parasympathetic activity.

Male AS patients are at a heightened risk of coronary artery disease. Peters et al.[14] reported that the risk of myocardial infarction is 2–3 times higher in AS patients compared to the general population. This increased risk is attributed to systemic inflammatory mediators, with elevated CRP and tumor necrosis factor-alpha (TNF-α) levels found in these patients. High-sensitivity CRP (hsCRP) serves as a biomarker for cardiovascular risk assessment in AS.[1]

Coronary flow reserve, an indicator of microvascular function, is assessed in AS patients using transthoracic TTE by measuring the ratio of coronary artery flow rate after adenosine administration to the baseline rate.[15] Studies have shown reduced coronary artery flow in AS patients, correlating with diastolic dysfunction findings on TTE.[16] A positive correlation has also been established between coronary artery flow reserve and inflammatory markers such as hsCRP and TNF-α.[16]

A study by Bewerton linked diastolic dysfunction with myocardial involvement in transthoracic TTE findings in AS patients.[17] Histopathological and TTE evaluations, including postmortem analyses, revealed mild diffuse interstitial fibrosis in patients with early diastolic dysfunction. Brunner et al.[18] studied 100 AS patients and identified diastolic dysfunction in 26%, which was associated with age and hypertension. Additionally, Caliskan et al.[16] suggested that AS contributes to coronary artery reserve reduction in diastolic dysfunction.

The TTE markers of diastolic dysfunction include reduced E wave (early diastolic filling), A wave (late diastolic filling), E/A ratio, E' (early diastolic mitral annular velocity), and A' (late diastolic mitral annular velocity). Furthermore, increased isovolumetric relaxation time (IVRT) and deceleration time (DT) are observed.[11]

Although multiple studies have supported TTE findings of diastolic dysfunction in the presence of myocardial fibrosis, variability exists in these parameters.[11,19] While transthoracic TTE cannot precisely localize myocardial fibrosis, studies have shown a correlation between diffuse myocardial fibrosis and the severity of TTE-confirmed diastolic dysfunction.[20,21] Comparisons of TTE and contrast-enhanced cardiac MRI have demonstrated that late contrast enhancement, indicative of fibrosis, aligns with the severity of diastolic dysfunction observed on TTE.[22]

Endomyocardial biopsy (EMB) is the gold standard for confirming myocardial fibrosis, typically involving tissue sampling from the left ventricular posterior wall and the interventricular septum.[23] However, due to its invasive nature, EMB carries a complication risk of less than 6%, including hematoma, transient right bundle branch block, arrhythmias, tricuspid regurgitation, pulmonary embolism and, in rare cases (less than 1%), right ventricular perforation.[24] Reported mortality from EMB is approximately 1 in 1,000 patients.[25]

Gated-SPECT imaging, compared to TTE, provides additional insights into regional wall motion abnormalities and enables the measurement of left ventricular function and volume. Stress SPECT is the standard method for evaluating myocardial perfusion. In AS patients, gated-SPECT has been used to assess mid-wall myocardial motion impairments, which have been associated with microvascular dysfunction.[1]

Studies comparing SPECT and cardiac MRI have shown good correlation in calculating ejection fraction (EF) and left ventricular volume.[26,27] However, cardiac MRI has superior spatial resolution compared to gated-SPECT, making it more effective for detecting small transmural and myocardial infarcts.[28,29] Moreover, cardiac MRI can identify micro-infarcts and subendocardial infarcts that do not result in detectable wall motion abnormalities on SPECT.[30,31]

Cardiac MRI is a valuable imaging modality for assessing myocardial morphology and function in both ischemic and non-ischemic cardiomyopathies. It detects myocardial thickness abnormalities, myocardial edema in myocarditis, early contrast uptake in acute myocarditis, and late contrast uptake indicating myocardial fibrosis.[32] The pattern of contrast agent uptake varies based on fibrosis etiology: ischemic cardiomyopathies show subendocardial or transmural involvement in line with coronary artery distribution, whereas non-ischemic cardiomyopathies present with intramural or subepicardial involvement in a wedge-shaped pattern or longitudinal striae.[33,34] Histopathological studies have confirmed a correlation between fibrosis severity and myocardial contrast enhancement on late contrast imaging.[35,36] Myocardial fibrosis affecting ≥30% of myocardial tissue has been linked to increased mortality risk.[37]

In cases of diffuse myocardial fibrosis, conventional MRI sequences may be insufficient for diagnosis as they lack a normal myocardium reference point. Recently, T1 mapping techniques in cardiac MRI have been utilized for detecting and quantifying diffuse myocardial fibrosis. T1 mapping evaluates the difference in myocardial relaxation times before and after contrast administration, aiding in fibrosis assessment and severity grading.[38]

In this study, late contrast images in cardiac MRI effectively visualized myocardial fibrosis. Patients with myocardial contrast agent uptake on MRI exhibited TTE findings consistent with diastolic dysfunction. Additionally, inflammatory markers (CRP, ESR) and BASDAI/BASFI scores were significantly higher in patients with myocardial fibrosis than in those without.

Previous studies have reported associations between cardiac conduction abnormalities, disease duration, and the Bath Ankylosing Spondylitis Metrology Index (BASMI) as indicators of myocardial involvement. Moreover, significant relationships have been established between elevated CRP, sedimentation rates, and heart rate abnormalities.[4,13] Notably, cardiac involvement has been observed even in HLA-B27-positive patients without skeletal manifestations.[4]

This study identified diastolic dysfunction parameters in TTE findings of HLA-B27-positive AS patients with high BASDAI and BASFI scores, despite the absence of cardiac symptoms. These results suggest that cardiac MRI provides a non-invasive method to detect myocardial involvement in AS patients suspected of having myocardial fibrosis and diastolic dysfunction based on TTE findings.

Conclusion

Cardiac MRI is a valuable tool for detecting myocardial fibrosis in AS patients with diastolic dysfunction. The findings highlight the potential role of inflammation in myocardial involvement and the importance of early cardiac assessment in AS patients to prevent progression to overt heart disease.

Limitations

A limitation of this study was the low number of patients. In addition, there was no application of conventional angiography directed to coronary artery narrowness which can accompany myocardial fibrosis determined in patients. There is a need for further studies made with cardiac MRI on a more extensive patient population with ankylosing spondylitis disease.

Disclosures

Ethics Committee Approval: The study was approved by Fatih Sultan Mehmet Education and Research Hospital Ethics Committee (No: 4.34.59-20/5536, Date: 07.03.2012).

Informed Consent: Informed consent was obtained from all individual participants included in the study.

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References

1. Yalcin H, Guler H, Gunay E, Yeral N, Turhanoglu A, Bolaç E, et al. Left ventricular wall function abnormalities in patients with ankylosing spondylitis evaluated by gated myocardial perfusion scintigraphy. Rev Esp Med Nucl 2011;30:292–6.
2. Owlia MB, Mostafavi Pour Manshadi SM, Naderi N. Cardiac manifestations of rheumatological conditions: A narrative review. ISRN Rheumatol 2012;2012:463620.
3. Ozkan Y. Cardiac involvement in ankylosing spondylitis. J Clin Med Res 2016;8:427–30.
4. Dik VK, Peters MJ, Dijkmans PA, Van der Weijden MA, De Vries MK, Dijkmans BA, et al. The relationship between disease-related characteristics and conduction disturbances in ankylosing spondylitis. Scand J Rheumatol 2010;39:38–41.
5. Ungprasert P, Srivali N, Kittanamongkolchai W. Risk of coronary artery disease in patients with ankylosing spondylitis: A systematic review and meta-analysis. Ann Transl Med 2015;3:51.
6. Momeni M, Taylor N, Tehrani M. Cardiopulmonary manifestations of ankylosing spondylitis. Int J Rheumatol 2011;2011:728471.
7. Babür Güler G, Karaahmet T, Tigen K. Myocardial fibrosis detected by cardiac magnetic resonance imaging in heart failure: Impact on remodeling, diastolic function and BNP levels. Anadolu Kardiyol Derg 2011;11:71–6.
8. Kobayashi Y, Giles JT, Hirano M, Yokoe I, Nakajima Y, Bathon JM, et al. Assessment of myocardial abnormalities in rheumatoid arthritis using a comprehensive cardiac magnetic resonance approach: A pilot study. Arthritis Res Ther 2010;12:R171.
9. O'Neill SG, Woldman S, Bailliard F, Norman W, McEwan J, Isenberg DA, et al. Cardiac magnetic resonance imaging in patients with systemic lupus erythematosus. Ann Rheum Dis 2009;68:1478–81.
10. Bulkley BH, Roberts WC. Ankylosing spondylitis and aortic regurgitation. Description of the characteristic cardiovascular lesion from study of eight necropsy patients. Circulation 1973;48:1014–27.
11. Yildirir A, Aksoyek S, Calguneri M, Oto A, Kes S. Echocardiographic evidence of cardiac involvement in ankylosing spondylitis. Clin Rheumatol 2002;21:129–34.
12. Kazmierczak J, Peregud-Pogorzelska M, Biernawska J, Przepiera-Bedzak H, Goracy J, Brzosko I, et al. Cardiac arrhythmias and conduction disturbances in patients with ankylosing spondylitis. Angiology 2007;58:751–6.
13. Toussirot E, Bahjaoui-Bouhaddi M, Poncet JC, Cappelle S, Henriet MT, Wendling D, et al. Abnormal autonomic cardiovascular control in ankylosing spondylitis. Ann Rheum Dis 1999;58:481–7.
14. Peters MJ, Visman I, Nielen MM, Van Dillen N, Verheij RA, van der Horst-Bruinsma IE, et al. Ankylosing spondylitis: A risk factor for myocardial infarction? Ann Rheum Dis 2010;69:579–81.
15. Hozumi T, Yoshida K, Akasaka T, Asami Y, Ogata Y, Takagi T, et al. Noninvasive assessment of coronary flow velocity and coronary flow velocity reserve in the left anterior descending coronary artery by Doppler echocardiography: Comparison with invasive technique. J Am Coll Cardiol 1998;32:1251–9.
16. Caliskan M, Erdogan D, Gullu H, Yilmaz S, Gursoy Y, Yildirir A, et al. Impaired coronary microvascular and left ventricular diastolic functions in patients with ankylosing spondylitis. Atherosclerosis 2008;196:306–12.
17. Brewerton DA, Gibson DG, Goddard DH, Jones TJ, Moore RB, Pease CT, et al. The myocardium in ankylosing spondylitis. A clinical, echocardiographic, and histopathological study. Lancet 1987;1:995–8.

18. Brunner F, Kunz A, Weber U, Kissling R. Ankylosing spondylitis and heart abnormalities: Do cardiac conduction disorders, valve regurgitation and diastolic dysfunction occur more often in male patients with diagnosed ankylosing spondylitis for over 15 years than in the normal population? Clin Rheumatol 2006;25:24–9.
19. Kuloglu O, Bayram NA, Erten Ş, Durmaz T, Keles T, Sari C, et al. Assessment of left ventricular function by tissue Doppler imaging in patients with ankylosing spondylitis. Rheumatol Rep 2012;4:e6.
20. Su MY, Lin LY, Tseng YH, Chang CC, Wu CK, Lin JL, et al. CMR-verified diffuse myocardial fibrosis is associated with diastolic dysfunction in HFpEF. JACC Cardiovasc Imaging 2014;7:991–7.
21. Ellims AH, Iles LM, Ling LH, Hare JL, Kaye DM, Taylor AJ. Diffuse myocardial fibrosis in hypertrophic cardiomyopathy can be identified by cardiovascular magnetic resonance, and is associated with left ventricular diastolic dysfunction. J Cardiovasc Magn Reson 2012;14:76.
22. Moreo A, Ambrosio G, De Chiara B, Pu M, Tran T, Mauri F, et al. Influence of myocardial fibrosis on left ventricular diastolic function: Noninvasive assessment by cardiac magnetic resonance and echo. Circ Cardiovasc Imaging 2009;2:437–43.
23. Takaoka H, Funabashi N, Ueda M, Fujimoto Y, Kishimoto T, Ota S, et al. Diagnostic comparison of cardiac magnetic resonance with endomyocardial biopsy in patients with dilated cardiomyopathy: A segment-by-segment analysis. Int J Cardiol 2016;220:739–41.
24. From AM, Maleszewski JJ, Rihal CS. Current status of endomyocardial biopsy. Mayo Clin Proc 2011;86:1095–102.
25. Wu LA, Lapeyre AC 3rd, Cooper LT. Current role of endomyocardial biopsy in the management of dilated cardiomyopathy and myocarditis. Mayo Clin Proc 2001;76:1030–8.
26. Williams KA, Taillon LA. Left ventricular function in patients with coronary artery disease assessed by gated tomographic myocardial perfusion images. Comparison with assessment by contrast ventriculography and first-pass radionuclide angiography. J Am Coll Cardiol 1996;27:173–81.
27. Vallejo E, Dione DP, Sinusas AJ, Wackers FJ. Assessment of left ventricular ejection fraction with quantitative gated SPECT: Accuracy and correlation with first-pass radionuclide angiography. J Nucl Cardiol 2000;7:461–70.
28. Fathala A. Comparison of cardiac magnetic resonance with gated SPECT for evaluation of left ventricular function and volumes in patients with severe and multiple myocardial perfusion defects. J Saudi Heart Assoc 2010;22:203–8.
29. Mahrholdt H, Klem I, Sechtem U. Cardiovascular MRI for detection of myocardial viability and ischaemia. Heart 2007;93:122–9.
30. Wagner A, Mahrholdt H, Holly TA, Elliott MD, Regenfus M, Parker M, et al. Contrast-enhanced MRI and routine single photon emission computed tomography (SPECT) perfusion imaging for detection of subendocardial myocardial infarcts: An imaging study. Lancet 2003;361:374–9.
31. Mahrholdt H, Wagner A, Parker M, Regenfus M, Fieno DS, Bonow RO, et al. Relationship of contractile function to transmural extent of infarction in patients with chronic coronary artery disease. J Am Coll Cardiol 2003;42:505–12.
32. Friedrich MG, Marcotte F. Cardiac magnetic resonance assessment of myocarditis. Circ Cardiovasc Imaging. 2013;6:833–9.
33. McCrohon JA, Moon JC, Prasad SK, McKenna WJ, Lorenz CH, Coats AJ, et al. Differentiation of heart failure related to dilated cardiomyopathy and coronary artery disease using gadolinium-enhanced cardiovascular magnetic resonance. Circulation 2003;108:54–9.
34. Cummings KW, Bhalla S, Javidan-Nejad C, Bierhals AJ, Gutierrez FR, Woodard PK. A pattern-based approach to assessment of delayed enhancement in nonischemic cardiomyopathy at MR imaging. Radiographics 2009;29:89–103.
35. Rehwald WG, Fieno DS, Chen EL, Kim RJ, Judd RM. Myocardial magnetic resonance imaging contrast agent concentrations after reversible and irreversible ischemic injury. Circulation 2002;105:224–9.
36. Papavassiliu T, Schnabel P, Schröder M, Borggrefe M. CMR scarring in a patient with hypertrophic cardiomyopathy correlates well with histological findings of fibrosis. Eur Heart J 2005;26:2395.
37. Kwon DH, Halley CM, Carrigan TP, Zysek V, Popovic ZB, Setser R, et al. Extent of left ventricular scar predicts outcomes in ischemic cardiomyopathy patients with significantly reduced systolic function: A delayed hyperenhancement cardiac magnetic resonance study. JACC Cardiovasc Imaging 2009;2:34–44.
38. Burt JR, Zimmerman SL, Kamel IR, Halushka M, Bluemke DA. Myocardial T1 mapping: Techniques and potential applications. Radiographics 2014;34:377–95.