Available Online at http://www.recentscientific.com

ISSN: 0976-3031

International Journal of Recent Scientific Research
Vol. 6, Issue, 4, pp.3690-3701, April, 2015

International Journal of Recent Scientific Research

RESEARCH ARTICLE

DETECTION OF EARLY LEFT VENTRICULAR DYSFUNCTION IN TYPE 1 DIABETES MELLITUS BY STRAIN AND STRAIN RATE IMAGING

Walaa F. Abdaziz*, Ghada M. Sultan, Rania S. El zayat and Hazem I. Maagouz

Cardiology Department –Faculty of Medicine-Menoufia University

ARTICLE INFO

Article History:

Received 14th, March, 2015
Received in revised form 23th, March, 2015
Accepted 13th, April, 2015
Published online 28th, April, 2015

Key words:

Type 1 Diabetes Mellitus, Echocardiography left Ventricular function, Strain

ABSTRACT

Objectives: This study was done to assess early left ventricular affection in asymptomatic type 1diabetes by echocardiographic strain and strain rate technique.

Background: Echocardiography is an excellent non-invasive and practical imaging tool for defining cardiac structure and function and allows 'real-time' visualization of the cardiac cycle. Two dimensional speckle tracking imaging allows non-invasive assessment of myocardial strain and has been shown to identify global and regional abnormalities in myocardial properties and has the advantage that it allows the measurement of all principal LV strains in an angle independent manner.

Methods: 50 subjects with type 1 DM(aged 11.64 ± 2.81years) and 20 healthy age-matched persons served as control subjects. In each patient an echocardiographic study with strain and strain rate imaging was performed.Analysis of LV deformation data included assessment of systolic strain, systolic and diastolic strain rate obtained from the apical,mid and basal segments of LV (apical two, apical three and apical four view).

Results: The result of the present study have demonstrated the presence of subclinical left ventricular systolic and diastolic dysfunction in patients with type 1diabetes mellitus, with no diabetic related complication. Significant subclinical LV dysfunction was present in 30 % diabetic patients about 15 patients of TIDM [9 female and 6 male] ($P<0.001$)

Conclusion: Despite a normal LV EF and normal LV diastolic measures with 2 D Echocardiography, the diabetic patients showed impairment of LV longitudinal strain and strainrate. The presence of diabetes was an independent predictor of impairment of LV longitudinal strain and strain rate and that give more sensitivity to strain and strain rate over conventional Doppler in earlier detection of systolic function.

INTRODUCTION

Diabetes Mellitus describes a metabolic disorder of multiple etiologies characterized by chronic hyperglycemia with disturbances of carbohydrate, fat and protein metabolism resulting from defects in insulin secretion, insulin action, or both. (1). A number of experimental, pathologic, and epidemiologic studies sup-port the existence of diabetic Cardiomyopathy (2), the clinical diagnosis of DCM is made when systolic and diastolic left ventricular dysfunction are present in diabetic patients without other known cardiac disease (3). other conditions where new cardiac imaging technologies have identified subclinical heart disease (4), myocardial backscatter and strain characteristics in patients with diabetes mellitus have been shown to be abnormal (5). Treatment to reverse this disorder is more likely to be effective at an early (preclinical) stage, defining the mechanism of diabetic cardiomyopathy may be important to its selective treatment (6). Cardiovascular autonomic neuropathy (CAN) is a common complication of diabetes that confers considerable morbidity and mortality (7, 8, 9). In type 1 diabetes mellitus, cardiovascular mortality is usually due to coronary artery disease (CAD), heart failure, or hypertension (10, 11). Abnormalities of LV function primarily reflect a diastolic abnormality which is an early sign of diabetic heart muscle disease preceding systolic damage.(12) suggesting direct metabolic effects on the heart—metabolic heart disease (13). Left ventricular twist, torsion, and strain provide information on regional and global cardiac tissue deformation (14) Strain rate (SR) imaging is a new method for detection of segmental myocardial contraction or stretching.(15)

PATIENTS AND METHODS

The study comprised 70 subjects referred to El Menoufia University Hospital for echocardiographic assessment, during the period of 24 months from October 2012 to October 2014,they were examined in a single centre(Cardiology Department, Menoufia University, Egypt) using a GE vivid 9 machine, divided into two groups group (1) representing the

*Corresponding author: **Walaa F. Abdaziz**
Cardiology Department –Faculty of Medicine-Menoufia University

type 1 diabetic patients (50 patients) and group (2) representing the control group (20 subjects).

Inclusion Criteria

1. Asymptomatic type 1 diabetes mellitus. The diagnosis of diabetes based on one or more of the following criteria : hemoglobin A1C ≥ 6.5% or fasting plasma glucose (FPG) ≥126 mg/ dL or 2-hour plasma glucose ≥200 mg/dL during an oral glucose tolerance test(OGTT) or a random plasma glucose ≥200 mg/dL or intake of anti diabetic drugs. (16,17,18)
2. Sinus rhythm.
3. Normal LV systolic function (LV ejection fraction [EF]>60%).

Exclusion criteria

Patients having any of the following criteria will be excluded from the study.

1. Left ventricular (LV) ejection fraction < 60%. Or any regional wall motion abnormality.
2. Arrhythmia.
3. Valvular or congenital heart diseases.
4. Pericardial diseases.
5. Endocrine or other system disease than D.M.

Methods

All the studied groups were subjected for

1. Full history taking with special emphasis on
   - Age
   - Gender
   - History of type1diabetes mellitus include duration of DM, treatment, dose of insulin, family history and complications of DM.
2. Clinical examination.
3. Weight and height will be measured and used to calculate the body mass index (BMI.)
4. Clinical manifestation of valvular and congenital heart disease.
5. Resting 12 leads surface electrocardiogram to exclude arrhythmia.
6. Laboratory assessment of fasting blood glucose was done by Oxidase test.
7. Quantitative Colorimetric Determination of Glycosylated hemoglobin in Whole Blood provided by (Stanbio Glycohemoglobin, Boerne, Texas). In the method presented, a preparation of hemolyzed whole blood is mixed with a weakly binding cation-exchange resin. The non glycosylated hemoglobin (HbA1C) binds to the resin, leaving (HbA1) free to be removed by means of a resin separator in the supernate. The percent of HbA1 is determined by measuring the absorbance values at415 nm of the HbA1 fraction and of the total Hb fraction. Calculating the ratio of absorbanccs (R), and comparing this ratio to that of glycohemoglobin standard carried through the same procedure. Results are express as HbA1, but can be converted or derived as HbA1C by using a conversion factor or when using a HbA1C value for the standard Results may also be reported as Hemoglobin Alc When compared to the reference Alc method**.** (19, 20)
8. Trans thoracic echocardiography:
   All Study participants underwent standard echocardiography with Doppler studies, using a GE vivid 9 machine all subjects were examined in the left lateral Decubitus position according to the recommendations of the American Society of Echocardiography. (21)

Assessment of LV Dimensions

- M-mode measurements were obtained from left parasternal with special attention was given not to include overlying trabeculations in the ventricular septum or posterior wall measurements, which may overestimate thickness.
- Measurements were taken at the end diastole –defined as the beginning of the QRS complex –but preferably using the widest LV cavity diameter, and at the end systole - using the narrowest LV cavity diameter.
- The diastolic measurements obtained were the interventricular sepal wall thickness, the LV internal diameter at end diastole and posterior wall thickness. In systole, the LV systolic diameter was measured.

Systolic function assessment

The measurement of LV ejection fraction (%) and LV fractional shortening (%) were performed to evaluate the systolic function using M mode tracing. Ejection fraction (EF%) was calculated as percentage change of LV chamber volumes between diastole and systole from apical four – and two – chamber views using modified biplane Simpson's rule. (22) NB: Ejection fraction ≥ 55 % indicated a normal systolic function and<55 % is considered systolic dysfunction. (22)

Diastolic Function assessment

Pulsed – wave Doppler echocardiography was used to evaluate diastolic LV function; Doppler studies were recorded from the apical 4- chamber view, with a sample volume positioned within the inflow portion of the LV, midway between the annular margins of mitral valve. Mitral velocity profiles were digitized from velocity of the Doppler tracings.

Waves measured by pulsed conventional Doppler

- The peak E (early rapid ventricular filling) wave velocity: It is an early filling wave occurs in the early diastole as the pressure in the left ventricle falls below that in the left atrium (N=50–85 cm / sec).
- The Peak A (late ventricular filling) wave velocity: It is a late filling wave occurs as the left atrial contraction causes acceleration of the flow from the left atrium to the left ventricle (N = 35 – 5 0 cm/sec). (23)
- E / A ratio: The ratio between the early filling and the late filling wave (N = 1 – 2). (23)

- Deceleration time (DT) Were made by computer software by placing the caliper to the peak of the E wave and by following the deceleration slope of the E wave down to the intersection with the A wave (N =160-220 ms). N.B Diastolic dysfunction is considered if DT > 220 ms and E / A ratio < 1.(24)

Two-dimensional speckle tracking analysis

All 2D grey-scale echocardiographic images were obtained using second harmonic imaging. Three LV apical views, apical four-chamber, two chamber, and apical three views were acquired at high frame rates (range: 59–82 frame/s; mean 72+6 frame/s), three consecutive cardiac cycles were acquired during a breath hold and digitally stored in a hard disk for off-line analysis. In order to measure the timing of cardiac events, LV inflow and outflow velocities were recorded using pulsed-wave Doppler echocardiography. Using 2D strain software (GE, Vivd 9), prope M5S, the endocardial border in the end systolic frame was manually traced. A region of interest was then drawn to include the entire myocardium. The software algorithm automatically segmented the LV into six equidistant segments and selected suitable speckles in the myocardium for tracking. The software algorithm then tracked the speckle patterns on a frame by frame basis using the sum of absolute difference algorithm. Finally, the software automatically tracks and accepts segments of good tracking quality and rejects poorly tracked segments, while allowing the observer to manually override its decisions based on visual assessments of tracking quality. The average value of strain at each level (basal, middle, and apical) and global strain obtained from averaging the strain values of 18 LV segments was calculated. The average value of SR at each level (basal, middle, and apical) and global SR obtained from averaging the SR values of 18 LV segments was calculated.(25)

Statistics

Statistical presentation and analysis of the present study was conducted, using the mean, standard deviation and chi-square test by SPSS V.20

1. **Mean value** $\left(\bar{x}\right)$ **:** the sum of all observations divided by the number of observation:

   $$\left(\bar{x}\right)= \frac{\Sigma x}{n}$$

   Where Σ = sum & n = number of observations.
2. **Standard Deviation [SD]:**

   It measures the degree of scatter of individual varieties around their mean:

   $$SD= \sqrt{\frac{\Sigma|x-x|^{-2}}{n-1}}$$
3. **Standard student "t test",** test of significance of the difference between two means:

   $$t = \frac{\bar{X}-\bar{X}_2}{\sqrt{\frac{(SD_1)^2}{n_1}+\frac{(SD_2)^2}{n_2}}}$$

   The calculated "t" was compared with tabulated one at different levels of significance at the degree of freedom (DF):

DF = (D + n_2) -*2* Where:

$\bar{x}$ = The mean value of group L

$\bar{x}_2$ = The mean value of group II.

SD_1 = the standard deviation of group I.

SD_2 = the standard deviation of group II.

n1 = the number of observations of group L

n2 = the number of observations of group II.

4. **Chi-square** the hypothesis that the row and column variables are independent, without indicating strength or direction of the relationship. Pearson chi-square and likelihood-ratio chi-square. Fisher's exact test and Yates' corrected chi-square are computed for 2x2 tables.

Chi-square test

For comparison between two groups as regards qualitative data.

$$X^2 = \Sigma \frac{(O-E)^2}{E}$$

Where:

Σ = Summation.

O = Observed value.

$$E = \text{Expected value} = \frac{\text{vertical total X Horizontal total}}{\text{grand total}}$$

5. **Linear Correlation Coefficient [r]**

$$r = \frac{\Sigma(X-\bar{X})(y-\bar{y})}{\sqrt{\{\Sigma(X-\bar{x})^2\}\{\Sigma(y-\bar{y})^2\}}}$$

Where

X= Independent variable.

Y= Dependent variable

Linear Correlation coefficient was used for detection of correlation between two quantitative variables in one group

The correlation was expressed as

- **P value< 0.05**wasconsideredstatistically mildly significant**.**
- **P value< 0.001**wasconsideredstatistically highly significant.
- **P value> 0.05**wasconsideredstatistically non significant**.**

All these tests were used as tests of significance at (P<0.05).

6. **ROC-curve:-**Receiver Operating Characteristic curve analysis

Sensitivity

Probability that the test results will be positive when the disease is present (true positive rate, expressed as a percentage).

Specificity

Probability that the test results will be negative when the disease is absent (true negative rate, expressed as a percentage).

PPV

Positive Predictive value (probability that the disease is present when the test is positive).

NPV

Negative Predictive value (probability that the disease is present when the test is negative).

Accuracy

The ratio of the true positive and true negative on all patients.

RESULTS

The study comprised 70 subjects referred to El Menoufia University Hospital for echocardiographic assessment, during the period of 24 months from October 2012 to October 2014, they were examined in a single centre (Cardiology Department, Menoufia University, Egypt) using a GE vivid 9 machine, they were divided into two groups: group (1) representing the diabetic patients (50patients) and group (2) representing the control group (20subjects).

Table 1 Age distribution

Age	Cases	Control
Range	7 – 17	8 – 19
Mean ± SD	11.64 ± 2.81	12.40 ± 3.19
T. test	0.967	
P. value	0.329	

Table 2 Gender distribution

Sex		Cases	Control	Total
Male	N	20	10	30
	%	40.0%	50.0%	42.9%
Female	N	30	10	40
	%	60.0%	50.0%	57.1%
Total	N	50	20	70
	%	100.0%	100.0%	100.0%
Chi-square		X^2	0.583	
		P-value	0.309	

Table 3 Comparison between patients and controls as regard laboratory data.

	Patients Mean± SD	Control Mean± SD	T-test T	p-value
Glycosylated hemoglobin %	5.74±0.25	5.66±0.27	1.768	0.188
Fasting glucose(mg/dl)	178.16±28.81	99.5±15.64	38.214	<0.001*

Demographic and clinical data

By comparing the two groups using the least significant difference between groups as regard age, sex, heart rate, duration of diabetes and body mass index ,there was statistically no significant difference between the group of type1 diabetes mellitus and the control as showing in table (1,2)

Laboratory data

Comparing the 2 groups as regard fasting blood glucose and glycosylated hemoglobin, there was statistically significant difference between the group of type 1 diabetes mellitus and the control. It was found that, the group of type1diabetes mellitus was significantly higher than the control ($P<0.05$)as regard to fasting blood glucose level and no significance to glycosylated hemoglobin, as showing in table (3).

Conventional echocardiography

Comparing the 2 groups as regard echocardiographic parameters (interventricular septal thickness in diastole, left systolic dimension, left ventricular end diastolic dimension, left ventricular posterior wall thickness in diastole, ejection fraction, fractional shortening, Ewave, Awave, E/Aratio), there was statistically significant difference between the group of type 1 diabetes mellitus and control as regard E wave and there was statistically no significant difference between the group of type1 diabetes mellitus and the control as regard interventricular septal thickness in diastole, left ventricular end systolic dimension, left ventricular end diastolic dimension, left ventricular posterior wall thickness in diastole, ejection fraction, fractional shortening , A wave, E/A ratio as showing in table (4)

Analysis of LV Deformation Data

Systolic strain

Comparing both groups as regard segmental systolic strain in the basal, mid, and apical segments of the anterior wall, inferior wall lateral wall, septal wall, posterior wall and antero septal wall, it was found that, there is a statistically highly significant difference between the group of type 1diabetes mellitus and the control with impairment of the longitudinal systolic strain at the level of global strain and basal septal, mid lateral, mid inferior, apical inferior, apical anterior and mid anterior myocardial segments in the diabetic group in comparison with the control as showing in tab(5 A,B).

Systolic Strain Rate

Comparing both groups as regard as regard segmental systolic strain rate in the basal ,mid, and apical segments of the anterior wall, inferior wall, lateral wall, septal wall, posterior wall and antero septal wall ,it was found that, there is a statistically highly significant difference between the group of type1 diabetes mellitus and the control with impairment of the longitudinal systolic strain rate at the level of basal septal, apical septal, basal lateral, apical anterior and mid anterior segments in the diabetic group in comparison with the control as showing in table(6A,B)

Table 4 Comparison between groups as regard conventional Echocardiography data showing the diabetic group was significantly higher than control group as regard E (cm/s).

Items		Range			Mean	±	S. D	t. test	p. value
LVEDD(mm)	Cases	3.50	–	4.40	4.140	±	0.405	1.462	0.148
	Control	3.80	–	4.70	4.285	±	0.285		
LVESD(mm)	Cases	1.80	–	3.00	2.466	±	0.290	1.374	0.174
	Control	2.10	–	3.50	2.580	±	0.367		
LVSWT(mm)	Cases	0.60	–	0.85	0.715	±	0.064	.000	1.00
	Control	0.65	–	0.80	0.715	±	0.048		
LVPWT(mm)	Cases	0.60	–	0.80	0.704	±	0.055	1.236	0.270
	Control	0.65	–	0.80	0.720	±	0.052		
LVEF%	Cases	60.00	–	78.00	69.98	±	4.288	0.059	0.809
	Control	63.00	–	77.00	70.25	±	4.024		
FS%	Cases	30.00	–	44.00	38.70	±	2.943	3.406	0.069
	Control	30.00	–	41.00	37.20	±	3.381		
E(cm/s)	Cases	5.90	–	8.00	6.994	±	0.564	12.336	0.001*
	Control	6.80	–	8.00	7.480	±	0.396		
A(cm/s)	Cases	3.10	–	5.00	4.050	±	0.457	2.401	0.126
	Control	3.30	–	5.00	4.230	±	0.386		
E / A ratio	Cases	1.35	–	2.06	1.727	±	0.164	1.380	0.244
	Control	1.53	–	2.06	1.777	±	0.147		

***EF:** ejection fraction, **FS:** fractional shortening, **IVSD:** interventricular septum in diastole, **LVPWD:**left ventricular posterior wall thickness in diastole, **LVEDD:** left ventricular end diastolic dimension, **LVESD:**left ventricular end systolic dimension, **E**: early mitral inflow velocity; **A:** atrial mitral inflow velocity; (**P value>0.05** insignificant, **P<0.05**significant, **P<0.01**highly significant.*)

Table 5A Comparison between both groups as regard systolic strain of the selected segments in both groups shows Significant reductions in systolic strain at the level of basal septum, mid septal, apical septal, apical lateral, mid lateral,Basal lateral, basal posterior, mid posterior, apical posterior and apical anteroseptal segments in the diabetic group in comparison with the control.

Systolic strain		Range			Mean	±	S. D	t. test	p. value
Basal septal	Cases	1.94	–	33.93	18.62	±	4.69	7.431	0.008*
	Control	13.74	–	29.07	21.82	±	3.71		
Mid septal	Cases	1.36	–	34.84	20.72	±	4.80	1.851	0.178
	Control	16.04	–	29.28	22.32	±	3.47		
Apical septal	Cases	13.55	–	38.66	23.63	±	6.08	0.45	0.505
	Control	17.08	–	32.55	24.62	±	3.90		
Apical lateral	Cases	3.71	–	37.43	22.13	±	7.48	0.701	0.405
	Control	13.40	–	31.94	23.67	±	5.24		
Mid lateral	Cases	7.96	–	32.43	19.53	±	6.10	4.379	0.040*
	Control	16.13	–	30.35	22.69	±	4.55		
basal Lateral	Cases	9.07	–	35.12	18.58	±	5.70	1.466	0.230
	Control	16.62	–	28.24	20.24	±	3.39		
Basal Posterior	Cases	1.66	–	38.65	18.60	±	6.71	1.279	0.262
	Control	1.98	–	34.88	20.68	±	7.52		
Mid posterior	Cases	6.50	–	41.05	18.76	±	5.99	0.035	0.851
	Control	6.50	–	32.20	19.06	±	5.97		
Apical posterior	Cases	9.59	–	43.92	20.97	±	6.40	0.01	0.919
	Control	13.50	–	27.22	21.13	±	4.33		
Apical anteroseptal	Cases	1.22	–	39.81	21.33	±	7.11	0.435	0.512
	Control	8.80	–	33.30	22.54	±	6.46		

Table 5B Comparison between both groups as regard systolic strain of the selected segments in both groups shows significant reductions in systolic strain at the level of mid antero septal, basal antero septal, basal inferior, mid inferior, apical inferior, apical anterior, mid anterior, basal anterior segments and global LV in the diabetic group in comparison with the control.

Systolic strain		Range			Mean	±	S. D	t. test	p. value
Mid anteroseptal	Cases	1.63	–	36.32	18.50	±	6.83	1.739	0.192
	Control	3.46	–	29.59	20.86	±	6.65		
Basal anteroseptal	Cases	0.26	–	40.21	17.26	±	7.52	0.109	0.743
	Control	0.26	–	28.44	17.91	±	6.96		
Basal inferior	Cases	2.09	–	29.60	19.67	±	4.58	0.251	0.618
	Control	5.50	–	28.44	20.33	±	5.97		
Mid inferior	Cases	8.34	–	28.11	21.00	±	3.79	10.248	0.002*
	Control	10.42	–	31.00	24.48	±	4.84		
Apical inferior	Cases	5.10	–	36.63	23.55	±	5.57	5.23	0.025*
	Control	19.19	–	31.94	26.74	±	4.35		
Apical anterior	Cases	3.04	–	43.02	21.27	±	8.93	6.91	0.011*
	Control	15.83	–	33.31	26.84	±	4.88		
Mid anterior	Cases	1.25	–	36.11	19.33	±	8.34	3.879	0.05*
	Control	18.18	–	29.88	23.16	±	3.63		
Basal anterior	Cases	1.72	–	38.83	19.32	±	7.06	0.803	0.373
	Control	14.89	–	25.31	20.79	±	2.83		
Global LV	Cases	5.80	–	31.20	19.70	±	4.26	19.072	0.001*
	Control	16.80	–	32.02	24.69	±	4.46		

Table6A,B Comparison between both groups as regard global strain rate of the selected segments shows significant reductions in global strain rate at the level of basal septal, apical septal ,basal lateral ,apical anterior and mid anterior myocardial segments in the diabetic group in comparison with the control.

Peak systolic strain rate		Range			Mean	±	S. D	t. test	p. value
Basal Septal	Cases	0.51	–	3.83	1.29	±	0.61	11.731	0.001*
	Control	0.88	–	2.94	1.87	±	0.69		
Mid Septal	Cases	0.42	–	3.32	1.40	±	0.51	3.057	0.085
	Control	1.08	–	2.27	1.62	±	0.38		
Apical Septal	Cases	0.21	–	2.68	1.58	±	0.52	3.677	0.05*
	Control	0.97	–	2.81	1.85	±	0.51		
Apical lateral	Cases	0.4	–	2.99	1.59	±	0.63	0.129	0.721
	Control	0.84	–	2.09	1.64	±	0.37		
Mid lateral	Cases	0.44	–	2.38	1.39	±	0.43	0.008	0.929
	Control	0.99	–	1.88	1.39	±	0.28		
Basal lateral	Cases	0.32	–	2.75	1.52	±	0.49	3.891	0.05*
	Control	1.12	–	3.14	1.81	±	0.68		
Basal posterior	Cases	0.63	–	3.3	1.67	±	0.70	2.999	0.088
	Control	0.95	–	6.73	2.08	±	1.24		
Mid posterior	Cases	0.33	–	3.57	1.45	±	0.56	2.772	0.101
	Control	0.37	–	2.02	1.21	±	0.42		
Apical posterior	Cases	0.62	–	4.03	1.61	±	0.73	0.004	0.952
	Control	0.64	–	2.8	1.62	±	0.65		
Apical anteroseptal	Cases	0.47	–	3.47	1.56	±	0.68	0.77	0.383
	Control	0.77	–	3	1.72	±	0.69		

Table 6 B

Peak systolic strain rate		Range			Mean	±	S. D	t. test	p. value
Mid anteroseptal	Cases	0.12	–	2.13	1.26	±	0.45	0.157	0.693
	Control	0.39	–	1.99	1.31	±	0.46		
Basal anteroseptal	Cases	0.08	–	2.58	1.40	±	0.49	0.874	0.353
	Control	0.91	–	2.21	1.52	±	0.42		
Basal inferior	Cases	0.06	–	2.3	1.46	±	0.47	2.23	0.140
	Control	1.1	–	2.1	1.63	±	0.29		
Mid inferior	Cases	0.32	–	2.99	1.48	±	0.41	1.499	0.225
	Control	1.19	–	2.2	1.60	±	0.24		
Apical inferior	Cases	0.12	–	2.47	1.66	±	0.44	3.363	0.071
	Control	0.99	–	2.6	1.88	±	0.47		
Apical anterior	Cases	0.31	–	2.65	1.55	±	0.58	5.455	0.022*
	Control	1.02	–	3.09	1.90	±	0.53		
Mid anterior	Cases	0.23	–	2.41	1.27	±	0.52	7.541	0.008*
	Control	0.89	–	2.68	1.64	±	0.50		
Basal anterior	Cases	0.26	–	2.27	1.45	±	0.48	0.842	0.362
	Control	0.93	–	3.16	1.56	±	0.49		
Global LV	Cases	0.61	–	2.49	1.45	±	0.31	15.822	0.001*
	Control	1.26	–	2.26	1.78	±	0.33		

Table 7 A,B Comparison between both groups as regard early diastolic strain rate of the selected segments shows significant reductions in global strain rate at the level of basal septal, septal ,basal lateral ,apical anterior and mid anterior myocardial segments in the diabetic group in comparison with the control.

Early diastolic Strain rate		Range			Mean	±	S. D	t. test	p. value
Basal Septal	Cases	0.3	–	3.62	1.08	±	0.61	11.731	0.001*
	Control	0.67	–	2.73	1.66	±	0.69		
Mid Septal	Cases	0.19	–	3.09	1.17	±	0.51	3.057	0.085
	Control	0.85	–	2.04	1.39	±	0.38		
Apical Septal	Cases	0.06	–	2.53	1.43	±	0.52	3.677	0.050*
	Control	0.82	–	2.66	1.70	±	0.51		
Apical lateral	Cases	0.26	–	2.85	1.45	±	0.63	0.129	0.721
	Control	0.7	–	1.95	1.50	±	0.37		
Mid lateral	Cases	0.32	–	2.26	1.27	±	0.43	0.008	0.929
	Control	0.87	–	1.76	1.27	±	0.28		
Basal lateral	Cases	0.09	–	2.52	1.29	±	0.49	3.891	0.050*
	Control	0.89	–	2.91	1.58	±	0.68		
Basal posterior	Cases	0.49	–	3.16	1.53	±	0.70	2.999	0.088
	Control	0.81	–	6.59	1.94	±	1.24		
Mid posterior	Cases	0.15	–	3.39	1.27	±	0.56	2.772	0.101
	Control	0.19	–	1.84	1.03	±	0.42		
Apical posterior	Cases	0.49	–	3.90	1.48	±	0.73	0.004	0.952
	Control	0.51	–	2.67	1.49	±	0.65		
Apical anteroseptal	Cases	0.3	–	3.30	1.39	±	0.68	0.77	0.383
	Control	0.6	–	2.83	1.55	±	0.69		

Table 7B

Strain rate		Range			Mean	±	S. D	t. test	p. value
Mid anteroseptal	Cases	0.08	–	2.09	1.22	±	0.45	0.157	0.693
	Control	0.35	–	1.95	1.27	±	0.46		
Basal anteroseptal	Cases	0.01	–	2.49	1.31	±	0.49	0.874	0.353
	Control	0.82	–	2.12	1.43	±	0.42		
Basal inferior	Cases	0.57	–	2.09	1.28	±	0.42	1.773	0.187
	Control	0.89	–	1.89	1.42	±	0.29		
Mid inferior	Cases	0.24	–	2.91	1.40	±	0.41	1.499	0.225
	Control	1.11	–	2.12	1.52	±	0.24		
Apical inferior	Cases	0.09	–	2.38	1.57	±	0.44	3.363	0.071
	Control	0.90	–	2.51	1.79	±	0.47		
Apical anterior	Cases	0.09	–	2.51	1.63	±	0.46	4.209	0.044*
	Control	0.21	–	2.55	1.53	±	0.49		
Mid anterior	Cases	0.92	–	2.99	1.80	±	0.53	7.541	0.008*
	Control	0.15	–	2.33	1.19	±	0.52		
Basal anterior	Cases	0.81	–	2.60	1.56	±	0.50	0.842	0.362
	Control	0.19	–	2.20	1.38	±	0.48		
Global LV	Cases	0.86	–	3.09	1.49	±	0.49	15.822	0.001*
	Control	0.19	–	3.09	1.41	±	0.48		

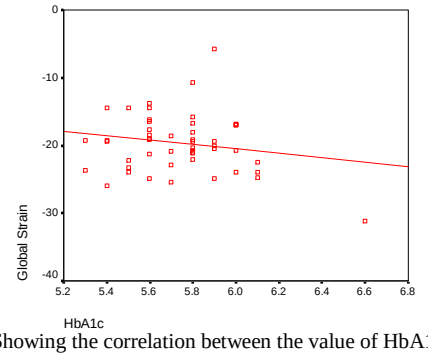

Figure 1A Showing the correlation between the value of HbA1C and the global systolic strain in the group of type 1 diabetes mellitus

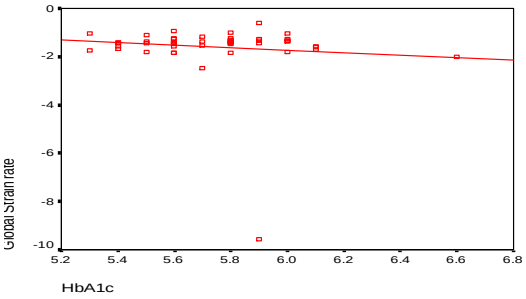

Figure 1B Showing the correlation between the value of HbA1C and the global systolic strain in the group of type 1 diabetes mellitus

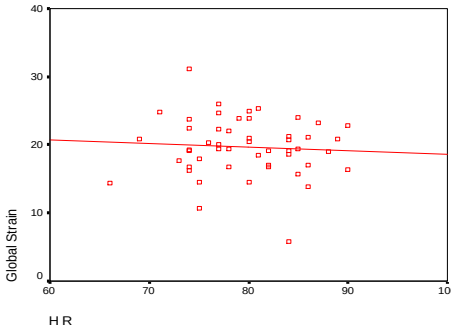

Figure 2A Showing the correlation between the value of HR and the global strain in the group of type 1 diabetes mellitus

Correlations

Regarding HbA1C

Correlation between the value of HbA1C and the global systolic strain in the group of type1 diabetes mellitus, the study found that there is no significant negative correlation between the value of HbA1C and the global systolic strain (r value=-0.187 ,p value=0.194) as shows in table (3). Similarly Correlation between the value of HbA1C and the global systolic strain rate, the study found that there is no significant negative correlation between the value of HbA1C and the global systolic strain rate (r value=-0.106, p value= 0.462)fig(1)

Regarding the heart rate

Correlation between the heart rate, the global systolic strain and global strain rate in both groups of type1 diabetes mellitus and control, the study found that there is no significant negative correlation between the heart rate and the global systolic strain and strain rate.fig(2)

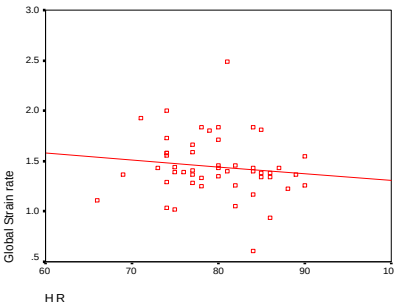

Figure 2B Showing the correlation between the value of HR and the global strain rate in the group of type 1 diabetes mellitus

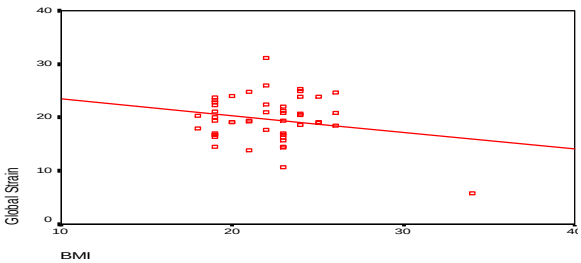

Figure 3 A Showing the correlation between the value of BMI and the studied parameters in the group of type 1 diabetes mellitus.

Regarding BMI

Correlation between the value of BMI , the global systolic strain and systolic strain rate in the group of type 1 diabetes

mellitus, the study found that there is no significant negative correlation between the value of BMI , the global systolic strain and strain rate as show in fig(3)

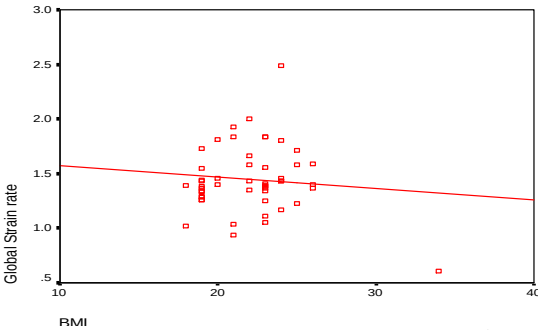

Figure 3B Showing the correlation between the value of BMI and the studied parameters in the group of type 1 diabetes mellitus.

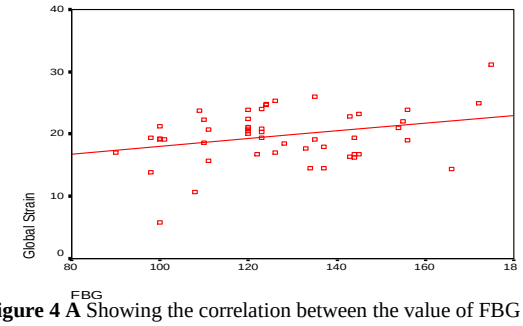

Figure 4 A Showing the correlation between the value of FBG and the global strain in the group of type 1 diabetes mellitus.

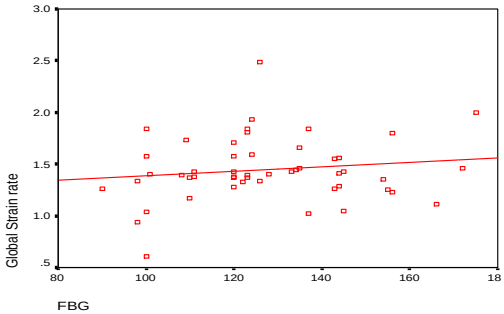

Figure 4 B Showing the correlation between the value of FBG and the global strain rate in the group of type 1 diabetes mellitus

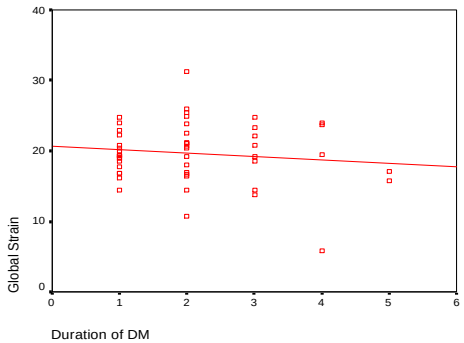

Figure5A Showing the correlation between the value of duration of DM and the global strain in the group of type 1 diabetes mellitus

Regarding FBG

Correlation between the value of FBG and the global systolic strain in the group of type1 diabetes mellitus, the study found that there is highly significant negative correlation between the value of FBG and global systolic strain and global strain rate as show in fig(4)

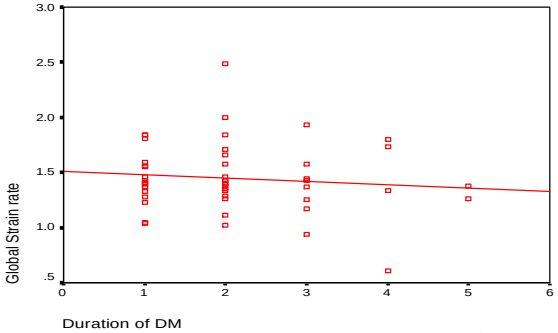

Figure 5B Showing the correlation between the value of duration of DM and the global strain rate in the group of type 1 diabetes mellitus

DISCUSSION

By the year 2030,there will be 8.6 million adults with diabetes in Egypt ,making it the country with the tenth largest population of diabetics in the world[26].

Strain rate(SR) imaging is a new method for detection of segmental myocardial contraction or stretching.(15) T1DM considered a disease of childhood and adolescentwith two peaks of onset, one between ages 5 and 9 years and a secondbetween ages 10 and 14 years[27]**.** Rising hemoglobin A1c levels and increased insulin resistance have also been observed prior to T1DM onset(29,30)

Stage 1: Genetic predisposition
Stage 2: Triggering of autoimmunity.
Stage 3: Development of autoantibodies and autoreactive T-cell clones.
Stage 4: Loss of β-cell function as manifested by abnormal responses in theintravenous glucose tolerance test progressing to abnormal glucose tolerance.
Stage 5: Overt diabetes (31)

Criteria for the diagnosis of diabetes according to the last report from international Expert Committee on Diagnosis and classification of diabetes mellitus on 2009 &confirmed by American Association on 2011:

1) Glycated hemoglobin (A1C) ≥6.5 % or
2) Fasting Plasma Glucose (FPG) ≥126 mg /dl (7.0 mmol/l). or
3) 2-h plasma glucose ≥200 mg/dl (11.1 mmol/l) or
4) In a patient with classic symptoms of hyperglycemia or hyperglycemic crisis ,a random plasma glucose≥200 mg/dl (11.1 mmol/l) (32)

The ability to predict the development of T1DM has been improved markedly with the combined use of genetic, islet autoantibody and metabolic testing (33) American Diabetes Association Guidelines for the target glucose and HbA1C level by age:

1) Preschooler 0-6years: target blood glucose before meals (100-180) and at bed time (110-200), HbA1C (7.5-8.5).

2) School age 6-12years ;(90-180) before meals and (100-180) at bedtime and HbA1C less than 8.
3) Adolescents and young adults 13-19year;(90-130)before meals and (90-150)at bedtime and HbA1C less than 7.5 (34)

Complications of poorly managed type 1 diabetes mellitus may include cardiovascular disease, diabetic neuropathy, and diabetic retinopathy, among others. However, cardiovascular disease (35) as well as neuropathy (36)

Diabetic cardiomyopathy was first advocated by Rubler *et al.* at 1972 based on postmortem finding of heart failure in diabetic patients without hypertension, coronary artery disease, valvular heart disease or other cardiovascular risk factors (37)

Four main causes are responsible for the development of heart failure in DCM:

1) microangiopathy and related endothelial dysfunction.
2) autonomic neuropathy.
3) metabolic alterations that include abnormal glucose use and increased fatty acid oxidation.
4) generation and accumulation of free radicals, and alterations in ion homeostasis, especially calcium transients. (38,39,40)

Strain is dimensionless parameter representing deformation of an object, relative to its original shape.

Strain is expressed as the percent (fractional) change from the original dimension S= L-L0/ L0

Strain Rate

Strain rate (SR) measures the time course of deformation, SR is the local rate of deformation or strain per unit time. Strain rate (SR) is a primary parameter of deformation derived from tissue Doppler, Indeed, SR seems to be a correlate of rate of change in pressure (dP/dt), a parameter that is used to reflect contractility, whereas strain is an analog of regional ejection fraction (41)

- The present study was aiming to identify the use of the strain and strain rate speckle tracking as a new technique for early evaluation of the systolic and diastolic function in patient with type 1 diabetes mellitus.
- The present study included 50 patients with type 1 diabetes mellitus an 20 subjects served as a control. Examination of the left ventricular systolic and diastolic function in the two studied groups has been based on parameters obtained from Trans - Thoracic Echocardiography.
- In the present study by using demographic data and by doing comparison to detect the significant difference between the studied groups as regard age ,sex, body mass index, heart rate and duration of T1DM was statically no significant difference between the group of type 1diabetes mellitus and the control.
- Conventional 2D Ttrans-thoracic echocardiography parameters shows no significant difference in any echocardiographic parameters between both groups, as regard interventricular septal thickness in diastole, left ventricular end systolic dimension, left ventricular end diastolic dimension, left ventricular posterior wall thickness in diastole , ejection fraction, fractional shortening, E wave, A wave, E/A ratio and but there is highly significant difference between the two groups as regard E wave was significantly higher in the diabetic group which is agree with Kapukn *et al.*,1993 and W.Kosmala *et al* (42) showed non-significant difference in LV geometry between diabetics and controls. Kadda *et al.*,2005 (43) and Kamile *et al.*,2009(44) who showed that interventricular septum & posterior wall thickness inend diastole were significantly higher in children & adolescent with Type 1diabetes than in controls. Also Suys *et al.*, 2004(45) reported that left ventricularwall dimensions were higher in diabetic population and reached statisticalsignificance for the left ventricular posterior wall. Devereux *et al.*,2000(46)reported that mean LV interventricular septaland posterior wall thickness were greater in diabetic patients compared tocontrols with no difference in LV chamber size.
- In the present study we have not found any significant correlations between the duration of diabetes and the severity of LV dysfunction, which is agree with W.Kosmala *et al* (42) and in contrast to the findings of Karamitos *et al.*(47) Eun &Yeo,2010(48) did not show significant positive correlation between duration of DM and echocardiographic parameters of LV systolic and diastolic function also Suys *et al.*, 2004(45) and Ismail *et al.*, agree with this. In contrary to our study , El-Shahed *et al.*,2008(49) found negative correlation between duration of DM and regional LV diastolic function .
- In the present study we have not found any significant correlations between the HbA1c and the severity of LV dysfunction, which is agree with W.Kosmala *et al*(42) &Lo *et al.*, (50)Ismail *et al.*, (51),.Suys *et al*,. (45), El-Shahed *et al*, (49), Kamile *et al*,.2009(44) and Hiromi Nakai *et al.*, 2009 and in contrast to the findings of Vinereanu *et al.* and Fang ZY A *et al*(6)
- No significant correlations were demonstrated in both groups between LV strain and strain rate parameters and indices of diabetic control (HbA1c) as well as duration of diabetes as in W.Kosmala *et al.*(42)
- In the present study by doing comparison between groups as regard fasting blood glucose there was statistically highly significant difference between the group of type 1 diabetes mellitus and the control as regard to fasting blood glucose.

Prevalence is seen particularly in heart failure patients with normal LVEF. However, LVEF is a relatively insensitive measure of LV systolic function compared to strain and strain rate imaging, especially in the context of subclinical LV systolic dysfunction. (53) As the LV myocardial architecture is a complex array of longitudinally and circumferentially orientated fibers located predominately in the epicedium / endocardium and mid-wall respectively, (54) multidirectional analyses of longitudinal, circumferential and radial function allow understanding of regional LV myocardial functional changes in subclinical diabetic heart disease.

- Strain and Strain rate parameters were measured by speckle tracking analysis through an off line technique application of strain and strain rate parameters in the programmed machine which produced LV systolic strain ,peak systolic strain rate and early diastolic strain rate were measured and compared between the two groups.
- LV global systolic strain was impaired in the diabetic group compared with the control group. comparing both groups as regard global and segmental systolic strain measured by speckle tracking parameters, it was found that, there is a statistically highly significant difference between the group of type 1 diabetes mellitus and the control with impairment of the longitudinal LV systolic strain at the level of the global and basal septal, mid lateral, mid inferior, apical inferior, apical anterior and mid anterior myocardial segments in the diabetic group in comparison with the control. And this agree with Arnold *et al,* *(52)*
- Longitudinal peak systolic strain rate was impaired at the level of the global and myocardial segments at the level of basal septal, apical septal ,basal lateral ,apical anterior and mid anterior myocardial segments the diabetic group in comparison with the control. And this agree with Arnold *et al,* *(52)*
- Early diastolic strain rate was impaired at the level of the global and myocardial segments at the level of basal septal, apical septal ,basal lateral ,apical anterior and mid anterior myocardial segments the diabetic group in comparison with the control. And this agree with Arnold *et al,* (52)
- The result of the present study have demonstrated the presence of subclinical myocardial systolic and diastolic dysfunction in patient with type 1 diabetes mellitus ,with no diabetic related complication, and were asymptomatic. Despite a normal LVEF and LV diastolic measures with 2 D Echocardiography. The diabetic patients showed impairment of LV longitudinal strain and strain rate. The presence of diabetes mellitus was an independent predictor for impairment of LV longitudinal strain and strain rate.This agree with the study of Arnold *et al,* (52) that demonstrated Findings from Left Ventricular Strain and Strain Rate Imaging in Asymptomatic Patient with Diabetes Mellitus. Where male patients (age 57±6 years) with asymptomatic type 1 diabetes mellitus were compared to those from 53 male controls. No differences were found in the LV end- diastolic volume index, end-systolic volume index, ejection fraction and fractional shorting Importantly, the diabetic patients have impaired LV peak systolic strain and peak early diastolic strain rate.

References

1. ADA "American Diabetes Association "(2011) Diagnosis and Classification of Diabetes Mellitus .Diabetic Care,volume 34,supplement 1:62-69.
2. ADA "American Diabetes Association "(2011) Diagnosis and Classification of Diabetes Mellitus .Diabetic Care, volume 34, supplement 1:62-69.
3. Amador-patarroya M.J.,Roariquez-roariquez A. and Montoya-ouizG. (2012):How Does Age at Onset Influence the Outcome of Autoimmune Diseases? Autoimmune Diseases; 2012: 251730.
4. Arafa NA and Amin GE.the Epidemiology of Diabetes Mellitus in Egypt ;results of a National Survey.*THE egyptian JOURNAL OF community Medicine* ,2010;28(3):29-43
5. *Arnold CT Ng, Delgado V, BertiniM, et al.* Findings from left ventricular strain and strain rate imaging in asymptomatic patients with type 2diabetes mellitus :*AmJ Cardiol.2009Nov 15;104:1398-401*
6. Bingley PJ, Mahon JL & Gale EA. (2008):European NicotinamideDiabetes Intervention Trial Group. Insulin resistance and progressionto type 1 diabetes in the European Nicotinamide Diabetes InterventionTrial (ENDIT).Diabetes Care;31(1):146–150
7. Boulton A.J., Vinik A.I., Arezzo J.C. Diabetic neuropathies: a statement by the American Diabetes Association. Diabetes Care. 2005;28:956–962.
8. Chung J., Abraszewski P., Yu X. Paradoxical increase in ventricular torsion and systolic torsion rate in type I diabetic patients under tight glycemic control. *J Am CollCardiol*. 2006;47:384–390
9. Cosson S.& Kevorkian JP.(2003) : Left ventricular diastolic dysfunction :an early sign of diabetic cardiomyopathy ?Diabetes metab;29(5):455-466.
10. *Devereux Richard B., Roman Mary J., Paranicas Mary, O'Grady Michael J., Lee Elisa T., Welty Thomas K., et al (2000):* Impact of Diabetes on Cardiac Structure and Function. The Strong Heart Study. Circulation;101:2271-2276.
11. Dutka DP, Donnelly JE, Palka P, *et al*. Echocardiographic characterization of cardiomyopathy in Friedrich's ataxia with tissue Doppler echocardiographically derived myocardial velocity gradients. Circulation. 2000;102:1276–1282.
12. *Elshahed Ghada S, Ahmed Mohamed I, El-Beblawy Nagham S,Kamal Hanan M, lsmaiel Magdy F & Bin Zheidan Ousama AS. (2008)*: Evaluation of Right and Left Ventricular Systolic and Diastolic Function in Patients with Type I Diabetes Using Echocardiography and Tissue Doppler Imaging . *Suez Canal Univ Med J* Vol. 11, No. 1 :65 -74 .
13. *Eun H. Kim & Yeo H. Kim (2010):* Left ventricular function in children & adolescents with type 1 diabetes mellitus. Korean Circ J;40:125-130
14. Fang ZY, Yuda S, Anderson V, Short L, Case C, Marwick TH. Echocardiographic detection of early diabetic myocardial disease. *J Am CollCardiol* 2003;41:611–7
15. Francis GS. Diabetic cardiomyopathy: fact or fiction? Heart. 2001;85:247–248.
16. *GalderisiM,deSimoneG,InnelliP et al* .Impaired inotropic response in type 2 diabetes mellitus: a strain rate imaging study. *Am J Hypertens* 2007; 20: 548-555.
17. *Garcio Fernandez MA, AzevedoJ, et al.* Segmental Isovolumic relaxation time of left ventricle myocardium by pulsed Doppler tissue imaging, Anew index of regional diastolic function and normal pattern description: *eurHeart J*1996;17: 3050-3062.
18. Golfman LS, Takeda N, Dhalla NS. Cardiac membrane

Ca(2+)-transport in alloxan-induced diabetes in rats. Diabetes Res ClinPract. 1996; 31(Suppl):S73–S77.
19. Granberg V, Ejskjaer N, Peakman M, Sundkvist G (2005). "Autoantibodies to autonomic nerves associated with cardiac and peripheral autonomic neuropathy". Diabetes Care 28 (8): 1959–64.
20. *Greenbaum RA, Ho SY, Gibson DG, et al.* Left ventricular fiber architecture in man. *BrHeartJ* 1981;45: 248-263.
21. *Ismail M. Hebatullah, Salah El-Din M. Nermine, Abdel Ghafour M. Mona & Mosaad A. Naglaa (2003):* Subclinical dibetic cardiomyopathy in children & adolescents with type 1 diabetes. M.Sc. thesis. Ped Department. Cairo university..
22. Janet Silverstein,Georgeanna Klingensmith,Kenneth Copeland Leslie Plotnick,*et al* (2008)Care of Children and adolescents With Type 1Diabetes.A statement of the American Diabetes Association. Diabetes Care .VOL31 (suppl 1)12-54.
23. Jennifer M. Barker & George S. Eisenbarth (2004):The NaturalHistory of Autoimmunity in Type 1A Diabetes Mellitus. Diabetes
24. *Kaada Ahmed, Abdel-sattar Inas and Magd El-Din Hisham (2005)*: Impact of type 1 diabetes mellitus on left ventricular structure and function.JAC., Vol.16, No.6: 667-673.
25. *Kamile Gul, Aksuyek Savas Celebi, Fehmi Kacmaz, Ozlem Celebi Ozcan, Ihsan Ustun, Dilek Berker, et. al., (2009):* Tissue Doppler imaging must be performed to detect early left ventricular dysfunction in patients with type 1 diabetes mellitus. *European Journal of Echocardiography*; 10: 841–846
26. *Karamitsos D. Theodoros, Tsapas Apostolos & Jayanth R. Arnold (2008):* Diabetic cardiomyopathy:a controversial entity. Letters to the editor. *European Heart Journal*; 29: 564–568
27. Karvounis HI, Papadopoulos CE, Zaglavara TA, *et al*. (2004). "Evidence of left ventricular dysfunction in asymptomatic elderly patients with non-insulin-dependent diabetes mellitus". Angiology 55 (5): 549–55.
28. Koyama J, Ray-Sequin PA, Davidoff R, Falk RH. Usefulness of pulsed tissue Doppler imaging for evaluating systolic and diastolic left ventricular function in patients with AL (primary) amyloidosis.*Am J Cardiol*. 2002;89:1067–1071.
29. Li Zhang & George S.Eisenbarth (2011)prediction and prevention of Type 1 diabetes mellitus .*Journal of Diabetes* .Volume 3,Issue 1,pages 48-57
30. *Lo SS, Leslie RD & Sutton MS (1995):* Effects of type 1 diabetes mellitus on cardiac function: a study of monozygotic twins.*Br Heart J*.73:450-455.
31. *MaronBJ, McKennaWJ, DanielsonGK, KappenbergerLJ, Kuhn HJ, SeidmanCE, et al*. ACC/ESC Clinical Expert Consensus Documenton Hypertrophic Cardiomyopathy. A repor tof the ACC Foundation. Task Force on Clinical Expert Consensus Documents and ESCCommitteeforPracticeGuidelines.*EurHeartJ*2003;24: 1965–91.
32. Marwick T.H. Diabetic heart disease.Heart. 2006;92:296–300
33. McCance DR, Hanson RL, Charles MA, Jacobson LTH, Pettitt DJ, Bennett PH *et al*. Comparison of tests for glycated haemoglobin and fasting and two hour plasma glucose concentrations as diagnostic methods for diabetes.BMJ 1994; 308: 1323-28.
34. Nunoda S, Genda A, Sugihara N, *et al*. Quantitative approach to the histopathology of the biopsied right ventricular myocardium in patients with diabetes mellitus. Heart Vessels. 1985;1: 43–47.
35. O'Brien I.A., McFadden J.P., Corrall R.J. The influence of autonomic neuropathy on mortality in insulin-dependent diabetes. *Q J Med*. 1991;79:495–502
36. Rubler S, Dlugash J, Yuceoglu YZ, Kumral T, Branwood AW, Grishman A (November 1972). "New type of cardiomyopathy associated with diabetic glomerulosclerosis". *Am. J. Cardiol*. 30 (6): 595–602.
37. Ruddy TD, Shumak SL, Liu PP, *et al*. (1988). "The relationship of cardiac diastolic dysfunction to concurrent hormonal and metabolic status in types I diabetes mellitus". *J. Clin. Endocrinol. Metab*. 66 (1): 113–8.
38. Sampson M.J., Wilson S., Karagiannis P. Progression of diabetic autonomic neuropathy over a decade in insulin-dependent diabetics. *Q J Med*. 1990; 75: 635–646.
39. Severson DL (October 2004). "Diabetic cardiomyopathy: recent evidence from mouse models of type 1 and type 2 diabetes". *Can. J. Physiol. Pharmacol*. 82 (10): 813–23.
40. *SimpsonIA.* Echocardiographic assessment of long axis function: a Simplesolutionto a complexproblem?Heart 1997;78:211-212.
41. Songer, TJ.(2002): Low blood sugar and motor vehicle crashes in persons with type 1 diabetes, AnnuProcAssocAdv Automotive Med, 46:424–427
42. Stene LC, Barriga K, Hoffman M, *et al*. (2006):Normal butincreasing hemoglobin A1c levels predict progression from isletautoimmunity to overt type 1 diabetes: Diabetes Autoimmunity Studyin the Young (DAISY). Pediatr Diabetes;7(5):247–253.
43. Stone P.H., Muller J.E., Hartwell T. The effect of diabetes mellitus on prognosis and serial left ventricular function after acute myocardial infarction: contribution of both coronary disease and diastolic left ventricular dysfunction to the adverse prognosis. The MILIS Study Group. *J Am CollCardiol*. 1989;14:49–57
44. *Suys E.Bert, Katier Nienke, Rooman P.A. Raoul, Matthys Dirk, Lieve OP DE Beeck, Mark V.L.,et al. (2004):* Female Children and Adolescents With Type 1 Diabetes Have More Pronounced Early Echocardiographic Signs of Diabetic Cardiomyopathy. Diabetes Care, 27; (8):1947-1953.
45. *Teske AJ, De Boeck BW, Melman PG, et al.* Echocardiographic quantification of myocardial function using tissue deformation imaging, aguidetoimageacquisition and analysisusingtissue Doppler and speckle tracking. Cardiovasc Ultrasound2007; 5 : 27.
46. Torffvit O., Lovestam-Adrian M., Agardh E. Nephropathy, but not retinopathy, is associated with the development of heart disease in type 1 diabetes: a 12-year observation study of 462 patients. Diabet Med. 2005;22:723–729
47. *Trivelli, L.A., et al.* Glycated hemoglobin. New Eng. J. Med. (1971); 284:353, 1971.
48. Vijan, S (March 2010)."Type 2 diabetes". Annals 152 (5):

ITC31-15.
49. W.Kosmala,M.Przewlocka-Kosmala and W.Mazurek 2006
50. *Wallach,J.B*; Interpretation of diagnostictests8thed,Brooklyn, New Yourk. Pp. (2007) 802-806.
51. *Weidemann F,Eyskens B, MertensL,et al* .Quantification of regional right and left ventricular function by ultrasonic strain rate and strain indexes after surgical repair of tetralogy of Fallot *Am J Cardiol* 2002;90:133-138.
52. William H Lamb and Stephen Kemp.(2012):Pediatric Type 1Diabetes Mellitus.,WWW.Medscape.com.Updated on Jun 20, 2012
53. World Health Organization Department of Non communicable Disease Surveillance (1999)."Definition, Diagnosis and Classification of Diabetes Mellitus and its Complications"
54. *Zilemr, GaaschWH, et al.* Heart failure with a normal ejection fraction: is measurement of diastolic function necessary to make the diagnosis of heart failure? Circulation 2001; 104: 779-82.

How to cite this article:

Walaa F. Abdaziz *et al*., Detection Of Early Left Ventricular Dysfunction In Type 1 Diabetes Mellitus By Strain And Strain Rate Imaging. *International Journal of Recent Scientific Research Vol. 6, Issue, 4, pp.3690-3701, April, 2015*

****