

## Echocardiographic assessment of the effect of type (2) Diabetes mellitus on cardiac performance

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### Summary:

**Background:** Diabetes mellitus (DM) causes damaging effects on the cardiac function; these effects can be observed on the diastolic performance of the heart reflected on the change in transmitral blood velocity, the cardiac wall and septum thickness.

**Objectives:** The present study was to assess the diastolic and systolic cardiac muscle performance for patients with type 2 diabetes mellitus compared with control individuals and to evaluate the index of myocardial performance.

**Patients and Methods:** The study involved 97 patients (35 male and 62 female of average age of 56.2 ±10.755) of type 2 diabetes mellitus (DM), they were investigated for their left ventricle performance and compared with 51 normal individuals "the control group" (20 male and 31 female of average age of 41.4 ± 13.196).

Measurements of isovolumetric contraction time IVCT, ejection time ET, ejection fraction EF%, isovolumetric relaxation time IVRT, the early and late peak velocities E and A of transmitral flow, left ventricle diameter in diastole and systole LVIDs, LVIDs, posterior wall thickness PWTd, and Interventricular septum thickness in diastole IVSTd were measured, and index of myocardial performance IMP was calculated.

**Results:** Results reveal differences in these parameters for patients group relative to controls, in IVRT, ET, E, A, E/A, EF%, IMP, LVIDs, PWTd and IVSTd all are strongly significant with p value <0.001 and for FS% p value = 0.0029 except for IVCT the change was 9.342% with p value 0.188 and the change in LVIDd -3.586%, p value 0.052 were not significant.

**Conclusion:** Diabetes mellitus can cause a deleterious effect on the myocardium. The effect causes impairment in the cardiac diastolic performance and muscle contractility caused by the damage inflicted by hyperglycemia (high blood sugar). Also results show that IMP is increased in type 2 DM patients. This increase may be an early sign of diabetic cardiomyopathy in diabetic patients.

**Key words:** Diabetes mellitus, Echocardiography, Index of myocardial performance (IMP).

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### Introduction:

Diabetes is a group of diseases characterized by elevated blood glucose concentration. It may be a consequence of either the body does not produce enough insulin or because cells do not respond to the insulin that is produced, it can be classified into three major classes: Type 1, Type 2 and Gestational diabetes mellitus. (1) Type 1 diabetes known as insulin-dependent diabetes) IDDM, (childhood diabetes or also known as juvenile diabetes, is characterized by loss of the insulin producing beta cells of the islets of Langerhans of the pancreas leading to a severe deficiency of insulin. (2) Type 2 diabetes mellitus known as adult-onset diabetes or non-insulin dependent diabetes mellitus) NIDDM (is characterized by insulin resistance which may be combined with relatively reduced insulin secretion. The defective responsiveness of body tissues to insulin is believed to involve the insulin receptor (3).

Gestational diabetes mellitus resembles type 2 diabetes in several aspects, involving a combination of inadequate insulin secretion and responsiveness. It occurs in about 2% to 5% of all pregnancies and may improve or disappear after delivery (4).

When the glucose increases in the blood it can cause serious complications including: Cardiomyopathy, nephropathy, neuropathy, and retinopathy (5).

Diabetes is a major risk factor for coronary artery disease and cardiovascular disease (6) is the most important cause of morbidity and mortality in patients with type 2 diabetes, accounting for approximately two-thirds of total mortality (7).

Diastolic dysfunction has been described as an early sign of diabetic heart muscle disease preceding systolic damage (8) it is associated with future occurrence of heart failure, is a predictor of cardiovascular morbidity and mortality in the general population (9).

The late effect of diabetes cardiomyopathy is characterized by LV hypertrophy and myocardial dilatation, which leads to LV

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diastolic and systolic dysfunction (10).

The aim of this study is to assess the cardiac performance for patients with diabetes mellitus compared with a control and the relation between diabetes mellitus with age on cardiac performance in comparison to the controls group.

**Patients and Methods:** This study was performed during 6 months in the echo unit of the cardiac care unit at Baghdad / medical city teaching hospital. A total of 148 subjects were included in the study, 51 control group (20 male and 31 female with mean age of  $41.4 \pm 13.196$ ) and 97 patients of type 2 DM (35 male and 62 female with mean age  $56.2 \pm 10.755$ ). The clinical characteristics are shown in (Table 1). The height, weight, blood pressure and heart rate were measured for both patients and normal subjects. The history of DM was recorded to help the cardiologist for more evaluations of echocardiography diagnosis. All control and patients were examined by use echocardiography instrument of type Sonoace X<sub>8</sub> with 2-5 MHz transducer used for cardiology. The measurements were taken using M-mode guided by two-dimensional echocardiography was performed from the standard left parasternal long axis view approximately at the mitral valve leaflet tips to measure left ventricular internal dimensions at end systole (LVIDs) and diastole (LVIDd), posterior wall thickness PWTd, and Interventricular septum thickness in diastole IVSTd. Two dimensional echocardiography provides excellent images of the cardiac anatomy and large vessels, but it depends on obtaining satisfactory windows from the body surface to the area of interest in the heart (11).

Pulsed wave Doppler echocardiography was used to assess left ventricular diastolic function from an apical four chambers view. The transducer was placed medially to the apex, directed backward, slightly medially and upward. Ideally, the sample volume is positioned on the tips of mitral valve leaflets.

Measurements of the early and late peak velocities E and A of transmitral flow, isovolumetric relaxation time (IVRT) is measured from the end of flow velocity pattern (aortic closing) to the beginning of transmitral flow, isovolumetric contraction time (IVCT) is measured from the end of diastolic flow to the beginning of out flow and LV ejection time (ET) is measured from the end of IVCT to the beginning of IVRT (the period of out flow) were measured. IMP is used to assessment left ventricle function and it is determined as  $(IVCT + IVRT)/ET$  figure 1.

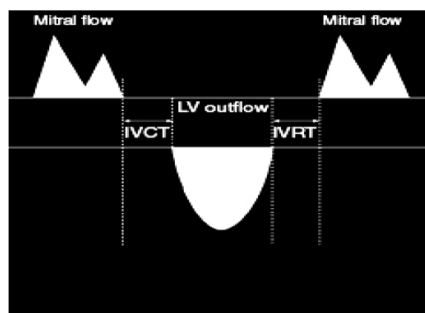


Figure 1: Measurement of Doppler interval from aortic valve (AV) opening to aortic valve (AV) closure.

$$IMP = \frac{(IVRT + IVCT)}{ET}$$

All values expressed as a mean value with  $\pm$  standard deviation, ( $p < 0.05$ ) is considered significant. Percentage change for every parameter for control and patients groups were calculated and a comparison between the mean values for each of the two groups was carried out by (unpaired student t-test).

#### Results:

Table 1 shows the characteristic of the population study  $\pm$  standard deviation for controls group and patients group. Results in table 2 reveal a difference in IVCT between patients and controls were (9.34%) giving insignificant p value of (0.188), while change percent in IVRT and ET are (-32.67%) and (30.9%) respectively and strongly significant p value  $< 0.001$  for both, consequently IMP value is (-67.28%) and it was also strongly significant  $p < 0.001$  (Table 2).

The change in EF% and FS% between patients and controls was (11.41% and 10.25% respectively) both are significant ( $p < 0.001$ ) and ( $p = 0.002$ ) respectively (Table 2). The transmitral early velocity (E) was increased by (19.612%), the late filling velocity (A) and the ratio (E/A) were decreased by (-21.898%), (34.15%) respectively, these results compared between patients and controls and found strongly significant with p value  $< 0.001$  (Table 1).

A comparison between patients and controls for both parameters PWTd and IVSTd has also given a strongly significant increase in both of them in patients (p value  $< 0.001$ ) table 1.

The change in LVIDd, between patients and controls is (-3.58%) and it is slightly insignificant ( $p = 0.052$ ) while the difference in LVIDs is (-9.216%) and it is strongly significant ( $p < 0.001$ ) and gave a change in the ratio of (LVIDd/LVIDs) between the two groups of (4.69%) is also strongly significant ( $p < 0.001$ ) (Table 2).

Results were plotted between the effect of diabetic patients and controls with age for all the measured parameters clear differences were observed between patients and controls, these differences appeared at early age and continue throughout the age and/or on the slope differences between both groups indicating a different rate for the cardiac muscle impairment figure 1.

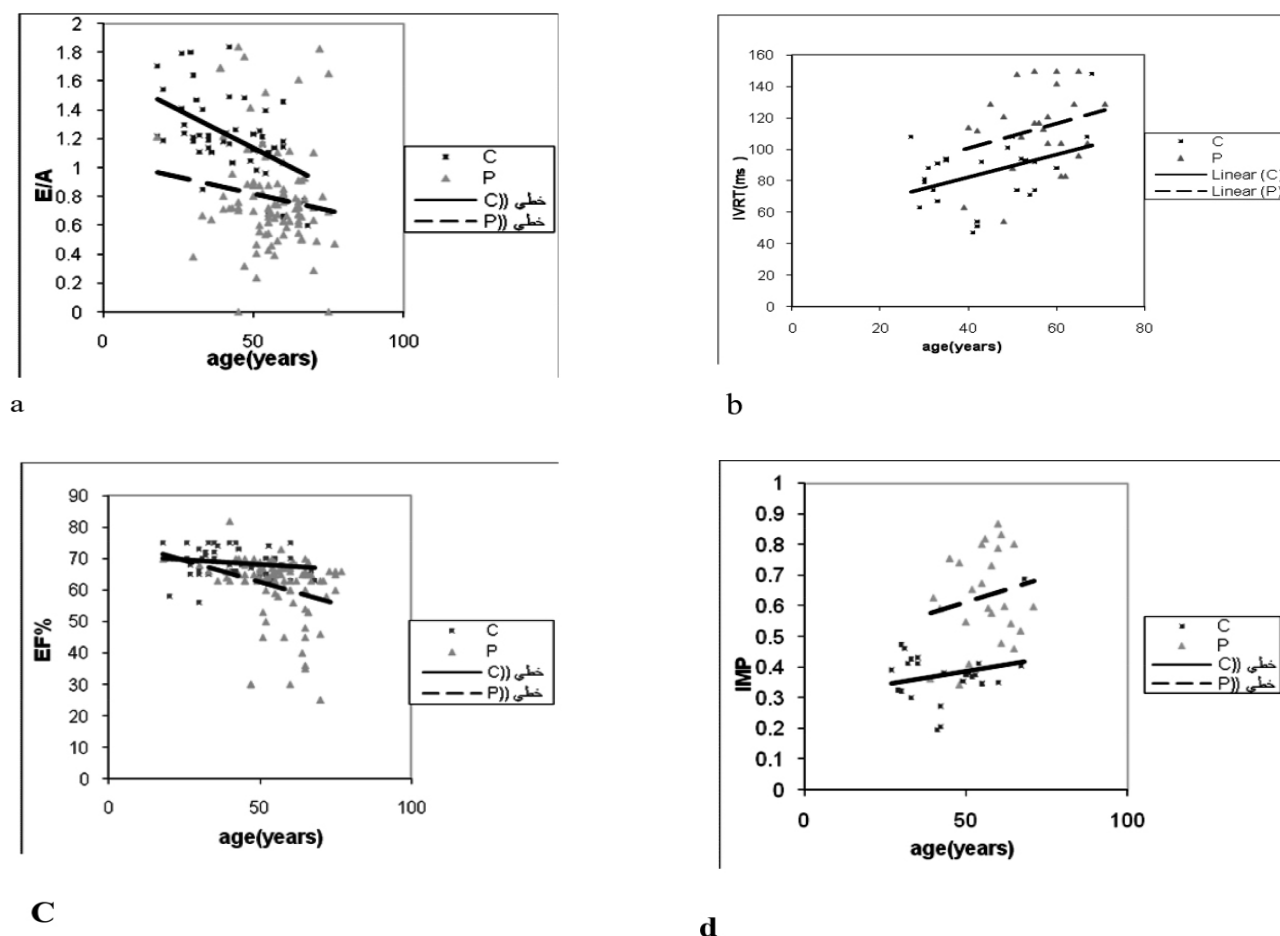


Figure 1: The change of cardiac parameters (a- E/A, b- IVRT, c- EF%, d- IMP) for controls (b- and patients with age.

The Slope of the graphs can be divided into two categories. Either diverges or converges such as in figure1(a,d), graphs

starting from the same point or intersect each other then diverge such as in figure1(c), or the two graphs are parallel there is either a small or slightly large gap between them such as figure1(b).

Table 1: M-mode and Doppler echocardiography data ± SD for controls group and DM groups.

Variable	Control Mean ± SD	DM Mean ± SD	Change% = (C-DM/C) × 100	p value
E (mm/s)	71.23± 13.051	57.26±23.185	19.612	<0.001*
A (mm/s)	60.46 ±15.605	73.7± 21.139	-21.898	<0.001*
E/A	1.224 ± 0.266	0.806± 0.344	34.15	<0.001*
LVIDd (mm)	50.667± 4.938	52.484± 6.086	-3.586	0.052
LVIDs (mm)	33.33± 3.834	36.402± 6.205	-9.216	<0.001*
LVIDd/LVIDs	1.532± 0.136	1.46± 0.169	4.699	0.005*
PWTd (mm)	10.352± 1.163	12.536± 1.620	-21.097	<0.001*
IVSTd (mm)	11.156± 0.784	13.68± 1.906	-22.624	<0.001*
EF%	68.71 ± 4.451	60.87 ±10.872	11.41	<0.001*
FS%	34.094 ±5.658	30.596 ±8.263	10.259	0.0029*
IVCT (ms)	60.8 ± 9.776	55.12± 18.802	9.342	0.188
IVRT (ms)	85.32 ±21.632	113.2± 25.872	-32.676	<0.001*
ET (ms)	398 ± 60.333	275 ± 64.951	30.904	<0.001*
IMP	0.376 ± 0.093	0.629 ± 0.150	-67.287	<0.001*

\* Significant <0.05

Discussion:

In the present study a comparison between control individuals and DM patients reveal that a significant statistical increase in isovolumetric relaxation time (IVRT) ( $P < 0.001$ ) also a significant decreases in peak E velocity and in the ratio E/A ( $p < 0.001$ ) and ( $p < 0.001$ ) respectively (Table 1). These parameters can show clearly the impairment of diastolic performance that involved impaired LV relaxation which lead to decreased early (E wave) and an increased filling with atrial contraction (A wave) as a compensatory action (12) in which the mitral inflow patterns show an E/A less than 1 indicating reduced cardiac performance, this is in agreement with the findings reported by (13). (Albanna et al 1998).

The difference between DM patients and control in the PWTd and IVSTd with p value ( $< 0.001$ ) (Table2) indicate the LV muscle at the beginning of lack in LV performance. Comparison of the echocardiographic parameters with age has shown a significant change in some of them and not for all parameters for example graphs for E/A reduced with age for patients and healthy people. This can be observed on figure 1 (a). We do not have a clear description for this but it is possible because of these parameters are well known to change with age and as we have seen earlier that DM influences these parameters also some patients with DM will have probably additive effect of both aging and DM. leading to a higher rate of loss of compliance leading to pseudo normalization and a less steeper graph of E/A with age fig 1 (a). The change is observed on the graph for comparison between IVRT and IMP for patients with DM and controls. The change in these parameters might be attributed to the impairment in the diastolic performance. Although results of patients for other parameters such as EF% gave rather small change from controls but are statistically significant. It is important to mention here that we have checked the change of the echocardiographic parameters with the disease duration and the results were not consistent and it may be because of the difference in the disease control. Figure 1 (a) show the slope of E/A for controls is steeper than that for DM patients but the values for DM are less than controls. This is because of that at the beginning of the graphs controls are much higher than DM patients i.e. at age of 20 so it gives higher values than DM patients even if the controls graph descending steeper than patients, it gives highly significant difference table (1). The less slope of DM graph may be explained on the bases of loss of compliance and pseudo normalization. The same effect can be observed on (IVRT, IMP) in figure 1 (b, d) respectively where we have a jump between the two groups of DM and controls but a very slight difference in the slope of the graph with age these parameters has given highly significant difference between DM and controls table 1.

**References:-**

1. Jameson J.L.: *Harrison's endocrinology*. New York. McGraw Hill Companies 2006. pp 283, 288.
2. Gardner D.G., Shoback D.: *Greenspan's basic and clinical endocrinology*. Eighth edition. New York. McGraw Hill

Companies 2007. pp 672, 673, 675.

3. Benedict C., Hallschmid M., Hatke A., Schultes B., Fehm HL., Born J., Kern W.: *Intranasal insulin improves memory in humans*. *Psychoneuroendocrinology*. PMID. Vol. 29 November 10 2004. pp 1326-34.
4. Lawrence JM., Contreras R., Chen W., Sacks DA.: *Trends in the prevalence of preexisting diabetes and gestational diabetes mellitus among a racially/ethnically diverse population of pregnant women, 1999-2005*. *Diabetes Care*. Vol. 31 May 5 2008. pp 899-904.
5. Cheung N., Wang JJ., Klein R., Couper DJ., Sharrett AR., Wong TY.: *Diabetic retinopathy and the risk of coronary heart disease The Atherosclerosis risk in communities study*. *The American Diabetes Association. Diabetes Care*. 2007; 30:1742-1746.
6. Somaratne JB., Whalley GA., Poppe KK., Ter Bals MM., Wadams G., Pearl A., Bagg W., Doughty RN.: *Screening for left ventricular hypertrophy in patients with type 2 diabetes mellitus in the community*. *Cardiovascular Diabetology of the Journal*. 2011; 10: 29.
7. Srivastava PM., Calafiore P., Macisaac RJ., Patel SK., Thomas MC., Jerums G., Burrell LM.: *Prevalence and predictors of cardiac hypertrophy and dysfunction in patients with Type 2 diabetes*. *The Authors Journal compilation*. 2008; 114: 313-320.
8. Raev DC.: *Which LV function is impaired earlier in the evolution of diabetic cardiomyopathy? An echocardiographic study of young type I diabetic patients*. *Diabetes Care*. 1994; 17: 633-9.
9. Ike SO., Ikeh VO.: *The prevalence of diastolic dysfunction in Adult Hypertensive Nigerian*. *Ghana Med. J*. 2006; 40(2): 55-60.
10. Galderisi M., Anderson KM., Wilson PW., Levy D.: *Echocardiographic evidence for the existence of a distinct diabetic cardiomyopathy (the Framingham Heart Study)*. *Am. J. Cardiol*. 1991; 68: 85-89.
11. Armstrong W.F., Ryan T.: *Feigenbaum's echocardiography*. Seventh edition. Philadelphia. Wolter kluwer; Lippincott Williams and Wilkins 2010. pp 9-13, 16, 17, 514-517.
12. Saglam H., Seyfeli E., Gul I., Duru M., Gokce C.: *Index of myocardial performance in patients with type 2 diabetes without hypertension and its relationship with clinical and echocardiographic parameters*. *Journal of Diabetes*. 2009; 1: 50-56.
13. Albanna II., Eichelberger SM., Houry PR., Witt SA., Standiford DA., Dolan LM., Daniels SR., Kimball TR.: *Diastolic dysfunction in young patients with insulin-dependent diabetes mellitus as determined by automated border detection*. *J. Am. Soc. Echocardiogr*. 1998; 11(4): 349-55.