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Correlation between Left Ventricular Electromechanical Delay and Body Mass Index

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ABSTRACT

Background: There have been reports of subclinical but substantial variations in the anatomy and function of the left ventricle (LV) in morbidly obese people who show no outward signs of cardiac disease. Between 11% and 14% of those with heart failure (HF) have a high body mass index (BMI).

Objective: The aim of the current study was to assess the effect of BMI on LV electromechanical delay in persons without cardiac illness. **Patients and methods:** A cross sectional study was conducted on 66 patients, recruited from Cardiology Department, Suez Canal University Hospital.

Results: A statistically significant difference was seen in our study regarding weight, BMI and body surface area (P values 0.001, 0.001 and 0.013, respectively) as obese group had the highest mean of weight (98.05±11.5). **Conclusion:** Left ventricular electromechanical changes in obese people with normal heart rhythm and no symptoms of heart trouble may have latent systolic dysfunction.

Keywords: Myocarditis, Obesity, Heart failure, Body mass index, Cross sectional study, Suez Canal University.

INTRODUCTION

A significant public health concern is cardiovascular disease, particularly in high-risk cases like obesity and diabetic populations. Obese individuals without overt heart disease have been shown to have subclinical alterations in the anatomy and function of left ventricle (LV) [(1)].

Excessive fat storage is the root of the obesity epidemic, making it a complicated health issue to handle. Obesity is more than simply a visual issue. If you are exceedingly obese, you probably have health issues that are related to your weight. It makes conditions and health issues like heart illness, type 2 diabetes, hypertension and early mortality more likely [(2)].

One of the greatest public health challenges of our time, and a major cause for concern on a global scale, is the alarming rise in childhood and adult obesity. The current epidemic of obesity currently affects about 60 million people in the United States alone.

Despite a dramatic rise in obesity incidence in the US beginning in the early 1980s (early 20th century), new estimates imply that the favorable shift in BMI distribution may have begun much earlier [(3)].

Adults in the Eastern Mediterranean region are most likely to be overweight or obese in United Arab Emirates, Saudi Arabia, Egypt, Bahrain and Kuwait. Overweight and obesity are pervasive in these countries, with female prevalence ranging from 74% to 86% and male prevalence from 69% to 77% [(4)].

Humans are often categorized as underweight, overweight, or obese according to their body mass index (BMI), a straightforward measure of weight-for-height. To get the person's BMI, we get their "kg/m2" by dividing their total body mass by their squared height in meters. A BMI of 25 or more is considered overweight by the World Health Organization, while a BMI of 30 or more is considered obese [(4)].

In diseased persons with heart failure with a prolonged QRS, resynchronization using biventricular pacing has been established to decrease hospital entrance for heart failure (HF) and death. This has led to an increase in interest in assessing LV dyssynchrony. Several distinct groups now believe that LV dyssynchrony predicts the future development of HF [(5)].

There are different types of dyssynchrony: Patients who have first degree AV block and dilated cardiomyopathy experience atrioventricular. The term "interventricular" refers to the discrepancy in the timing of right ventricular (RV) and LV contraction. Because of delayed electrical conduction, intraventricular contraction of the LV wall segments can occur either early or late [(6)]. Uncoordinated regional myocardial contractions are known as intra-ventricular dyssynchrony, and they may be caused by any of the following factors: The inconsistency in the timing of myocyte depolarization due to a lag in electrical conduction (primary electrical dyssynchrony), Disruptions in the normal pattern of excitation and contraction, also known as excitation-related dyssynchrony. Irregular strain or contractility in the heart (primary mechanical dyssynchrony) causes a lag in the shortening of fibers in a specific area. This suggests that mechanical dyssynchrony seen may have both electrical and non-electrical etiologies [(7)].

These results sparked the hunt for new and more practical measures of mechanical dyssynchrony. Recent advancements in tissue Doppler imaging (TDI) allow for the high temporal resolution valuation of electrical events in various places.

The ventricular electromechanical delay is the time it takes for the heart to contract to its maximum capacity, as measured by TDI, from the start of the QRS complex on the electrocardiogram (ECG) [(8)].

In recent years, narrow QRS complex has been shown to indicate intra-LV dyssynchrony in high-risk patients without symptoms of diabetes [(9)], hypertension

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[10], or renal insufficiency [11]. Most research on LV dyssynchrony has focused on diseased persons with dilated heart failure and low ejection fraction (HFrEf) and wide or narrow QRS complexes, respectively [10].

A common and pervasive health issue, HFpEF affects 30–55percent of all individuals with chronic HF [12]. Although the pathophysiological causes of HFpEF are many and intricate, anomalies of diastolic function, such as aberrant active relaxation and increased passive stiffness, are frequently blamed [13]. It indicates that myocardial hypertrophy and decreased diastolic but not systolic performance are related with LV mechanical dyssynchrony in HFpEF cases with no significant electrical dyssynchrony, suggesting that mechanical dyssynchrony may be involved in the aetiology of HFpEF [14]. Therefore, in the current study, we focus in on LV intra-ventricular dyssynchrony because it is well-established that this condition reduces pump function and exacerbates HF.

PATIENTS AND METHODS

A cross sectional study was conducted on 66 cardiac patients, recruited from Cardiology Department, Suez Canal University Hospital.

Inclusion criteria: There will be 3 groups of subjects (ages 18-60) of both sexes who have no history of cardiovascular disease based on their BMI: **Group A:** cases with normal BMI (18.5-24.9 kg/m^2**), Group B:** overweight cases with BMI 25-29.9 kg/m^2, and **Group C:** obese cases with BMI ≥30 kg/m^2. Groups were comparable in terms of age, gender, and potential for harm.

Exclusion criteria: Patients with QRS >120 msec, patients with LVEF <50%, patients with significant structural heart disease (infective endocarditis, rheumatic heart disease, artificial valves valve anomalies, congenital heart disease), clinical or ECG evidence of coronary artery disease, cases with HF and patients with any type of arrhythmia.
The estimated sample size was 66 patients.

Data collection procedure: All eligible patients were informed regarding the procedure and they were submitted to the following questionnaire.
Personal History: Age, gender and tobacco use, cardiovascular Risk Factors: history of hypertension, diabetes or dyslipidemia.
Physical Examination: General examination**,** weight and Height were measured and blood pressure were measured.

Laboratory Investigation: Venous blood sample were obtained by a highly trained nurse and analyzed by an experienced clinical pathology specialist after overnight fasting for assay of: Estimated Glomerular filtration rate (e-GFR), Glycated Hemoglobin (HbA1c) and lipid Profile (LDL, HDL, Triglyceride).

ECG: using 12 leads was done at rest.
Imaging: An expert cardiologist performed the transthoracic echocardiography on a Vivid 7 machine, adhering to the standard methodology established by the ASE. Standard views: Parasternal long axis (PLX), parasternal short axis (PSX), apical 4, three and two chambers, subcostal and suprasternal views were obtained.

Ethical Consideration: This study was ethically approved by the Institutional Review Board of the Faculty of Medicine, Suez Canal University. Written informed consent was obtained from all participants. This study was executed according to the code of ethics of the World Medical Association (Declaration of Helsinki) for studies on humans.

Statistical analysis

The collected data were introduced and statistically analyzed by utilizing the Statistical Package for Social Sciences (SPSS) version 20 for windows. Qualitative data were defined as numbers and percentages. Chi-Square test and Fisher's exact test were used for comparison between categorical variables as appropriate. Quantitative data were tested for normality by Kolmogorov-Smirnov test. Normal distribution of variables was described as mean and standard deviation (SD), and ANOVA test was used for comparison between groups. P value ≤0.05 was considered to be statistically significant.

RESULTS

Patients had female predominance than males, with mean age of 35.61 years. About 30.3% of patients were smokers (**Table 1**).

Table (1): General characteristics of the study participants

Characteristics	(N=66)
Age (Mean±SD)	35.61±11.28
Sex	
Male	22 (33.3%)
Female	44 (66.7%)
Smoking	20 (30.3%)
Hypertension	18 (27.3%)
Diabetes Mellitus	17 (25.8%)
Dyslipidemia	18 (27.3%)

Table 2 shows that mean age was higher among obese groups with statistically insignificant difference. Also, females were more predominant than males especially in overweight and obese groups with statistically insignificant difference. DM and smoking were higher among obese group, while hypertension had the highest prevalence among overweight group, with statistical insignificant differences between study groups.

Table (2): **Age, gender and clinical history differences among study groups**

Characteristics	Normal weight (Group I)	Overweight (Group II)	Obese (Group III)	*P*-value
Age (Mean±SD)	31.73±10.5	37.77±13	43.23±9.8	0.091[1]
Sex Male Female	 10 (45.5%) 12 (54.5%)	 6 (17.3%) 16 (72.7%)	 6 (17.3%) 16 (27.7%)	 0.336[2]
Smoking	9 (30%)	3 (13.6%)	8 (36.4%)	0.0892
Hypertension	5 (22.7%)	8 (36.4%)	5 (22.7%)	0.503[2]
Diabetes Mellitus	6 (27.3%)	3 (13.6%)	8 (36.4%)	0.2222
Dyslipidemia	3 (13.6%)	4 (18.2%)	11 (50%)	**0.013*[2]**

1: ANOVA-test, 2: Chi square.

Table 3 shows that there was a statistically significant difference among groups of the study regarding Standard Echo parameters, except for relative wall motion.

Table (3): Standard Echo parameters among study groups

Measurements	Normal weight (Group I)	Overweight (Group II)	Obese (Group III)	*P*-value
Septal thickness (mm)	8.4±1.4	8.7±1.4	10±1.5	**0.001*[1]**
Posterior wall thickness (mm)	8.4±1.2	8.7±1.5	10±1.2	**<0.0001*[1]**
LVDD (mm) Left ventricular diastolic diameter	42.4±6.2	43.6±5	48±6.1	**0.006*[1]**
LVM/BSA (left vent mass index)	67.7±20.4	71±26.1	89.9±22.4	**0.004 *[1]**
RWT (mm) relative wall thickness	0.389±0.06	0.401±0.05	0.418±0.05	0.398[1]
LVESV (ml) Left ventricular end systolic volume	44.5±21.5	44.4±13.5	63.7±28.5	**0.006 *[1]**
LVEDV (ml) Left ventricular diastolic volume	81.9±23.1	85.9±24.4	115.1±30.6	**<0.0001 *[1]**
LVESV/BSA (left vent. systolic vol. index)	48.5±12	47.7±13.5	58.9±16.3	**0.017 *[1]**
LVEDV/BSA (left vent. diastolic vol. index)	48.5±12	47.7±13.5	58.9±16.3	**0.017 *[1]**

LVM: left ventricular mass, BSA: body surface area. *1: ANOVA-test.*

Table 4 shows that there is statistically significant difference among groups of the study regarding Parameters of LV diastolic function except E/A ratio, E wave and A wave.

Table (4): Parameters of left ventricular diastolic function among study groups

Measurements	Normal weight (Group I)	Overweight (Group II)	Obese (Group III)	*P*-value
LAVi	23.82±4.1	25.45±4.6	31.23±4.7	**<0.001*[1]**
E/A ratio	1.06±0.18	1.00±0.30	1.2±0.44	0.113[1]
E wave	51.77±7.8	52.64±10	58.64±14.9	0.099[1]
A wave	49.9±8.9	55.27±13.9	52.5±15.7	0.504[1]
Septal e'	7.7±1.07	7.07±1.2	5.89±1.0	**<0.001*[1]**
lateral e'	11.23±1.7	10.59±2.1	9.00±1.7	**0.001*[1]**
Average e'	9.45±1.4	8.83±1.5	7.44±1.4	**<0.001*[1]**
Septal E/e'	6.95±1.5	7.82±2.7	10.5±3.7	**<0.0001*[1]**
lateral E/e'	4.68±1.2	5.23±1.8	6.82±2.4	**0.001*[1]**
Average E/e'	5.59±1.2	6.09±2.1	8.14±2.8	**0.001*[1]**
Tricuspid regurge velocity	1.64±0.66	1.82±0.66	2.27±0.7	**0.008*[1]**

LAVi: left atrium volume index, E: early phase of mitral inflow, A: late phase of mitral inflow, E': myocardial early diastolic velocity, S': myocardial systolic velocity, QRS complex: represents ventricular depolarization in ECG.

Table 5 shows that there is statistically significant difference among groups of the study regarding lateral Sa and average Sa. Obese group shows the lowest mean of lateral Sa and average Sa.

Table (5): Parameters of left ventricular systolic function and left ventricular electromechanical delay among study groups

Measurements	Normal weight (Group I)	Overweight (Group II)	Obese (Group III)	*P*-value
Ejection fraction (EF%)	60.32±4.1	60.82±4	59.18±3.5	0.365[1]
Septal Sa	7.41±1.7	7.05±1.8	6.23±1.4	0.113[1]
Lateral Sa	9.73±1.7	9.77±1.9	8.41±1.6	**0.017*[1]**
Average Sa	8.57±1.3	8.41±1.2	7.32±1.3	0.003*[1]
Time from onset of QRS to peak of S' (septal)	93.95±27.35	84.59±27.46	93.41±24.66	0.426[1]
Time from onset of QRS to peak of S' (lateral)	133.36±31.15	133±30.58	156.23±34	**0.025*[1]**
(lateral to septal delay) onset of QRS to peak of S'	39.41±9	48±10.4	62.82±15.3	**<0.0001*[1]**
Time from onset of QRS to onset of S' (Septal)	80.9±25.7	70.9±24.6	74.68±19.9	0.369[1]
Time from onset of QRS to onset of S' (lateral)	104.26±5.3	95±27.3	109.9±26.3	0.172[1]
(lateral to septal delay) onset of QRS to onset of S'	23.36±7.6	24.05±5.9	35.18±9.3	**<0.0001*[1]**

1. ANOVA test.*Statistical significant when p-value <0.05.
S': myocardial systolic velocity, QRS complex: represents ventricular depolarization in ECG

Table 6 shows that LAVi, Diastolic dysfunction, Septal E/e', lateral E/e', and Average E/e have significant direct intermediate correlation with BMI, while (E/A ratio, E wave and Tricuspid regurge velocity have significant direct weak correlation with BMI. In contrast Septal e', lateral e', and Average e' have significant indirect intermediate correlation with BMI. Septal Sa and lateral Sa have significant indirect weak correlation with BMI while Average Sa has significant indirect intermediate correlation with BMI. Time from onset of QRS to peak of S'(lateral) and Time from onset of QRS to onset of S'(lateral) have significant direct intermediate connection with BMI, whereas Time from onset of QRS to peak of S'(lateral) has significant direct weak correlation with BMI.

Table (6): Association among BMI & parameters of left ventricular diastolic function, left ventricular systolic function and left ventricular electromechanical delay among study groups

Measurements	BMI	
	R	*P*-value
LAVi	0.611	**<0.0001***
E/A ratio	0.301	**0.014***
E wave	0.338	**0.005***
A wave	-0.16	0.9
Septal e'	-0.574	**<0.0001***
lateral e'	-0.471	**<0.0001***
Average e'	-0.533	**<0.0001***
hSeptal E/e'	0.532	**<0.0001***
lateral E/e'	0.500	**<0.0001***
Average E/e'	0.511	**<0.0001***
Tricuspid regurge velocity	0.365	**0.003**
Ejection fraction (EF%)	-1.00	0.426
Septal Sa	-0.337	**0.006***
Lateral Sa	-0.354	**0.004***
Average Sa	-0.442	**<0.0001***
Time from onset of QRS to peak of S'(septal)	0.065	0.602
Time from onset of QRS to peak of S'(lateral)	0.341	**0.005***
(lateral to septal delay) onset of QRS to peak of S'	0.633	**<0.0001***
Time from onset of QRS to onset of S' (Septal)	-0.045	0.722
Time from onset of QRS to onset of S' (lateral)	0.177	0.156
(lateral to septal delay)onset of QRS to onset of S'	0.633	**<0.0001***

LAVi: left atium volume index, E: early phase of mitral inflow, E': early myocardial diastolic velocity A: late phase of mitral inflow.

DISCUSSION

Obesity is becoming more and more common throughout the world, and it is a significant contributor to adult cardiovascular disease, mortality, and morbidity. Obesity is an independent risk factor for HF and atrial fibrillation [15].

Due to hyperdynamic circulation, chronic volume overload, and rise in peripheral resistance, being overweight or obese is related with an increase in cardiac preload and afterload. As a result, left ventricular diastolic dysfunction develops as a result of persistent obesity because the left ventricle's ability to fill is impaired [16].

The purpose of the research was to evaluate the association among BMI and LV electromechanical delay in persons free of cardiac illness.

As regard our research, it was found that the mean age was higher among obese group (43.23 years) and females were more prominent than males especially in overweight and obese groups without statistically significant difference.

Smoking, and diabetes mellitus were higher among obese group (36.4% for both), and hypertension was higher among overweight group (36.4%) without any statistical significance. Those findings were similar to **Hizal *et al*.** [17] study about electromechanical delay in the left atrium was detected in a group of 70 morbidly obese patients. Patients were first assessed in terms of their BMI, functional ability and fasting blood sugar before being separated into two groups: those with a BMI of 30 or above were classified as obese (35 patients), while those with a BMI of 25 or below were classified as non-obese. Obese group had higher mean age with higher female's percentage in obese group with statistically insignificant difference.

Also **Hizal *et al*.** [17] reported that although smokers were more among obese group but with statistical insignificant difference.

The DARIOS study revealed that females who are overweight or obese have a higher risk of HF (14% vs. 11%) than men who are overweight or obese (11%) [15].

In this research, the conventional echocardiography for valuation of LV morphology the following was observed; Obesity was associated with proliferation in the septal wall thickness, posterior wall thickness, LV mass index, elevated end diastolic volumes and left atrial enlargement.

After controlling for other clinical factors, we still found that BMI was a significant predictor of these functional changes.

Due to the amplified LVMI but not the rised RWT, eccentric hypertrophy of the LV was evaluated.

Previous research has shown that obesity is linked to eccentric rather than concentric LV remodeling, lending credence to the observation of eccentric LVH [18]. This is in line with **Hizal *et al*.** [17] research in which LV mass index, Patients who were overweight had thicker septums and thicker posterior walls. This contradicts the results of other investigations, which have found a strong correlation between obesity and elevated levels of LVM and RWT.

Differences in the individuals' age, gender, number of co-morbidities, length of obesity, or exclusion criteria may account for this divergent finding. Before the emergence of increasing wall thickness; it was hypothesized that the left ventricular end-diastolic dimensions and LVMI could serve as an early indication of left ventricular dysfunction in morbidly obese individuals [19].

The reason for eccentric hypertrophy of the left ventricle in obese one include rise in the cardiac preload due to chronic volume overload because of increased vascular bed .in the excess adipose tissue, cardiac output [20], hyperdynamic circulation & increase in peripheral resistance and resultant increased afterload resulting in a greater output state of the heart, which can lead to dilation of the heart and eventually heart failure.

These alterations lead to LV diastolic dysfunction, which indicates a decrease in the left ventricle's filling capacity [21].

In our study regarding assessment of left ventricular diastolic dysfunction; increased BMI was showed to be an independent determinant of LV diastolic dysfunction (LVDD). The incidence and severity of LVDD was progressively greater from group 1 (normal) to group 3 (obese) with highest means of parameter in obese group than overweight group.

Among this study BMI was found to be statistically significant correlated with various parameters of diastolic dysfunction; such as LAVi, average E/e' and tricuspid regurge velocity.

Peterson *et al*. [22] showed that as (BMI) increased, so did the load-independent measure of diastolic function derived from tissue Doppler known as the E' wave. For reasons that are not entirely understood, it is believed that diastolic function is impaired first by obesity and then systolic function.

E (r=0.108), A (r=0.123), E′ (r=0.229), and the E/E′ ratio (r=0.138) were all correlated with BMI in **Seo *et al*.** [23] study (all P values <0.05). Higher A, lower E′ and higher E/E′ were independently linked with BMI in multivariate analysis. There was also a striking disparity in the risks of systolic dysfunction in the normal-weight and obese groups.

The LA diameter of obese patients was also observed to be considerably larger than that of non-obese patients by **Hizal *et al*.** [17] (P=0.039). Additionally, the E/A ratio was significantly lower, and the A velocity and E/E' were both significantly higher in obese patients (P=0.002 and P=0.001, respectively).

Cil *et al*. [24] observed a connotation among obesity and LV diastolic dysfunction in a large community study that controlled for other potential risk factors.

Histological hallmarks of obesity-related heart illness observed in autopsies include diffuse myocyte hypertrophy, increased tissue density, and myocardial fibrosis, all of which contribute to the functional abnormalities seen in diastolic failure [25].

Left atrial enlargement and LV diastolic dysfunction are both associated with obesity, which in turn increases left atrial pressure [(26)].

However, atrial functions have significant effects on cardiac performance. Patients with impaired LV function are more susceptible to this impact. Obese people may be at risk for heart failure due to poor LA mechanical performance [(27)].

Additionally, Kossaify and Nicolas assessed 99 cases (mean age 61.59 years) by measuring their BMI and waist circumference, classifying them into 1 of 3 categories based on their BMI (kg/m2): [normal, (18.5-24.9); overweight, (25-29.9); obese, (.29.9)]. Early (E) and late (A) diastolic mitral annulus velocities, as well as early (E′), were measured. Tissue Doppler imaging reveals that excess weight and obesity have a direct, adverse effect on diastolic function [(28)].

CONCLUSION

Left ventricular electromechanical changes and obese people with normal heart rhythm and no indications of heart trouble may have latent systolic dysfunction.

DECLARATIONS

- **Consent for publication:** I attest that all authors have agreed to submit the work.
- **Availability of data and material:** Available
- **Competing interests:** None
- **Funding:** No fund
- **Conflicts of interest:** no conflicts of interest.

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