Diastolic Function Indices among Obese Patients

Amjad Ajil Shandookh[1]; Abdulameer A. Abdulhameed[2], Mohammed Mutar Al-Lami [3]

1. Thiqar health directorate AL-jabaish general hospital
2. University of Basrah college of medicine head department of medicine
3. Interventional cardiologist Collage of medicine Duhok university

Received:14.7.2024 **Accepted:3.12.2024**

Abstract

Background: The increased prevalence of obesity and its strong association with cardiovascular disease has resulted in exceptional awareness for obese individuals. Obesity affects the hemodynamics of the cardiovascular system in general and left ventricular diastolic function in specific. In this study, we aimed to estimate the effect of obesity on the echocardiographic left ventricular diastolic function indices.

Patients and Methods: In this case-control study, we included 113 patients from Fayhaa General Hospital in Basrah between Feb and Aug 2016. The patients who were free of diabetes mellitus, hypertension, and any history of heart diseases -structural or functional met the eligibility criteria. The patients were divided into three groups based on their BMI including; 45 patients with normal BMI as the control group, 35 overweight patients, and 33 obese patients as case groups.

Results: BMI is positively and directly correlated to both Peak A Velocity and DT, and negatively on the E/A Ratio with inverse correlation. Higher BMI is correlated with worsening diastolic function regardless of traditional risk factors for diastolic dysfunction. The increase in age of more than 45 years has a significant association with all the indices of LVDD except for DT. The study reveals that smoking resulted in a significant acute alteration in the LV diastolic function.

Conclusions: All obesity grades have unfavorable consequences on the LV diastolic function, with subclinical changes in diastolic indices (Peak E, Peak A, E/A Ratio, and DT), in individuals with no previous comorbidities, even after adjustment for (age, sex, and smoking).

Keywords: BMI, Diastolic Dysfunction, Peak E Velocity, Peak A Velocity, E/A Ratio, Deceleration Time

Corresponding author: Abdulameer A. Abdulhameed

✉ **E-mail:** drabd7@gmial.com

Introduction

Obesity is a chronic disease of multifactorial etiologies and represents a high risk for health, reaching epidemic proportions (1). Obesity directly affects cardiovascular hemodynamics and indirectly increases overall morbidity and mortality (2). Body Mass Index (BMI) is the standard measure of obesity with the impact of the LV mass, wall thickness, and internal dimension (3), regardless of the existence of comorbidities like diabetes mellitus, renal failure, and hypertension (4), even in the young and otherwise healthy subjects (5, 6).

The differences in the regional or global strain of LV structure may be identified even years before they develop signs and symptoms of heart failure (HF) (2). Diastolic dysfunction (DD) may occur in individuals with isolated obesity due to defective ventricular loading as the increase in cardiac output and stroke volume leads to dysfunction (4, 7).

Unlike the systolic dysfunction that is not so common (7), the DD is the most important component of cardiac function impairment reflecting abnormalities of diastolic dimensions, filling or relaxation of the LV (1, 8, 9). Diastolic function abnormalities can lead to an increase in the

Left Ventricular End Diastolic Pressure i.e. (LVEDP), Mean Left Atrial Pressure (MLAP), and Pulmonary Capillary Wedge Pressure (PCWP) (collectively named LV filling pressure). Filling pressures are elevated if the mean PCWP is more than 12 mmHg, and LVEDP is more than 16 mm Hg (10).

Diastolic function evaluation has become a vital part of any full echocardiographic examination and is recommended by the current guidelines. No parameter is specific for DD prediction and elevated filling pressure because of load dependence and the effects of an increase in heart rate and ageing. So, integrations of two-dimensional echo findings with multiple Tissue Doppler Imaging (TDI) parameters are essential for diastolic function assessment (10).

The diagnosis of DD is made more challenging by obesity and limiting its assessment by flow Doppler, given that most diastolic indices are load-dependent (1) such as mitral inflow velocities and LV ejection fraction (LVEF). The evaluation of functional abnormalities whether obesity-related or loading condition-related related cannot be assured (8).

DD contributes to the development of Heart Failure with a Preserved Ejection Fraction (HFpEF) (1, 8), which occurs with no valvular or pericardial lesions and carries a better short-term prognosis as compared to HF due to systolic dysfunction, however, the hospitalization and mortality on long-term are almost the same (10).

The high prevalence of obesity increases cardiovascular risk because it is a modifiable and independent risk factor for several morbidities (11), especially in women (12), and requires thorough echocardiography in obese individuals, with respect for their peculiarities to identify high-risk individuals for early intervention (1, 4). The morbidity and mortality may occur at a younger age because of the initial commencement of the disease process and may be avoided by initiation of weight loss primarily (1, 13). This study aimed to evaluate the effect of obesity on the Echocardiographic Left Ventricular Diastolic Indices.

Patients and Methods

Study design

This case-control study included 113 individuals from Fayhaa General Hospital in Basrah from February to August 2016. These persons were referred from the outpatient clinics for clinical evaluation. After receiving, the informed consent from these patients, we obtained a history of the patients and collected the basic information including age, sex, and smoking history. The overweight and obese patients were considered cases and the normal weight was the control group in this study.

Inclusion and exclusion criteria

The obese and non-obese individuals aged 18 years and older of both male and female genders were eligible for this study. The patients with the following comorbidities were excluded from the study; cardiovascular diseases like ischemic heart, peripheral vascular and cerebrovascular diseases; hypertension with or without antihypertensive treatment; diabetes mellitus with or without antidiabetic treatment; valvular heart diseases. The patients were excluded from the study if they had systolic BP≥140 mmHg with or without diastolic BP≥90 mmHg which defined hypertension and confirmed the next day. We excluded eight patients from the study because they had DM i.e. (HbA1c ≥ 6.5 and fasting blood sugar ≥126 mg/dl with confirmation of abnormal results on repetition).

Data collection and measures

The general information of the patients was obtained through a self-reported technique and recorded in a pre-designed questionnaire. The patients were labeled as current smokers if they have smoked one hundred cigarettes or more in their lifetime and presently smoke on a daily or non-daily basis. Non-smokers were those who were former smokers or never smokers.

Anthropometric Measurements

Initially, the height, weight, and BMI of 147 patients were measured according to the World Health Organization, BMI was calculated using:

BMI= (Weight in Kilogram) / (Height in M^2) So, individuals with BMI less than 25kg/m^2 were determined as normal weight, those with BMI range (25 - 29.9) kg/m^2 were considered overweight, and patients with BMI ≥ 30kg/m^2 were considered obese patients. Blood pressure was measured using a standard sphygmomanometer with the patient seated in a quiet environment following five minutes of rest. An average of two readings, at least thirty seconds apart were considered the mean value of blood pressure. Accordingly, 26 patients were labeled as newly diagnosed hypertension (sixteen patients obese, eight overweight, and two below 25 kg/m^2) and excluded from the study.

Biochemical Measurements

From each patient, after an 8-12 hour fasting, 10 ml of blood was taken and divided into two tubes (EDTA tube, and clot activator tube), centrifuged, and analyzed. HbA1c measurement was done by Ion Exchange High-Performance Liquid Chromatography HPLC using a Biorad D10. Fasting blood sugar from clot activator tube measured using Biolyzer 300, Germany.

Echocardiographic Measurements

We divided the remaining 113 patients who met the enrollment criteria into three groups (45 patients with BMI<25 kg/m^2 as a control group), 35 with overweight, and 33 with obese as case groups). One trained echocardiographer examined the patients using transthoracic echocardiography (Philips CX50). The LV diastolic function assessment was performed using an apical 4-chamber view. The Pulsed-Wave Doppler Imaging tested the transmitral flow at the level of mitral valve leaflet tips. We evaluated the peak velocities of the Early Phase (Peak E) and Late Phase (Peak A) of the mitral inflow; the ratio between them (E/A), and the Deceleration Time (DT) – that is the time taken from the maximum E point to baseline and peak pulmonary artery pressure. Assessment of all parameters was according to the ASE and the EACVI recommendations (14).

Statistical Analysis

Description of continuous variables (age, BMI, and diastolic function indices E, A, E/A ratio, and DT) was performed as Mean ± Standard Deviation SD. The categorical variables (BMI, age, sex, and smoking) were presented as frequencies and percentages. Assessment of these variables was done using One-way ANOVA with Post Hoc analysis and Mean Plots tests. The results are statistically significant only if the p-value is less than 0.05. Data were tested using IBM SPSS statistical software version 22.0 (IBM Corp. Released 2013. IBM SPSS Statistics for Windows, Version 22.0. Armonk, NY: IBM Corp.).

Results

General characteristics of patients by BMI Categories

We enrolled 113 patients with a mean age (46.2±6.9 years, ranging from 38 - 63 years). Out of them 46 (40.7%) were males and 34 (30.1%) were current smokers. By using the 45 years as a cut point to divide the patients, patients aged < 45 years were 58 (51.3%), and those with age ≥ 45 years were 55 (48.7%).

The mean BMI of the sample is (27.24±3.80 kg/m^2). The BMI was the parameter to divide the patients into three groups: Patients with BMI (<25 kg/m^2): (Normally weighed group) were 45 (39.8%), males were 12 (26.1%), 9 (26.5%) were current smokers and 22 (40%) aged 45 years and more. The mean BMI in this group is (23.54±0.17 kg/m^2). Patients with BMI (25 - 29.9 kg/m^2): (Overweight group) was 35 (31%), in this group 23 (50%) were males, 13 (38.2%) were current smokers, and 11 (20%) were aged ≥ 45 years. The mean BMI here is (26.96±0.66 kg/m^2). Patients with BMI (≥ 30 kg/m2): (Obese group) were 33 (29.2%), 11

(23.9%), 12 (35.3%) current smokers, and 22 (40%) aged ≥45 years. The mean BMI here is (32.57±1.20 kg/m^2; Table 1).

Table 1: General characteristics of the study population by BMI Categories (N=1١3)

General characteristics		All patients N (%)	BMI Categories Kg/m^2 N (%)		
			Normal <25 45 (39.8)	Overweight 25-29.9 35 (31)	Obese ≥30 33 (29.2)
Age Mean (SD)		46.23 (6.91)	43.3 (3.50)	34.8 (4.30)	52.6 (8.30)
Age Group (year)	<45	58 (51.30)	23 (39.70)	24 (41.40)	11 (19)
	≥45	55 (48.70)	22 (40)	11 (20)	22 (40)
Male No (%)		46 (40.70)	12 (26.10)	23 (50)	11 (23.90)
Smokers No (%)		34 (30.10)	9 (26.50)	13 (38.20)	12 (35.30)
Diastolic Function Indices M(SD)	Peak E cm/s	77.92 (4.48)	75.69 (4.88)	82.00 (3.14)	76.67 (0.48)
	Peak A cm/s	65.26 (13.64)	53.96 (4.70)	68.43 (14.15)	77.33 (8.18)
	E/A ratio	1.24 (0.25)	1.53 (0.15)	1.13 (0.24)	1.00 (0.19)
	DT ms	172.50 (16.87)	157.22 (7.73)	170.63 (2.95)	195.33 (6.00)

Relation between BMI and the Left Ventricular Diastolic Function

The means of peak E-wave, peak A-wave, E/A ratio, and DT in the study population were (77.9±4.48 cm/s, 65.2±13.6 cm/s, 1.24±0.25, and 172.5±16.8 ms) respectively. The mean Peak E-wave (cm/s) was increased from (75.6±4.8) in patients with normal weight to be (82±3.14) in overweight patients and decreased to 76.67±0.47 in obese patients. The mean peak A-wave (cm/s) was increased from 53.9±4.6 cm/s in normal BMI to 68.42±14.1 cm/s in overweights, and to 77.33±8.18 cm/s) in obese. The E/A ratio in the normal BMI was 1.41±0.17 was decreased to 1.25±0.25 in overweight, and decreased further to 1.0±0.11 in obese. The DT (ms) in the normal weight group 157.2±7.73 ms) was increased in overweight's to 170.63±2.9 ms and in the obese patients to 195.33±5.9 ms (Table 2a).

Table 2a: Correlation between BMI and Diastolic Function Indices

BMI Kg/m^2	Diastolic function indices Mean (SD)			
	Peak E*	Peak A*	E/A ratio	DT#
Normal weight <25	75.69 (4.88)	53.96 (4.70)	1.41 (0.17)	157.22 (7.73)
Overweight 25-29.9	82.00 (3.14)	68.42 (14.16)	1.25 (0.25)	170.63 (2.95)
Obese ≥30	76.67 (0.48)	77.33 (8.18)	1.00 (0.11)	195.33 (6.00)
P <0.0005 * By cm/s # by ms				

The study showed that the mean value of the Peak E of the overweight patients (82.00 cm/s) was significantly higher than the patients with normal weight (75.69 cm/s) and obese patients (76.67 cm/s). However, in terms of Peak A, the patients with higher BMI had higher mean values compared to those with lower BMI. A similar pattern was found for the DT between study groups as well (Table 2b and Fig 1).

Table 2b: Pairwise comparisons of Peak E between study groups

	BMI kg/m^2		*P*- value
Peak E cm/s	Normal weight <	Overweight	<0.0005
	Normal weight	Obese	<0.233
	Overweight >	Obese	<0.0005
* In between-group comparison for all of the Peak A, E/A ratio, and DT were statistically significant (p<0.0005)			

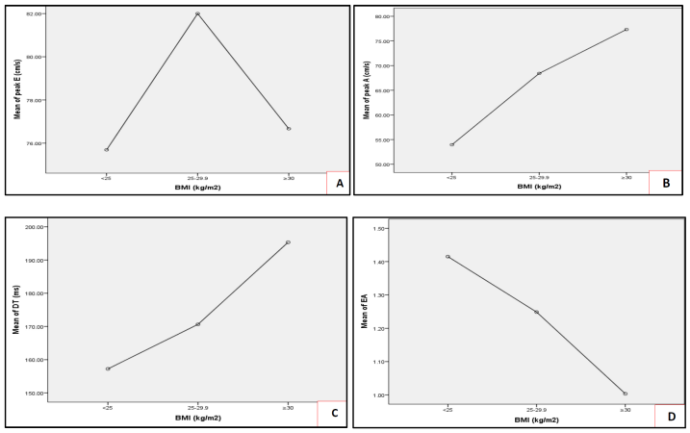

Figure 1: Mean Plot of LV Diastolic Function Indices vs. BMI

The Echo Indices among patients' characteristics

Peak E: In terms of Peak E, the study showed that the younger patients (<45 years old) had significantly higher mean values (80.51) compared to those patients aged ≥45 (75.20; P<0.0005). In addition, its mean value was significantly higher in female patients (77.14) vs. male patients (79.06; P=0.025). Its mean values were not statistically significant among smoker and non-smoker patients (P=0.195).

Peak A: About Peak A, in contrast with Peak E, the study showed that the mean value of Peak A was significantly higher in older patients vs. younger patients (70.20 vs. 60.58; P<0.0005) and in male vs. female patients (69.58 vs. 62.29; P=0.005). A similar pattern was found in terms the Peak A in smoking categories.

E/A ratio: The mean value of E/A ratio was significantly higher in younger patients (1.37) vs. older patients (1.10; P<0.0005). Its level was not statistically significant between genders (P=0.11) and smoking (P=0.307).

DT: The level of DT was not statistically significantly different in different age groups (P=0.429) and sex (0.058). But, its level was significantly higher in current smokers compared to non-smokers (179.44 vs. 169.51; P=0.004; Table 3).

Table 3: Comparison of Echo Indices in patients with different characteristics

Cofounders		Echo indices mean (SD)			
		Peak E* (*p-Value*)	Peak A* (*p-Value*)	E/A ratio (*p-Value*)	DT# (*p-Value*)
Age	<45	80.51 (4.05)	60.58 (12.01)	1.37 (0.24)	171.27 (15.94)
	≥45	75.20 (3.08)	70.20 (13.60)	1.10 (0.17)	173.80 (17.86)
	p-value	<0.0005	<0.0005	<0.0005	0.429
Sex	Male	79.06 (4.32)	69.58 (15.75)	1.19 (0.28)	176.13 (14.69)
	Female	77.14 (4.45)	62.29 (11.16)	1.27 (0.22)	170.01 (17.91)
	p-value	0.025	0.005	0.11	0.058
Smoking	Current	78.76 (4.44)	68.58 (14.94)	1.21 (0.28)	179.44 (16.47)
	Non	77.56 (4.48)	63.83 (12.87)	1.25 (0.24)	169.51 (16.25)
	p-value	0.195	0.089	0.307	0.004

* By cm/s; # by ms

Discussion

Our study has shown that the E, A, E/A, and DT values did not differ so much across the spectrum of obesity groups, and the associations of these indices with obesity are significant and as reported in many studies (6, 11, 12, 15-17). We found that different degrees of changes in LV diastolic function indices were common in our cohort, and these alterations in diastolic function correlated strongly with BMI (15, 17). The overall changes in diastolic function indices in our study were similar to those of AlJaroudi et al. and Kuznetsova et al. (18, 19), with a similar distribution of patients among BMI groups. Our results were additionally strengthened and supported because the majority of our patients were hypertension-free, with a much younger cohort than that presented by Russo (11, 18) and AlJaroudi (18) et al. Of particular interest are patients who are <45 years old.

So, pathophysiologically speaking, the presence of lone obesity may be a determinant of the subclinical abnormalities in cardiac function and subsequent LVDD, which is present in all grades of isolated obesity and with a correlation to the BMI (7, 15, 17). Peterson et al. studied uncomplicated obese individuals and found that there was only impaired diastolic function with normal or hyperdynamic systolic function (12). We found that diastolic function derangements are common, not only in severely obese individuals but also in overweight, and alterations in diastolic function correlated strongly with BMI; that is

similar to Pascual et al. and Pandey et al. results (7, 15).

Due to obesity-associated chronic volume overload, the cardiac adaptive and remodeling mechanism occurs in the form of an early eccentric ventricular hypertrophy and DD (8, 15, 17, 20, 21), indicating that all obese individuals may have some architectural cardiac changes and cardiomyopathy (7, 15, 17). The increasing body adiposity will alter the cardiac metabolism, leading to myocardial fatty infiltration, inflammation and cardiac lipotoxicity (18), insulin resistance, and neuroendocrine activation (22), impairment of the relaxation of LV, and elevation in LV diastolic filling pressures (8), and increased dependency on LA contraction for average filling (7, 15). Afterload is elevated, not only because of increased preload but also because obesity will raise vascular resistance and heighten the conduit artery stiffness (20, 21).

Higher BMI is associated with diastolic function worsening regardless of the traditional risk factors known to cause DD (1), or by promoting other risk factors such as hypertension, dyslipidemia, and DM with unknown mechanisms (16, 22).

The Doppler method of measuring indices of LV filling is valuable in assessing diastolic function. However, it is known that obesity causes volume overload, so normal values of the diastolic function parameters may result. The increased left atrial pressure due to intravascular volume can mask the derangements observed in the early phases of abnormal diastolic relaxation (15).

Regarding the diastolic function in this study, there was an inverse association between E/A ratio which is showing a DD predominantly of abnormal relaxation type to the extent that BMI increases. These findings corroborate the literature, in which increased BMI is a predictor of LVDD, regardless of age and presence of comorbidities (15, 23).

When we introduce other confounders to BMI to affect the LV diastolic function like age, sex, and smoking, there was no statistical significance in the deceleration time of the obese group, especially in males and in those older than 45 years when compared to the patients with BMI<25 kg/m^2. Our results are similar to Shalaby et al. because the mitral DT was significantly greater in the BMI≥30 kg/m^2 group (8).

It is important to note that the rate and extent of ventricular relaxation are not the only leading causes of the mitral inflow changes, but also it is affected by age, and loading premorbid conditions (8, 21). The DT was significantly prolonged in the obese patients (7, 17). Our findings matched the observations of Chadha et al. (17) and Di Bello et al. (21).

Taking the age as a lone confounder to the BMI in different groups strengthens the results of our study. The total age mean was (46.2±6.9 years and a range of 38-63 years) which is similar to Ilyas et al. (1) and Koç et al. (24), with similar distribution between groups. It is less than that of Rubio et al. and Russo et al. (11, 25), because they dealt with a larger number of elderly obese patients, and it is more than that of Kumar (6), Pandey (7), Yassen (13), and Pascual (15) et al. who dealt with a smaller number of young adult patients.

The second confounder is gender. We try to decrease the gender-related biased effect on the LV diastolic function among obese individuals, our sample is nearly regularly distributed between the two genders, with a male population constituting 41% of the patients and similar BMI means (27.68±3.29 kg for males and 26.92±4.11 kg for females). There is a significant relation between this confounder and the diastolic indices except for the DT which is similar to Russo (11), Sokmen (26), and Lee (27) et al. with a similar distribution.

Poyraz et al. (9) neglected the gender effect on obesity-related DD by having a male-to-female ratio of 6:1. Peterson et al. (12) and Pascual et al. (15), dealt only with obese females, and Shekharappa et al. (28), dealt only with obese males.

Past studies suggested that women diminish their diastolic function more quickly than men, even in a normal ageing process regardless of the BMI (29, 30), and with obesity, it is worsening (15, 30), which may be due to the lost protective effect of estrogen in preventing the cardiac remodeling and DD (31).

The effect of smoking as a significantly associated confounder for BMI on DD is evident in this study, especially in the obese patients group, but not we consider the patient in the cohort as a unity, which is similar to Karakaya et al. (32). About one-third of the enrolled patients are current smokers, which is similar to their prevalence in Bennet et al. (33) study.

Chronic smoking has acute on chronic effects on the diastolic function in healthy individuals, which may be due to changes in the mitral inflow that cause impairment of the endothelial relaxation (33-35), producing a significant decrease in (Peak E) and increase in (Peak A) velocities with consequent reduction in E/A ratio (35, 36). The etiology of these changes is multifactorial, and cannot be elucidated only by the heart rate increase or loading conditions changes (32, 36).

Even in the presence of age, gender, and smoking as confounders to BMI to affect the DD in our cohort, the effect of BMI per se is evident on all the diastolic function indices.

Limitation of the Study

The study faced several limitations due to its single-centre design, small sample size, and overall design, which restricted its ability to identify confounders and draw cause-effect inferences. Additionally, despite efforts to select healthy subjects, we cannot guarantee that all participants were indeed healthy, potentially affecting our results. The lack of a detailed history of obesity duration and the absence of a longitudinal follow-up to assess whether diastolic dysfunction (DD) was worsening further limited our ability to evaluate the prevalence of clinically evident morbidities accurately.

Moreover, the evaluation of left ventricular diastolic dysfunction (LVDD) in our study did not include high load dependence parameters, such as isovolumic relaxation time or pulmonary venous flow, which would have allowed us to detect DD even in pseudo-normalized flow patterns. Another limitation was that body mass index (BMI) was the only measure of obesity used. Investigating body fat distribution, cytokines, leptin, and renin-angiotensin-aldosterone system activity could have provided more insight into the underlying mechanisms.

Finally, the study did not include any invasive measures of LVDD. The echocardiographic measurements and velocities used are quantitative traits subject to measurement errors, which could impact the study's findings.

Conclusions

The study highlights that all grades of obesity have unfavorable consequences on LV diastolic function, significantly associating with all subclinical LVDD indices in patients with no previous comorbidities, even after adjustments for age, sex, and smoking. Specifically, BMI shows a positive and direct correlation with both Peak A Velocity and deceleration time (DT), while negatively impacting the E/A Ratio, revealing an inverse relationship. The correlation between BMI and Peak E velocity is described as funnel-shaped. The worsening of LV diastolic function occurs with higher BMI irrespective of traditional risk factors, underlining the critical impact of obesity on cardiac health. Overwhelming evidence supports the importance of obesity in the pathogenesis of LVDD. Furthermore, there is a significant association between increases in age beyond 45 years and LVDD indices, except for DT, and also a significant association between gender and LVDD, except for DT and E/A ratio. The study also reveals a statistically significant relationship between smoking and DT, indicating that cigarette smoking can result in a significant acute alteration in LVDD.

Recommendations

As recommended by recent guidelines, the evaluation and integration of diastolic baseline and follow-up function must become an integral part of a comprehensive echocardiographic assessment. When evaluating obese individuals, it is essential to consider the effect of higher BMI values on the cardiovascular system in general, and left ventricular diastolic dysfunction (LVDD) specifically, as these individuals may have treatable preclinical or subclinical diastolic dysfunction.

To gain further insights, a prospective long-term longitudinal study should be conducted to answer key questions, such as the effect of weight reduction on LVDD and whether significant weight loss can reverse diastolic dysfunction earlier and potentially alter outcomes. Given the high frequency of changes in LV diastolic indices in the preclinical phase among morbidly obese individuals, a careful echocardiographic assessment is required to identify those at greater risk, enabling earlier interventions.

References

1. Ilyas, M, I Ullah, F Ahmad, and U Ali. Correlation of body mass index with indices of diastolic function in normotensive, nondiabetic patients. Pakistan Heart Journal. 2016. 49(1).

2. Mathew, B, L Francis, A Kayalar, and J Cone. Obesity: effects on cardiovascular disease and its diagnosis. The Journal of the American Board of Family Medicine. 2008. 21(6): 562-568.

3. Rashid, MA, BA Qureshi, N Ahmed, and MA Sherwani. Impact of body mass index on left ventricular mass. Journal of Ayub Medical College Abbottabad. 2014. 26(2): 167-169.

4. Tavares, IdS, ACS Sousa, RS Menezes Filho, MHd Aguiar-Oliveira, JA Barreto-Filho, AFd Brito, et al. Left ventricular diastolic function in morbidly obese patients in the preoperative for bariatric surgery. Arquivos Brasileiros de Cardiologia. 2012. 98: 300-306.

5. El Solh, A, *Critical care management of the obese patient*. 2012: John Wiley & Sons.

6. Kumar, HP, M Maheshwari, and H Badjatiya. A Study on Prevalence of Diastolic Dysfunction in Normotensive Obese Young Adults. 2013.

7. Pandey, AK, A Das, BM Sreehari, Y Himabindu, N Farha, R Parvati, et al. Assessment of myocardial mechanics in overweight and obese Indian subjects. Indian J Physiol Pharmacol. 2013. 57(2): 35-43.

8. Tanalp, AC, A Bitigen, C Cevik, D Demir, O Ozveren, K Tigen, et al. The role of tissue Doppler study in the assessment of left ventricular dysfunction in obesity. Acta cardiologica. 2008. 63(5): 541-546.

9. Poyraz, F, M Turfan, SA Kocaman, HU Yazici, N Sen, M Tulmac, et al. Effect of overweight and obesity on the left ventricular systolic and diastolic functions in patients with acute myocardial infarction. Clinical and Investigative Medicine. 2012. 35(4): E229-E236.

10. Parashar, S. Diastolic functions: evaluation & clinical applications. Cardiovascular Journal. 2009. 1(2): 132-141.

11. Russo, C, Z Jin, S Homma, T Rundek, MS Elkind, RL Sacco, et al. Effect of obesity and overweight on left ventricular diastolic function: a community-based study in an elderly cohort. Journal of the American College of Cardiology. 2011. 57(12): 1368-1374.

12. Peterson, LR, AD Waggoner, KB Schechtman, T Meyer, RJ Gropler, B Barzilai, et al. Alterations in left ventricular structure and function in young healthy obese women: assessment by echocardiography and tissue Doppler imaging. Journal of the American College of Cardiology. 2004. 43(8): 1399-1404.

13. Yaseen, RI, MK Ahmed, and WA Hamed. Assessment of abnormal LV myocardial deformation properties in obese patients by 2D based strain and strain rate imaging. The Egyptian Heart Journal. 2015. 67(3): 183-191.

14. Nagueh, SF, OA Smiseth, CP Appleton, BF Byrd, H Dokainish, T Edvardsen, et al.

Recommendations for the evaluation of left ventricular diastolic function by echocardiography: an update from the American Society of Echocardiography and the European Association of Cardiovascular Imaging. European Journal of Echocardiography. 2016. 17(12): 1321-1360.

15. Pascual, M, D Pascual, F Soria, T Vicente, A Hernandez, F Tebar, et al. Effects of isolated obesity on systolic and diastolic left ventricular function. Heart. 2003. 89(10): 1152-1156.

16. Alkersh, AM, MA Ebada, RI Yaseen, and WF Abdelazez. Effects of isolated obesity on left ventricular function: a longitudinal strain imaging study. Menoufia Medical Journal. 2014. 27(1): 130-135.

17. Chadha, D, A Swamy, S Malani, R Ganjoo, and O Mathew. Impact of body mass index on left ventricular function. Medical Journal Armed Forces India. 2009. 65(3): 203-207.

18. Aljaroudi, W, C Halley, P Houghtaling, S Agarwal, V Menon, L Rodriguez, et al. Impact of body mass index on diastolic function in patients with normal left ventricular ejection fraction. Nutrition & diabetes. 2012. 2(8): e39-e39.

19. Kuznetsova, T, L Herbots, B López, Y Jin, T Richart, L Thijs, et al. Prevalence of left ventricular diastolic dysfunction in a general population. Circulation: Heart Failure. 2009. 2(2): 105-112.

20. Barbosa, MM, AM Beleigoli, M de Fatima Diniz, CV Freire, AL Ribeiro, and MCP Nunes. Strain imaging in morbid obesity: insights into subclinical ventricular dysfunction. Clinical cardiology. 2011. 34(5): 288-293.

21. Di Bello, V, F Santini, A Di Cori, A Pucci, C Palagi, M Delle Donne, et al. Relationship between preclinical abnormalities of global and regional left ventricular function and insulin resistance in severe obesity: a Color Doppler Imaging Study. International journal of obesity. 2006. 30(6): 948-956.

22. Iacobellis, G, MC Ribaudo, G Leto, A Zappaterreno, E Vecci, U Di Mario, et al. Influence of excess fat on cardiac morphology and function: study in uncomplicated obesity. Obesity research. 2002. 10(8): 767-773.

23. Cil, H, S Bulur, Y Türker, A Kaya, R Alemdar, A Karabacak, et al. Impact of body mass index on left ventricular diastolic dysfunction. Echocardiography. 2012. 29(6): 647-651.

24. Koc, F, M Tokac, C Kaya, M Kayrak, T Karabağ, M Vatankulu, et al. Diastolic functions and myocardial performance index in obese patients with or without metabolic syndrome: a tissue Doppler study. Turk Kardiyoloji Dernegi Arsivi: Turk Kardiyoloji Derneginin Yayin Organidir. 2010. 38(6): 400-404.

25. Rubio, L, G Molina, and M Anaya. Relationship of body mass index with changes in cardiac geometry and function in 5,898 patients evaluated by transthoracic echocardiography. Arq Bras Cardiol. 2015. 28: 3-16.

26. Sokmen, A, G Sokmen, G Acar, A Akcay, S Koroglu, M Koleoglu, et al. The impact of isolated obesity on right ventricular function in young adults. Arquivos brasileiros de cardiologia. 2013. 101: 160-168.

27. Lee, SL, M Daimon, MR Di Tullio, S Homma, T Nakao, T Kawata, et al. Relationship of left ventricular diastolic function to obesity and overweight in a Japanese population with preserved left ventricular ejection fraction. Circulation Journal. 2016. 80(9): 1951-1956.

28. Shekharappa, KR, S Smilee Johncy, P Mallikarjuna, K Vedavathi, and MP Jayarajan.

Correlation between body mass index and cardiovascular parameters in obese and non obese in different age groups. Int J Biol Med Res. 2011. 2(2): 551-555.

29. Redfield, MM, SJ Jacobsen, BA Borlaug, RJ Rodeheffer, and DA Kass. Age-and gender-related ventricular-vascular stiffening: a community-based study. Circulation. 2005. 112(15): 2254-2262.

30. Carvalho, J-C, P Farand, HD Do, M-C Brochu, F Bonenfant, and S Lepage. Effect of age and sex on echocardiographic left ventricular diastolic function parameters in patients with preserved ejection fraction and normal valvular function. Cardiology journal. 2013. 20(5): 513-518.

31. Zhao, Z, H Wang, JA Jessup, SH Lindsey, MC Chappell, and L Groban. Role of estrogen in diastolic dysfunction. American Journal of Physiology-Heart and Circulatory Physiology. 2014. 306(5): H628-H640.

32. Karakaya, O, I Barutcu, AM Esen, D Kaya, M Turkmen, M Melek, et al. Acute smoking-induced alterations in Doppler echocardiographic measurements in chronic smokers. Texas Heart Institute Journal. 2006. 33(2): 134.

33. Bennet, L, C Larsson, M Söderström, L Råstam, and U Lindblad. Diastolic dysfunction is associated with sedentary leisure time physical activity and smoking in females only. Scandinavian journal of primary health care. 2010. 28(3): 172-178.

34. Eroglu, E, S Aydin, F Yalniz, AK Kalkan, F Bayrak, and M Degertekin. Chronic cigarette smoking affects left and right ventricular long-axis function in healthy young subjects: a Doppler myocardial imaging study. Echocardiography. 2009. 26(9): 1019-1025.

35. Alshehri, AM, AM Azoz, HA Shaheen, YA Farrag, MAA Khalifa, and A Youssef. Acute effects of cigarette smoking on the cardiac diastolic functions. Journal of the Saudi Heart Association. 2013. 25(3): 173-179.

36. Ghaidari, M, MA Akbarzadeh, S Yazdani, MA Pirnfar, and A Aslani. Effect of acute smoking on diastolic function. International Cardiovascular Research Journal. 2010. 4(2).

مؤشرات الوظيفة الانبساطية عند المرضى الذين يعانون من السمنة

المقدمة: كان للزيادة الكبيرة في انتشار السمنة وعلاقتها القوية مع أمراض القلب والأوعية الدموية الاثر الكبير في الاهتمام غير المسبوق في فهم تأثير السمنة على الديناميكية القلبية الوعائية بصورة عامة، وعلى الوظيفة الانبساطية للبطين الايسر على وجه الخصوص. هدف هذه الدراسة هو تقييم تأثير السمنة على مؤشرات فحص صدى القلب للوظيفة الانبساطية للبطين الايسر.

المرضى والوسائل: دراسة مستعرضة مستقبلية قصيرة الأمد لمائة وثلاث عشر مريضا اقيمت في مستشفى الفيحاء العام في البصرة للفترة من شباط الى اب ٢٠١٦ تتضمن المرضى المحالين الى العيادة الخارجية للفحص السريري. أهم شرط لادخال المريض الدراسة هو كونه سالما من الاصابة بمرض السكري، ارتفاع ضغط الدم واي امراض قلبية سواءا اكانت هيكلية او وظيفية. بعد ان تم اخذ موافقة المريض، يتم تدوين كل من معدل كتلة الجسم، العمر، الجنس، ضغط الدم، قياس الجلوكوز في الدم (صائما) مع او بدون نسبة HbA1c. تم بعدها تقسيم المرضى الى ثلاث مجموعات حسب معدل كتلة الجسم: (خمسة واربعون مريضا بمعدل كتلة جسم طبيعية اعتبروا كمجموعة سيطرة)، (٣٥ مريضا من ذوي الوزن الزائد)، و (٣٣ مريضا سمينا).

النتائج: يوثر معدل كتلة الجسم بصورة موجبة ومباشرة على كل من السرعة القصوى للجريان في طورها المتاخر (Peak A) وعلى سرعة التباطؤ، ويؤثر بعلاقة سلبية على نسبة (E/A). اقترنت المستويات العالية لمعدل كتلة الجسم مع تدهور الوظيفة الانبساطية بدون الاخذ بنظر الاعتبار عوامل الاختطار التقليدية للاعتلال الانبساطي.فيما عدا سرعة التباطؤ, كان لزيادة العمر فوق خمسة و اربعين عاما ارتباطا ملحوظا مع جميع مؤشرات اعتلال الوظيفة الانبساطية للبطين الايسر. وارتبط الجنس ارتباطا ملحوظا مع مؤشرات الاعتلال الانبساطي للبطين الايسر عدا كل من نسبة (E/A) وسرعة التباطؤ. ومن الناحية الاخرى، كان ارتباط التدخين ملحوظا مع سرعة التباطؤ، وقد اكدت هذه الدراسة على ان التدخين من الممكن ان ينتج اضطرابا حادا في الوظيفة الانبساطية للبطين الايسر.

الاستنتاج: كانت لكل درجات السمنة عواقب غير مرغوبة على الوظيفة الانبساطية للبطين الايسر, و كانت مرتبطة ارتباطا ملحوظا مع كل الاختلالات دون السريرية في مؤشرات الوظيفة الانبساطية (اي السرعات القصوى للجريان عبر الصمام التاجي في طورها المبكر (E) و في طورها المتأخر (A), و من ثم النسبة بين السرعتين (E/A) و سرعة التباطؤ) عند الاشخاص الذين لا يعانون من امراض سابقة, حتى بعد التوفيق مع كل من العمر, الجنس, و التدخين كمربكات احصائية.