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ORIGINAL RESEARCH

A Community-based Study on the impact of Obesity and Overweight on Left Ventricular Diastolic Function in an Elderly Cohort

[1]Panchanan Sahoo, [2]Ritesh Acharya

[1]Associate Professor, [2]Senior Resident, Department of Cardiology, Kalinga Institute of Medical Sciences, Bhubaneswar, India

Correspondence:
Panchanan Sahoo
Associate Professor, Department of Cardiology, Kalinga Institute of Medical Sciences, Bhubaneswar, India

ABSTRACT

Objective: Left Ventricular diastolic dysfunction (LVDD) is an asymptomatic disease linked to eventual heart failure. Obesity and overweight are not known to be independently linked to LVDD. The objective is to adjudicate whether increased body size has an independent influence on LVDD.

Study design and sample: For this study, a total of 495 subjects were selected. LV diastolic function was estimated using both traditional and tissue-Doppler imaging. Peak early and late trans-mitral diastolic flow velocities (E, A) and early diastolic mitral annulus velocity (E′) were utilized to calculate E/A and E/E′. The individuals were categorised into three groups: overweight (BMI 25.0–29.9), normal weight (BMI 25.0), and obesity (BMI 30).

Results: BMI was independently linked with greater E, A, and E/E′, an indication of LV filling pressure, in multivariate models (all $p<0.01$). Overweight and obese participants demonstrated lower E′ (both $p<0.01$) and greater E/E′ (both $p<0.01$) than respondents with normal weight. Obese participants had a lower E/A than normal weight respondents ($p<0.01$). When contrasted with normal weight people, the perils of diastolic dysfunction were considerably greater among overweight and obese people.

Conclusion: Irrespective of LV mass or other risk variables, having an elevated BMI was connected to poor LV diastolic performance. Both overweight and obese patients have higher chances of LVDD, which may explain some of the heart failure associated risks with both conditions.

INTRODUCTION

Obesity is becoming more common across the world, and it is a serious public health concern due to its links to cardiovascular diseases, mortality and morbidity. Obesity is a crucial causality of incident heart failure among the general populace, and there exists evidence that being overweight heightens the chances of heart failure to a level that is halfway between obese and lean people. In 2016, 39 percent of adults over the world were overweight, and 13% were obese (Gomez et al., 2022; Kosmala et al., 2008; Loehr et al., 2009). Obesity is linked to elevated risks of cardiovascular illness and death. Obesity is a strong predictor of Diastolic Dysfunction (DD), heart failure, and LV hypertrophy (LVH), although the relationship with systolic function is more complicated (Rozenbaum et al., 2019; Nagueh et al., 2009).

Left Ventricular systolic dysfunction, Left Ventricular diastolic dysfunction, as well as Left Ventricular hypertrophy, can be encountered amongst people with T2 Diabetes Mellitus. LVDD is a syndrome wherein the filling properties of the left ventricle are compromised. In community settings, it is a determinant of subsequent heart failure development (Ng et al., 2018; Norton et al., 2009). Henceforth, LVDD might be a potential pathophysiological link between increased body weight and the furtherance of heart failure (Larsen et al., 2018; Russo et al., 2010). Multifactorial pathophysiological pathways are likely mediators of diabetic cardiomyopathy with LVSD and LVDD suggesting the start of congestive heart failure, regardless of coronary artery disease or hypertension (Sharp et al., 2010; Shemirani et al., 2022). Although the extent to which obesity correlates to LV systolic function impairment in T2DM patients is unknown, it is believed to be linked to reduced systolic function as determined by both LVEF and deformation imaging.

The goals of our study are to:

1. investigate the link between body size and LV diastolic function as determined by transthoracic echocardiography in a community-based cohort of people over 50
2. assess the impact of associated risk factors on this relationship, and
3. look into the effect of different degrees of increased body size on the risk of LVDD.

METHODOLOGY

SAMPLE

For this study, 495 subjects were selected. The research participants were categorised into three groups: normal weight (BMI 25.0), overweight (BMI 25.0–29.9), and obese (BMI 30).

TESTS CONDUCTED

Traditional and tissue-Doppler imaging was used for investigating LVDD. E/A and E/E′ were estimated using peak early and late trans-mitral diastolic flow velocities (E, A) and early diastolic mitral annulus velocity (E′). Medical history and ongoing medical therapy data were collected from patient records as part of the inquiry.

ECHOCARDIOGRAPHIC ASSESSMENT

During enrolment, each participant had an echocardiographic assessment. Physical parameters of the patients were taken into account. A fully automated oscillometric instrument was utilised to monitor blood pressure in a supine posture at rest. A validated algorithm was instrumentalised to compute LV mass, which was indexed for both BSA and height. 2x posterior wall thickness/end-diastolic diameter was utilised to compute LV Relative Wall Thickness (RWT). The biplane modified Simpson's rule was utilised to compute the LV ejection fraction.

STATISTICAL ANALYSIS

The clinical intent of this research was to see how obesity and overweight influence the left ventricular diastolic function. The independent correlation of BMI with the parameters of diastolic function was assessed using multiple linear regressions. Both unstandardized (B) and standard (β) coefficient estimates and standard errors were presented for the predictors and outcome variables, with associated standard deviations. After correcting for confounders, ANCOVA was utilised for analysing differences in diastolic function parameters across groups, and 95 percent confidence intervals were calculated. A 2-tailed $p<0.05$ was deemed significant in all statistical analyses. SPSS software version 17.0 was used for statistical analysis.

RESULTS

The independent connection of BMI and cardiovascular risk factors with LV mass, which demonstrated larger LV mass when indexed by height was investigated upon. An elevated occurrence of cardiovascular risk factors, which are deemed responsible for an increase in LV mass and decline in diastolic performance was witnessed. The best independent predictor of increased LV mass/height was a higher BMI (β = 0.28, p<0.001).

	Peak A			**Peak E**			**E/A ratio**			**Peak E'**			**E/E'**		
Model 1	B	β	P-value	B	β	P-value	B	β	P-value	B	β	P-value	B	β	P-value
BMI	0.92	0.21	<0.001	0.41	0.10	<0.001	-0.005	-0.08	0.005	-0.04	-0.13	<0.001	0.13	0.18	<0.001
Age	0.73	0.32	<0.001	-0.02	-0.01	0.58	-0.01	-0.18	<0.001	-0.08	-0.44	<0.001	0.11	0.32	<0.001
Model 2	B	β	P-value	B	β	P-value	B	β	P-value	B	β	P-value	B	β	P-value
BMI	0.56	0.12	<0.001	0.32	0.08	0.006	-0.004	-0.-7	0.01	-0.01	-0.02	0.29	0.04	0.07	0.01
Age	0.67	0.29	<0.001	-0.04	-0.02	0.38	-0.01	-0.18	<0.001	-0.06	-0.37	<0.001	0.09	0.25	<0.001
Male	-5.53	-0.12	<0.001	-4.0	-0.10	0.001	-	-	-	-	-	-	-0.71	-0.09	0.001
LV mass index	0.10	0.07	0.01	-	-	-	-	-	-	-0.02	-0.23	<0.001	0.04	0.19	<0.001
Heart rate	0.36	0.19	<0.001	-0.21	-0.13	<0.001	-0.005	-0.19	<0.001	-	-	-	-0.02	-0.08	0.003
Hypertension	-	-	-	-	-	-	-	-	-	-0.60	-0.15	<0.001	0.60	0.07	0.01
Diabetes	5.62	0.11	<0.001	3.18	0.07	0.01	-	-	-	-	-	-	0.70	0.08	0.002

Body Mass Index was connected to lower E/A (β = 0.12, p = 0.03), greater A (β = 0.14, p = 0.02), and lower E′ (β = 0.12, p = 0.03) in male individuals. BMI was essential to greater E (β = 0.15, p<0.001), higher A (β = 0.17, p<0.001), and higher E/E′ (β = 0.12, p = 0.004) among the women participants. Regardless of age, LV mass index, or heart rate, the connection between BMI and E/E′ ratio was remained significant (β = 0.13, p = 0.04).

Comparing the data collected from the three groups, it was established that peak E was substantially greater in obese participants than in controls (p<0.01). When contrasted with participants of normal weight, Peak A was considerably greater in the obese and the overweight (both p<0.01) categories. Among obese individuals, E/A was considerably lower

than the participants had normal weight (p<0.01). Peak E′ was substantially lower in overweight and obese people than in cases of patients having normal weight (both p<0.01). When compared to normal weight people, E/E′ was considerably greater in overweight and obese people (both p<0.01).

	Adjusted for sex, age, LV mass index, heart rate, diabetes mellitus and hypertension			**Adjusted for sex and age**		
	Odds ratio	95% CI	P-value	Odds ratio	95% CI	P-value
Normal weight	Reference	-	-	Reference	-	-
Overweight	1.52	1.04-2.22	0.03	1.66	1.15-2.40	0.006
Obese	1.60	1.06-2.41	0.02	1.92	1.30-0.001	0.001

The total incidence of LVDD was 53.5 percent (n = 508). DD was witnessed in 50.8 percent of healthy people, 54.2 percent of overweight people, and 57.1 percent of obese people (p = 0.34). The risk of LVDD amongst participants with obesity and overweight was estimated using a multivariate logistic model. Both the obese (OR: 1.60, 95 percent CI: 1.06–2.41, p = 0.02) and overweight (OR: 1.52, 95 percent CI: 1.04 – 2.22) groups exhibited a substantially larger risk of DD than the respondents with normal weight after controlling for variables. BMI as a continuous variable was similarly linked to heightened risks of DD (adjusted OR: 1.04, 95 percent CI: 1.01–1.07, p = 0.04) and pseudo-normalized diastolic pattern (adjusted OR: 1.05, 95 percent CI: 1.01–1.08, p = 0.02). On LVDD, there was no significant interaction between sex and BMI (p-value for the interaction = 0.61).

In this multivariate linear regression analysis, a link between waist circumference with peak A (β = 0.11, p<0.001) and E/A (β = 0.06, p = 0.05) was shown. In the multivariate logistic model, a higher waist circumference (defined as ≥88 cm in women and ≥102 cm in men) was linked with a substantial enhancement in the risk of DD (adjusted OR: 1.69, 95 percent CI 1.22–2.35, p = 0.002). Waist circumference was likewise linked to a higher risk of DD as a continuous variable (adjusted OR for each unit increase: 1.03, 95 percent CI 1.01 – 1.04, p = 0.01). Effects of sex and waist size on LVDD was not found to be crucial (p = 0.88).

DISCUSSION

As revealed by the findings, the relationship between CVD risk indicators such LV hypertrophy, diabetes, and high blood pressure. It was also discovered that being obese is linked to lower possibilities of LVDD, which is similar to what is witnessed among obese persons. Between overweight and obese persons, there were no significant differences in main diastolic function parameters. Madsen et al. (2021), Soga et al. (2018), and Canada et al. (2019) discovered a relationship between diastolic performance and obesity in small samples of young women. A similar relationship had been observed by Shim et al. (2018) in respondents with cardiovascular risk indicators, and by Willens et al. (2004) in obese people. Obese people's LV end-diastolic pressure was depicted as significantly higher than respondents with a BMI lesser than 25 by Kagiyama et al. (2020), Rayner et al. (2018), and Turkbey et al. (2010), and Whitlock et al. (2009). These studies also claimed that not BMI but circumference measurements were connected to reduced ventricular filling, which is consistent with our findings.

CONCLUSION

In conclusion, LVDD is a significant pathophysiological link between being overweight or obese and the risk of heart failure. While past research has focused on obesity, our findings reveal that overweight adults can also have subclinical signs of LV diastolic function

impairment and that these irregularities are independent of other risk variables. Therapeutic approaches aimed at reaching a healthy body weight resulted in improvements in LV systolic and diastolic function, indicating that they might help prevent or delay the onset of heart failure in the future, a hypothesis that needs to be investigated further.

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