Assessment of Cardiac Involvement Using Colour Tissue Doppler Imaging In Patients with Juvenile Idiopathic Arthritis

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Abstract: Juvenile idiopathic arthritis (JIA) is the most common form of rheumatic disease in children. It is estimated that JIA affects up to 1 in 1000 children worldwide. Cardiac involvement in the form of pericarditis, pericardial effusion, myocarditis and valvular disease is found in JIA. The aim of this work was to assess the cardiac involvement in patients with JIA using colour tissue Doppler imaging. The study was carried out on 23 patients (5 boys and 18 girls) without any cardiac symptoms. They aged from 5 to 16 years old (mean 11.83± 3.68 years) and 23 apparently healthy age-sex matched control group. We used colour tissue Doppler imaging to assess cardiac involvement inpatients with JIA. No abnormal echo findings were detected among control group. Regarding cases group; no cases were detected showing hypokinisis,21.7% had mild pericarditis, 4.3% showed aortic affection and 17.4 % mitral affection. Diastolic dysfunction pattern of the right and left ventricles was detected, also there was significant increase in pulmonary artery pressure. the M-mode measurements showed mild increase in left ventricle diastolic dimension LVIDd **(**cases 3.8±0.4 cm, controls 3.5±0.41 cm, p=0.02) and systolic dimension LVIDs (cases 2.49±0.3 cm, controls 2.28±0.22 cm, p=0.01) mild hypertrophied left ventricle posterior wall LVPWd **(**cases 0.87±0.17 cm, controls 0.78±0.14 cm, p=0.05). Using tissue Doppler imaging in our study, the myocardial tissue velocities were studied, we detected statistical difference and decreased myocardial tissue velocity in JIA patients in S wave septum, S wave tricuspid valve, E and A waves in free wall, septum and tricuspid valves. No correlation between Tissue Doppler measurements and JADAS of the JIA patients.
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1. Introduction

Juvenile idiopathic arthritis (JIA) describes a clinically heterogeneous group of chronic inflammatory arthritides with onset under the age of 16 years. It is the most common rheumatic disease in childhood and young people with an incidence of approximately 1 in10 000 and a prevalence of 0.1% **(1).**

JIA encompasses several subtypes of arthritis with the most recent and widely used classification being that described by the International League of Associations for Rheumatology (ILAR)**(2).**

Over recent decades, there has been considerable interest in the long-term outcomes of individuals with chronic inflammatory arthritis and an area of particular concern has been the increased prevalence of cardiovascular disease (CVD) **(3).**

This increased risk is attributed to a higher prevalence of traditional cardiovascular risk factors and the role of systemic inflammation in the acceleration of atherosclerosis. In adults with RA, CVD is the leading cause of death, with cardiovascular mortality rates 50% higher than the general population **(4).**

However, the long-term risk of CVD for individuals with JIA is unclear and guidance on risk assessment is not available. The clinical course and prognosis in JIA is variable. Disease remission is increasingly achieved with modern approaches but for many patients this is a chronic disease requiring long-term immune-modulatory treatment and undoubtedly there is a marked impact on quality of life. The literature cites that at least one-third of adults with JIA have persistent active disease, particularly those with polyarticular course **(5).**

Since sustained systemic inflammation is known to accelerate atherosclerosis, individuals with JIA and in particular those with persistent inflammation may be at increased risk of CVD **(6).**

Aim of the Work

The aim of this work was to assess the cardiac involvement in patients with juvenile idiopathic arthritis using colour tissue Doppler imaging.

2. Patients and Methods:

After approval of the institutional review board (IRB) committee, this study was carried out on 46 subjects recruited from the inpatient department and

outpatient clinic of rheumatology &rehabilitation and pediatrics departments, Faculty of Medicine, Zagazig University. All of the parents of participants gave a written consent for ethical consideration.

The study group was composed of 23 patients of JIA (5 boys and 18 girls). They aged from 5 to 16 years old (mean 11.83± 3.68 years), and 23 healthy children (4 boys and 19 girls) with mean age of (11.91± 3.4) years served as the control group. All of the patients fulfilled the International League against Rheumatism criteria for diagnosis and classification of JIA (ILAR) (**2**). All patients and control group were selected randomly.

Children excluded from the study were those older than 16 years, children with congenital or rheumatic heart disease, children with family history of cardiac affection and children with diabetes mellitus or essential hypertension. Also children with history of any clinical evidence of cardiac manifestations, chronic lung disease, coronary artery disease, valvular or ischemic heart diseases have been excluded. All patients and controls were free of cardiovascular symptoms.

All patients had complete medical history, general and articular examinations. Patients were mainly treated with nonsteroidal anti-inflammatory drugs (NSAIDS), methotrexate (MEX), coricosteroids and some patients received salazopyrin and tumor-necrosis factor alpha blokers.

The following laboratory investigations were performed to all patients: complete blood picture (CBC)**,** C–Reactive protein (CRP)**,** erythrocyte sedimentation rate (ESR)**,** rheumatoid factor (RF)**,** anti-nuclear antibody (ANA) and fasting blood glucose (FBG).

Disease activity:

Was recorded on the day of examination using juvenile arthritis disease activity score-27 (JADAS-27) **(7).**

Echocardiographic examination:

All patients and controls were connected to electrocardiographic tracing of the echocardiographic machine GE Vivid 7 Multidimension and the following were acquired:

M mode Echocardiography

The following parameters were measured: assessment of cardiac dimensions using M-Mode & 2-D echocardiograph (IVS, LVDD, PW, Ao, LA) and ejection fraction (EF) & fraction of shortening (FS) were automatically calculated. (**8).**

Pulsed Wave Doppler echocardiography:

We measured peak early filling (E-wave) velocity, peak atrial filling (A-wave) velocity, and E/A ratio at the tip of the mitral leaflets from the 4-chamber apical view. **(9).** Also we measured peak early filling (E-wave) velocity, peak atrial filling (A-wave) velocity, and E/A ratio at the tip of the Tricuspid leaflets from the 4-chamber apical view. (**8**).

Tissue Doppler imaging:

Tissue velocity mode was selected to start the study. Tissue Doppler images were acquired and 3 cardiac cycles were captured per projection and the following was measured: Pulsed DTI was performed at the lateral aspect of the mitral annulus (DTI MV) from the apical 4-chamber view to obtain lateral peak systolic tissue velocity (S') and early (E') diastolic mitral annulus velocity and peak late diastolic annular velocity (A') waves. **(Image 1).** Also Pulsed DTI was performed at the interventricular septum (DTI septum) and the tricuspid valve free wall(DTI TV) from the apical 4-chamber view to obtain lateral peak systolic tissue velocity (S') and early (E') diastolic mitral annulus velocity and peak late diastolic annular velocity (A') waves at the interventricular septum and the tricuspid valve respectively.(10)

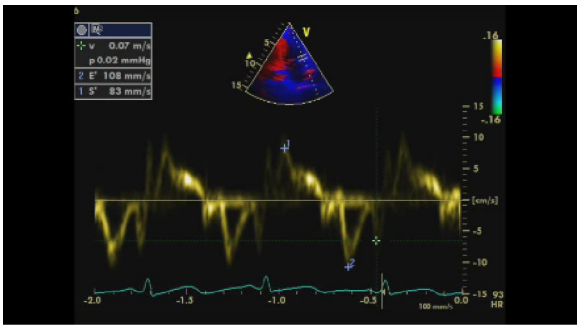

Image (1) Pulsed DTI at the lateral aspect of the mitral annulus

3. Results:

The demographic characteristics of the study population are summarized in Table 1. The clinical and the laboratory data of the JIA patients were represented in Table 2.

Table (1): Demographic data of the two studied groups

Variable	Cases (n=23)		Control (N=23)		T	P
Age (years): Mean±SD Range	11.83 ±3.68 5-16		11.91 ±3.4 5- 15		0.08	0.94 NS
Duration of symptoms: Mean±SD Range	66.78 ±42. 5 6-120		—		—	—
Variable	No	%	No	%	X2	P
Sex: Male Female	5 18	21.7 78.3	4 19	17.4 82.6	0.14	0.71 NS

Table (2): Clinical and laboratory data of the JIA patients

Variable	(n=23)	
VAS: Mean±SD Range	 7.04 ±2.64 1 -9	
Function: Mean±SD Range	 7.26 ±2.88 1-9	
Active joints: Mean±SD Range	 10.48 ±7.05 0-20	
ESR: Mean±SD Range	 72.43 ± 35.6 8-115	
JADAS: Mean±SD Range	 30.03 ±14.68 2.5-45.2	
Variable	No	%
Type of JIA: Systemic Oligoarticular Polyarticular	 8 2 13	 34.8 8.7 56.5
Ms: No Yes	 6 17	 26.1 73.9
Fever: No Yes	 8 15	 34.8 65.2
Rash: No Yes	 12 11	 52.2 47.8
S.C nodules: No Yes	 20 3	 87 13
Hb: Mean±SD Range	 11.99 ±1,07 9.7-13.8	
CRP: Mean±SD Range	 19.13 ±20.2 4-71	
ESR: Mean±SD Range	 72.43 ±35.6 8-115	
RF: -Ve +Ve	 15 8	 65.2 34.8
ANA: -Ve +Ve	 21 2	 91.3 8.7

Table 3 shows that there was statistical significance difference between cases and control in systolic blood pressure but no differences were found between them in heart rate and diastolic blood pressure.

Regarding echocardiography, Table 4 shows that no abnormal Doppler echo findings were detected among control group, regarding JIA patients no cases were detected showing hypokinesis, 5 patients (21.7%) had mild pericarditis, one patient (4.3%) showed aortic affection and 4 patients (17.4 %) had mitral affection. Table 5 shows the pulsed wave Doppler findings of the tricuspid and mitral valves and the maximal pressure gradient of tricuspid and pulmonary regurges, there was statistical significance difference between cases and control in all pulsed wave parameters except E/A ratio of TV, A wave velocity was significantly higher (cases 0.61 ± 0.15m/sec versus controls0.49 ± 0.1m/sec) andE wave velocity was significantly lower (cases 0.73 ± 0.22 versus controls 0.9 ± 0.07)in mitral valve. There was also significant difference between maximal pressure gradient of the tricuspid valve (**TR)** (23.26 ± 7.56 for cases 13.91 ± 3.68 for controls) and pulmonary valve (**PR)** (10.09 ± 3.58 for cases, 5.7 ± 1.18 for controls). Both reflect increase in pulmonary artery pressure.

In table 6, the M-mode measurements showed mild increase in left ventricle diastolic dimension **LVIDd (**cases 3.8±0.4 cm, controls 3.5±0.41 cm, p=0.02) and systolic dimension **LVIDs** (cases 2.49±0.3 cm, controls 2.28±0.22 cm, p=0.01) mild hypertrophied left ventricle posterior wall **LVPWd (**cases 0.87±0.17 cm, controls 0.78±0.14 cm, p=0.05).

Using tissue Doppler imaging in our study, the myocardial tissue velocities were studied at level of the free wall mitral valve (free wall), septum at level of atrioventricular valves (septum)and at level of tricuspid valve free wall (tricuspid valve), we detected statistical difference and decreased myocardial tissue velocity in JIA patients in S wave septum, S wave tricuspid valve, E and A waves in free wall, septum and tricuspid valves (table 7).

Correlation between Tissue Doppler measurements and JADAS of the JIA patients shows that there was –ve significant correlation between JADAS and both E TV and A FW. Also There were +ve significant correlation between it and IRT TV (Table 8).

Table (3): Cardiovascular parameters of the two studied groups:

Variable	Cases (n=23)	Control (n=23)	T	P
Heart rate: Mean±SD	83.13 ±7.6	79.34 ±8.17	1.63	0.11 NS
Systolic Bl. pressure: Mean±SD	112.5 ±6.35	107.9 ±6.43	2.44	0.02*
Diastolic Bl. pressure: Mean±SD	69.23 ±6.7	65.28± 7.17	1.93	0.06 NS

Table (4): Doppler echo findings of the JIA patients:

Variable	Cases (n=23)		Control (n = 23)		X^2	P
	No	%	No	%		
Pericarditis	5	21.7	0	0	—	—
Hypokinesis	0	0	0	0		
Aortic affection	1	4.3	0	0		
Mitral affection	4	17.4	0	0		

Table (5): pulsed wave parameters of the two studied groups:

Variable	Cases(n=23)	Control(n=23)	T	P
MVE: Mean ±SD	0.73 ±0.22	0.9 ±0.07	8.87	<0.001*
MVA: Mean ±SD	0.61 ±0.15	0.49 ±0.1	3.11	0.003**
EA ratio of MV Mean ±SD	1.26 ±0.26	1.87 ±0.26	7.84	<0.001*
TVE: Mean ±SD	0.67 ±0.1	0.6 ±0.05	3.04	0.004**
TVA: Mean ±SD	0.51 ±0.07	0.42 ±0.06	4.12	<0.001*
EA ratio of TV: Mean ±SD	1.35 ±0.25	1.42 ±0.23	1.06	0.29 NS
TR: Mean ±SD	23.26 ±7.56	13.91 ±3.68	4.81	<0.001*
PR: Mean ±SD	10.09 ±3.58	5.7 ± 1.18	3.52	0.001**

Table (6): M mode measurements of left ventricle in the two studied groups:

Variable	Cases(n=23)	Control(n=23)	T	P
IVSd: Mean ±SD	0.63 ±0.11	0.59 ±0.1	1.36	0.18 NS
IVSs: Mean ±SD	0.9 ±0.15	0.87 ±0.17	0.63	0.53 NS
LVIDd: Mean ±SD	3.8 ±0.4	3.5 ±0.41	2.48	0.02*
LVIDs: Mean ±SD	2.49 ±0.3	2.28 ±0.22	2.64	0.01*
LVPWd: Mean ±SD	0.87 ±0.17	0.78 ±0.14	2.05	0.05*
LVPWs: Mean ±SD	1.16 ±0.27	1.04 ±0.18	1.8	0.08 NS
EF: Mean ±SD	64.04 ±4.76	64.35 ± 6.64	0.18	0.86 NS
FS: Mean ±SD	34.43 ±3.4	34.48 ±5.3	0.03	0.97 NS
Ao: Mean ±SD	2.46 ±0.37	1.96 ±0.23	5.56	<0.001**
LA: Mean ±SD	2.13 ±0.33	2.01 ± 0.29	1.3	0.2 NS

Table (7): Tissue Doppler findings of the two studied groups:

Variable	Cases(n=23)	Control(II=23)	T	P
S wave FW: Mean±SD	9.78 ±1.48	10.54 ±1.31	1.85	0.07 NS
S wave septum: Mean±SD	8.07 ±0.56	8.62 ±0.57	3.29	0.002**
S wave TV: Mean±SD	12.74 ±0.72	13.87 ±0.45	6.44	<0.001**
E wave FW: Mean±SD	18.1 ±1.97	19.35 ±1.35	2.51	0.02*
E wave septum: Mean±SD	13.3 ±1.08	14.13 ±0.91	2.82	0.007**
E wave TV: Mean±SD	15.63 ±0.67	17.004 ±0.4	8.48	<0.001**
A wave FW: Mean±SD	6.14 ±0.49	6.64 ± 0.41	3.81	<0.001**
A wave septum: Mean±SD	5.56 ±0.36	6.02 ±0.22	5.14	<0.001**
A wave TV: Mean±SD	9.08 ±0.47	10.03 ±0.41	7.44	<0.001**
ICT FW: Mean±SD	79.78 ±3.3	77.8 ±2.88	2.17	0.04*
ICT Septum: Mean±SD	86.99 ±2.88	85.57 ±1.96	1.95	0.06 NS
ICT TV: Mean±SD	99.1 ±7.27	96.004 ± 7.8"?	1.39	0.17 NS
IRT FW: Mean±SD	69.76 ±3.74	64.82 ±4.01	4.32	<0.001**
IRT Septum: Mean±SD	76.61 ±6.5	73.06 ±6.21	1.9	0.07 NS
IRT TV: Mean±SD	66,67 ± 2.89	61.12 ± 3.16	6.21	<0.001**

Table (8): Correlation between Tissue Doppler measurements and JADAS of the JIA patients:

	JADAS	
	R	P
SFW	-0.05	0.83 NS
S septum	-0.04	0.88 NS
S TV	-0.19	0.38 NS
EFW	-0.17	0.43 NS
E septum	0.07	0.73 NS
E TV	-0.42	0.04*
A FW	-0.52	0.01*
A septum	-0.15	0.49 NS
A TV	-0.10	0.46 NS
ICT FW	-0.20	0.36 NS
ICTS	0.22	0.31 NS
ICT TV	-0.18	0.41 NS
IRT FW	-0.04	0.87 NS
IRTS	0.09	0.65 NS
IRT TV	0.56	0.005*

Discussion:

CVD is an important cause of mortality and morbidity in patients with JIA and possibly other forms of inflammatory arthritis. EULAR has published guidelines recommending that cardiovascular risk is assessed annually in those patients (11).

Children with JIA shares pathogenetic mechanisms with adult forms of rheumatoid arthritis. It remains unclear as to whether there is increased risk of CVD in JIA but clinical CVD is rare in children and may not manifest until adulthood. It is unclear as to whether the risk extends to all subtypes or only to those with sustained inflammation. One can hypothesize that adults with systemic JIA and persistent active polyarticular disease are likely to have the highest risk of CVD due to their higher level of systemic inflammation. Studies of adults with JIA can give valuable insight into the pathogenesis of CVD although clearly older patients reflect treatment strategies from several decades ago and may have different cardiovascular risk profiles to the emerging group of younger adults who have had greater access to potent immune-suppressive treatment from early in their disease course and are more likely to have improved disease control (6).

We aimed in this work to assess the cardiac involvement in patients with JIA using colour tissue Doppler imaging.

In this study, regarding cardiovascular parameters there was statistical significant difference between cases (112.5 ± 6.35)and control (107.9 ± 6.43) in systolic blood pressure but no differences were found between them in Heart rate and diastolic blood pressure, these findings are similar to Bharti et al,(12) who evaluated left ventricular systolic and diastolic function in 35children with JIA using echocardiography; patients with JIA had significantly higher systolic but they found also significant higher diastolic blood pressures. Such differences in systolic blood pressure in childhood may lead to overt hypertension as age advances. The explanation of this might be related to steroid therapy and nonsteroidal anti-inflammatory drugs intake. Hypertension is an important cause of cardiac mortality in adults with RA. So, patients with JIA need close blood pressure monitoring and early onset hypertension can be detected and treated.(13, 12)

Pericarditis is one of the exrta-articular manifestations of JIA which is asymptomatic and resolves completely in most of the patients. in this study, 5 cases (21.7 %) were detected showing mild pericarditis which was not manifested clinically. This is consistent with the study done by Ozer et al, (14) and Bernstein et al, (15), who reported the occurrence of pericarditis in 30 and 36 %, respectively, of their series of JIA patients. Also in one study of55 JIA patients, echo cardiographic evidence of pericardial effusions was noted in 16 of 30 patients (53%) with systemic-onset disease, 4 of 15 (27%) with polyarticular-onset JIA, and none of 10 with pauciarticular-onset JIA. Of the 20 patients with echocardiographic evidence of pericarditis, 9 had an enlarged cardiac silhouette, 8 had electrocardiographic (ECG) evidence of pericarditis, and 4 had pericardial friction rubs. In at least 11 of the 20 instances, a diagnosis of pericarditis could not have been made without echocardiography (6).

In contrast, Oguzetal (13), Bharti et al, (12) and Huppertz et al, (16) reported the absence of pericardial effusion in their JIA series.

In our study there were no cases with hypokinesis as we selected asymptomatic cases with no cardiopulmonary manifestations. One patient 4.3% showed aortic affection and four patients 17.4% mitral affection. This is similar to Coulson et al,(6) who found that valvulopathy has been documented in their JIA patients and can be in the form of aortic or mitral insufficiency. Also, our results are in accordance to Sircar et al, (17) who found 5 (10 %) and 4 (8 %) of their series of JIA showed mild mitral regurgitation and cusp thickening, respectively. Chen et al,(18) and Lee and Schueller (19) reported mitral and aortic affection in JIA patients. In contrast to our results, Oguz et al, (13) and Bharti et al,(12) did not found valvular heart affection in their groups of JIA patients.

This study showed the pulsed wave Doppler of tricuspid and mitral valve shows beginning of diastolic dysfunction pattern of the right and left ventricles respectively. This can be concluded from high statistical difference regarding A wave velocity in Mitral and Tricuspid valves between cases and controls, also there was high significant difference regarding E wave velocity in Mitral and Tricuspid valves but slight significant difference regarding E/A ratio in Mitral valve while no significant difference in Tricuspid valve.

This is similar to what Aslam et al,(20) who found similar results in his meta-analysis for detecting of diastolic dysfunction in rheumatoid arthritis patients. And also similar to Abdul Muizz et al,(21) who perform a cross sectional study for evaluation of diastolic dysfunction in rheumatoid arthritis patients and its relation to disease activity. Also it is similar to what Morris et al (22) who found in his study of right ventricle functions in rheumatoid arthritis patients with normal left ventricle. The non-significant difference in E/A ratio of the tricuspid valve in our study may be attributed to low number of studied cases.

There was also significant difference between maximal pressure gradient of the tricuspid valve(TR) (23.26 ± 7.56 for cases 13.91 ± 3.68 for controls) and pulmonary valve(PR) (10.09 ± 3.58 for cases 5.7 ± 1.18 for controls). Both reflect increase in pulmonary artery pressure. The cases are a little bit higher but still within the normal range. This is similar to what Aslam et al, (20) found in his meta-analysis of RA research that pulmonary artery pressure is significantly higher in RA patients than in controls higher mean systolic pulmonary artery pressure (mean difference 5.87 mm Hg [95% CI 4.36, 7.38]; P <0.00001). This is also similar to what Shariffet al,(23) found in his study of pulmonary artery pressure in rheumatoid arthritis patients.

The M-mode measurements showed mild increase in left ventricle diastolic dimension LVIDd (cases 3.8±0.4 cm, controls 3.5±0.41 cm, p=0.02) and systolic dimension LVIDs (cases 2.49±0.3 cm, controls 2.28±0.22 cm, p=0.01) mild hypertrophied left ventricle posterior wall LVPWd (cases 0.87±0.17 cm, controls 0.78±0.14 cm, p=0.05).

Larger left ventricular systolic and diastolic dimensions were noted in individuals with longer disease duration. It is important to note that all blood pressures and ejection fractions (EF) remained within the normal range. The mild increase in Left ventricle dimensions may be due to subclinical myocarditis. Though myocarditis occurs infrequently in JIA, it can lead to life-threatening congestive heart failure and arrhythmias. Statistics combined from three studies suggest that myocarditis was the cause of death in 9 of 81 patients (11%) who died with JIA. Svantesson et al, (24) reported the occurrence of symptomatic myocarditis in 4 of 320 patients (1.2%). The suggestive evidence of myocarditis was found in 10% of studied patients.

A number of non-invasive techniques like Doppler echocardiography, M mode, tissue Doppler imaging (TDI) and magnetic resonance imaging can be used for the evaluation of LV functions (25).

Using tissue Doppler imaging in our study, the myocardial tissue velocities were studied at level of the free wall mitral valve (free wall), septum at level of atrioventricular valves (septum) and at level of tricuspid valve free wall (tricuspid valve), we detected statistical difference and decreased myocardial tissue velocity in RA patients in S wave septum, S wave tricuspid valve, E and A waves in free wall, septum and tricuspid valves. These findings are similar to what Arslan et al,(26) found and concluded that tissue Doppler is better than conventional Doppler echocardiography in evaluation of cardiac functions in rheumatoid arthritis patients and can be used alone or combined with Doppler echocardiography in evaluating these patients. These results also similar to Alparslan et al,(27) who investigated the use of tissue Doppler of the right and the left ventricle similar to our study and detected decreased S wave, E wave and A wave velocities in tissue imaging of the tricuspid and mitral annuli. The isovolumetric contraction and relaxation times were mildly prolonged in free wall; Isovolumetric relaxation time in tricuspid valve was also prolonged. However other areas fail to show significant prolongation from controls this may be due to short disease duration as our cases are all pediatric age group. These results are similar to Arslan et al,(26) who compared tissue Doppler with conventional Doppler in active Rheumatoid arthritis patients; he found prolongation in is volumetric relaxation and contraction times but no statistically significant difference was found.

A correlation between tissue Doppler measurements and JADAS of the JIA patients was done showing –ve significant correlation between JADAS and both E TV and A FW. Also There were +ve significant correlation between it and IRT TV. This also similar to Di Franco et al,(28) who found that diastolic function worsen as the duration of disease lengthen and as the disease severity increases.

In conclusion, JIA is associated with cardiac abnormalities, affecting all cardiac layers, the pericardium, the endocardium and the myocardium even though cases are asymptomatic. Diastolic dysfunction (of both ventricles) is a common finding in JIA. Tissue Doppler echocardiography is a simple, noninvasive technique, can be used to detect subtle, asymptomatic myocardial abnormalities. It can be

used alone or add to the value of pulsed Doppler and M mode echocardiography to detect diastolic dysfunction in JIA patients.

References:

1. Ravelli A, Martini A. Juvenile idiopathic arthritis. The Lancet. 2007 Mar 9; 369(9563):767-778.
2. Petty RE, South wood TR, Manners P, et al. International League of Associations for Rheumatology classification of juvenile idiopathic arthritis: second revision, Edmonton 2001. J Rheumatol2004; 31(2): 390-392.
3. Colebatch-Bourn AN, Edwards CJ, Collado P, et al. EULARPReS Points to consider for the use of imaging in the diagnosis and management of juvenile idiopathic arthritis in clinical practice. Ann Rheum Dis. 2015 Nov; 74(11):1946-1957.
4. Avina-Zubieta JA, Choi HK, Sadatsafavi M, et al. Risk of cardiovascular mortality in patients with rheumatoid arthritis: a meta-analysis of observational studies. Arthritis Rheum. 2008;59:1690-1697.
5. Minden K, Niewerth M, Listing J, et al. Long-term outcome inpatients with juvenile idiopathic arthritis. Arthritis Rheum. 2002;46:2392-2401.
6. Coulson EI, Ng WF, Goff I, et al. Cardiovascular risk in juvenile idiopathic arthritis. Rheumatology. 2013; 1-9.
7. Consolaro A, Ruperto N, Bazso A, et al. Development and validation of a composite disease activity score for juvenile idiopathic arthritis. Arthritis Rheum 2009; 61:658–666.
8. Lopez L. Recommendations for quantification methods during the performance of a pediatric echocardiogram: a report from the pediatric measurements writing group of the American society of Echocardiography Pediatric and Congenital heart disease council. J Am Soc Echocardiogr. 2010;23:465–495.
9. Dokainish H, Rajaram M, Prabhakaran, et al. incremental valueof left ventricular systolic and diastolic function to determine outcome in patients with acute myocardial infarction. Echocardio-graphy2014; 31(5):569-578.
10. Cui W. et al. Systolic and diastolic time intervals measured from Doppler tissue imaging: normal values and Z-score tables, and effects of age, heart rate, and body surface area. J Am Soc Echocardiogr. 2008;21(4):361–370.
11. Peters MJL, Symmons DPM, McCarey D, et al. EULAR evidence based recommendations for cardiovascular risk management in patients with rheumatoid arthritis and other forms of inflammatory arthritis.2010.
12. Bharti BB, Kumar S, Kapoor A, Agarwal A, Mishra R, Sinha N. Assessment of left ventricular systolic and diastolic function injuvenile rheumatoid arthritis. J Postgrad Med. 2004; 50(4):262–265.
13. Oguz D, Ocal B, Ertan U, NarinH, Karademir S, Senocak F. Left ventricular diastolic functions in juvenile rheumatoid arthritis. Pediatric Cardiol. 2000, 21:374-377.
14. Ozer S, Alehan D, Ozme S, Bakkaloglu A, Soylemezoglu O,. Mitral and aortic insufficiency in polyarticular juvenile rheumatoid arthritis. Pediatr Cardiol. 1994, 15:151-153.
15. Bernstein B, Takahashi M, Hanson V, Cardiac involvement in juvenile rheumatoid arthritis. J Pediatr, 1984, 85:313-317.
16. Huppertz H, Voigt I, Muller-Scholden J, Sandhage K, Cardiac manifestations in patients with HLA B27-associated juvenile arthritis. Pediatr Cardiol. 2000, 21(2):141-147.
17. Sircar D, Ghosh A, Haldar S, Juvenile idiopathic arthritis, Indian Pediatr. 2006, 43917):429-433.
18. Chen YS, Yang YH, Lin YT, Chiang BL. A patient diagnosed with pauciarticular juvenile rheumatoid arthritis after a mechanical prospective valve replacement due to aortic regurgitation. J Microbial Infect. 2004, 37:200-202.
19. Lee SJ, Im HY, Schueller WC. HLA-B27positive juvenile arthritis with cardiac involvement preceding sacroiliac joint changes. Heart, 2001, 86:e19.
20. Aslam F, Salman j. Bandeali, et al. Diastolic Dysfunction in Rheumatoid Arthritis: A Meta-Analysis and Systematic Review. Arthritis Care & Research 2013; 65(4):534-543.
21. Abdul Muizz AM, Mohd Shahrir MS, Sazliyana S, et al. Across-sectional study of diastolic dysfunction in rheumatoidarthritis and its association with disease activity. Int J Rheum Dis2011;14:18–30.
22. Morris DA, Gailani M, Vaz Perez A, et al. Right ventricular myocardial systolic and diastolic dysfunction in heart failure with normal left ventricular ejection fraction. J Am Soc Echocardiogr 2011; 24: 886–897.
23. Shariff N, Kumar A, Narang R, et al. A study of pulmonaryarterial hypertension in patients with rheumatoid arthritis. Int J Cardiol 2007; 115:75–76.
24. Svantesson H, Bjorkhem G, Elborgh R. Cardiac involvement injuvenile rheumatoid arthritis. ActaPaediatrScand 2001;72:345-350.
25. Mandinov L, Eberli FR, Seiler C, et al. Diastolic heart failure. Cardiovasc Res 2000; 45:813–825.

26. Arslan S, Bozkurt E, Sari RA, et al. Use of tissue Doppler and its comparison with other conventional Doppler techniques in the assessment of diastolic functions in patients with active rheumatoidarthritis. Rheumatol Int 2006; 26:229–233.
27. Alparslan B, Cengiz K, Necmi A et al. Tissue Doppler Imagingin the Evaluation of the Left and Right Ventricular Diastolic Functions. Echocardiography, 2007, 24(5):485-493.
28. Di Franco M, Paradiso M, Mammarella A et al. Diastolicfunction abnormalities in rheumatoid arthritis. Evaluation by Echo Doppler Transmitral flow and pulmonary venous flow: relation with duration of disease. Ann Rheum Dis 2000; 59:227–229.

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