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# Assessment Of Left Ventricular Structures And Functions In Patients With Metabolic Syndrome By Transthoracic Echo Cardiography And Tissue Doppler Study

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# **Abstract**

**Background**: MetS is a cluster of cardiovascular risk factors related with rose mortality and morbidity. Its impact on heart function and structure, particularly left ventricular (LV) performance, remains incompletely defined.

**Objective**: To assess LV functional and structural changes in cases with MetS using transthoracic echocardiography and tissue Doppler imaging (TDI).

Methods: This research involved 60 cases. All of them underwent echocardiographic assessment at Echocardiography Unit, Al-Azhar University Hospital (Assiut), Those cases have been categorized into three groups: Control Group(A): twenty persons with absent (0) any criteria of metabolic syndrome. Study Group(B): 20 persons with pre-metabolic syndrome (1-2 criteria). Study Group(C): 20 persons with metabolic syndrome (≥3 criteria).

**Results**: left ventricular systolic function by ejection fraction has been preserved across all groups. However, MPI by PWD and TDI was significantly greater in MetS patients (0.48±0.08 and 0.55±0.12, correspondingly) in comparison with controls (0.41±0.02 and 0.47±0.02; p<0.001). S' velocity was reduced in MetS (9.3±2.08 cm/s) compared with controls (12.4±1.46 cm/s; p<0.0001). Diastolic function parameters were impaired in MetS, with lower E/A ratio (0.83±0.20 vs. 1.35±0.13, p<0.0001), higher E/E' (13.29±3.92 vs. 5.32±1.09, p<0.0001), and prolonged IVRT (100.55±24.91 ms vs. 74.65±4.91 ms, p<0.0001). LVMI/Ht^2.7 and RWT were significantly elevated in MetS than controls, indicating hypertrophy and concentric remodeling.

**Conclusion**: MetS is related to subclinical LV diastolic & systolic dysfunction, increased LV mass, and concentric remodeling despite preserved ejection fraction. Echocardiography with TDI provides sensitive detection of early myocardial impairment in these cases.

**Keywords**: Left ventricular hypertrophy; Metabolic syndrome; Diastolic dysfunction; Echocardiography

# 1. INTRODUCTION

Metabolic syndrome (MetS) is a complex risk factor resulting from IR related with aberrant function and deposition of adipose tissue. It is a risk factor for CHD, fatty liver illness, diabetes, and many malignancies.

This syndrome may appear clinically as hyperglycemia, hypertriglyceridemia, hypertension, diminished high-density lipoprotein cholesterol (HDL-C), and abdominal obesity (1).

The metabolic syndrome denotes a convergence of cardiovascular risk factors impacting about twenty-two percent of the adult population in industrialized nations & over forty percent in individuals fifty years of age and above (2). According to the present guidelines, updated in 2005 by the American Heart Association (AHA), and the National Heart, Lung & Blood Institute (NHLBI), MetS is identified when a case exhibits at least 3 of the following 5 criteria: fasting glucose not less than hundred milligram per deciliter or having pharmacological management for hyperglycemia; BP not less than 130/85 millimeters of mercury or having pharmacological management for hypertension; triglycerides above or equal to 150 milligram per deciliter or having pharmacological management for hypertriglyceridemia; HDL-C below forty milligram per deciliter in men or below fifty milligram per deciliter in females or undergoing pharmacological management for low HDL-C; and waist circumference above or equal to 102 centimeters (forty inches) in men or ≥88 centimeters (thirty-five inches) in women. For Asian Americans, the threshold values are above or equal to ninety centimeters (thirty-five inches) for men and ≥ eighty centimeters (32 inches) for women. The International Diabetes Federation (IDF) guidelines permit the utilization of a body mass index (BMI) greater than thirty kilograms per square meter as a substitute for the waist circumference requirement (3). Extensive data indicate that cases who fulfill these diagnostic criteria face an elevated risk of severe clinical outcomes, including the onset of DM and coronary heart disease (Hanley et al., 2005). Aggregated data from 37 research encompassing over 170,000 participants indicate that metabolic syndrome increases the possibility of CHD (4). It additionally elevates the possibility of stroke, hepatic steatosis, and malignancy

LVH elevates the risk of cardiovascular death and morbidity, encompassing the onset of diastolic and systolic dysfunction, and the advancement to cardiac failure. The incremental accumulation of MetS risk factors, including obesity, diabetes, and/or dyslipidemia, correlates with augmented left ventricular mass irrespective of hypertension; however, the influence of MetS and its individual components on heart function and structure remains inadequately defined (7).

This goal of this research is to estimate the impact of MetS and its distinct components on left ventricular architecture and functions by transthoracic echocardiography and tissue Doppler imaging.

# 2. PATIENTS AND METHODS

Selected 60 persons were included. All of them underwent echocardiographic assessment at Echocardiography Unit, Al-Azhar University Hospital (Assiut), Those cases have been classified into three groups: Control Group (A): twenty persons with absent (0) any criteria of metabolic syndrome. Study Group (B): 20 persons with pre-metabolic syndrome (1-2 criteria). Study Group (C): 20 persons with metabolic syndrome (≥3 criteria).

# **Inclusion Criteria:**

MetS is identified when a case present with at least 3 of the following 5 conditions. The first criterion is fasting glucose above or equal to 100 milligrams per deciliter, or the patient may be receiving drug therapy for hyperglycemia. The second criterion is blood pressure above or equal to 130/85 millimeters of mercury, or the patient may be receiving drug therapy for hypertension. The third criterion is triglycerides  $\geq 150$  mg/dL, or the patient may be receiving drug therapy for hypertriglyceridemia.

The fourth criterion is HDL-C below forty milligrams per deciliter in men or < fifty milligrams per deciliter in females, or the case may be receiving drug treatment for reduced HDL-C. The fifth criterion is waist circumference above or equal to 102 centimeters (forty inches) in males or  $\ge$  eighty-eight centimeters (35 inches) in females. For Asian American populations, the threshold is  $\ge$  ninety centimeters (thirty-five inches) in men or  $\ge$  eighty centimeters (thirty-two inches) in women. According to the IDF criteria, a BMI >30 kg/m² may be used in lieu of the waist circumference criterion (8).

**Exclusion Criteria:** History or results of cardiovascular disorder include: -cardiac failure symptoms or systolic dysfunction (LVEF below fifty-five percent).

- -Pregnancy or lactating.
- -Significant valvular cardiac illness (i.e. higher than mild valvular stenosis or insufficiency).
- -Coronary artery disease.
- -Hypertrophic cardiomyopathy.
- Major systemic illness (e.g. advanced liver disease and renal disease).

# Methods

All patients underwent the following: history taking and clinical assessment

A complete clinical examination has been carried out on every case, with special emphasis on the following information: blood pressure, rhythm and pulse rate, lower and upper limb examination, neck and head examination, and chest and heart examination. The latter involved assessment of heart sounds, additional heart sounds, and murmurs, while the back was examined for evidence of lung congestion.

Resting 12-lead electrocardiography was performed for all patients. Echocardiographic evaluation was also carried out using a Vivid 7 phased array system. A comprehensive transthoracic echocardiographic assessment has been done, involving both conventional echocardiography & tissue Doppler echocardiography. All echocardiographic assessments have been conducted following twenty to thirty minutes of rest, with the case in a state of calm respiration in the partial left lateral decubitus position, utilizing a two-to-four-megahertz transducer, and have been supplemented by the recording of resting electrocardiography. All measures have been attained online, and echocardiographic variables have been assessed in line with the American Society of Echocardiography standards, with each variable derived from the average of 3 consecutive cardiac cycles.

The assessment of LV systolic function has been conducted utilizing both conventional echocardiography and p-wave Doppler methods. The ejection fraction (EF) has been determined with the M-mode technique, which assesses the left ventricular dimension from the anterior boundary of the septal endocardial echo to the anterior boundary of the posterior wall endocardium. Tiecholz's equation has been utilized as follows:  $EF(\%) = (LVIDd^3 - LVISd^3) / LVIDd^3 \times 100$  (9). Moreover, the biplane method involved manual delineation of the endocardial boundary of the left ventricle in both the apical four-chamber and apical two-chamber perspectives, identifying LVEDV and LVESV in each view to compute EF (10).

The pulsed-wave myocardial performance index (PW-MPI) has been determined as well. Doppler time intervals have been determined from mitral input and LV outflow velocity-time intervals. The interval 'A', defined as the duration from the cessation to the commencement of mitral inflow, equaled the aggregate of ejection time (ET), isovolumetric contraction time (IVCT), and isovolumetric relaxation time (IVRT). LV-ET 'B' represented the period of the ejection phase throughout systole. The sum of IVCT and IVRT has been determined by subtracting 'B' from 'A'. The MPI has been subsequently computed as (A - B)/B (11). The standard MPI is roughly 0.4, but elevated readings, generally between 0.6 and over 1.0, signify ventricular dysfunction (12).

TDI has been conducted as well. The TDI function of the echocardiography equipment was activated to record mitral annular velocities via p-wave TDI. A variable incidence phased-array transducer (two to four megahertz) has been utilized with low filter settings (fifty Hertz) and gains calibrated to ideal values to achieve high-quality velocity recordings. Longitudinal mitral annular velocities have been measured from the anterior, lateral, septal, and inferior left ventricular locations utilizing the apical four-chamber view. The measurements comprised the positive peak systolic velocity (S wave), indicative of longitudinal contraction of the left ventricle, and two negative E-wave: one through the early diastolic phase (E') and the other throughout the late diastolic phase (A') (12).

The average S wave was determined by measuring the mitral annular positive peak S-wave from the lateral, septal, inferior, and anterior left ventricular locations, with the mean of these four sites applied to evaluate global systolic function. The standard range for the average S wave is deemed to be between 6.8 and 12.2 centimeters per second (14). The MPI by TDI has been evaluated. TDI velocity time intervals have been determined at the lateral and septal segments of the mitral annulus. The TDI-IVCT was quantified from the termination of the A' wave to the initiation of the S' wave, whereas the TDI-ET has been determined from the commencement to the conclusion of the S' wave. The Tissue Doppler

imaging IVRT has been assessed from the termination of the S wave to the initiation of the E' wave. The MPI by tissue Doppler imaging was subsequently computed as (IVCT + IVRT) / ET (15). As previously mentioned, MPI values between 0.6 and greater than 1.0 signify ventricular dysfunction.

# Assessment of LV diastolic function has been done as the following:

**Transmitral LV inflow measurement:** The Doppler beam has been aligned in the flow direction, and a one- to two-millimeter sample volume was positioned among the tips of the mitral leaflets throughout diastole, in the apical four-chamber view, to detect: Transmitral early velocity wave (E wave)

Trans mitral velocity wave (A wave): Deceleration Time (DT); quantified along the descending gradient of mitral flow A wave. Deceleration Time ranging from 150 to 200 milliseconds is deemed normal (12). E/A ratio: normally above 0.8 (16).

**Isovolumic Relaxation Time:** Assessed using TDI-PWD at the left ventricular basal lateral wall from the conclusion of the S-wave to the initiation of the early E` wave. The standard range for IVRT is between sixty and one hundred milliseconds (12).

**MV** E/E` ratio: Utilized TDI-PWD to measure the early E` wave of mitral inflow, followed by the calculation of the E/E` ratio. E/E' pertains to LA pressure. E/E' below eight indicates normal left atrial pressure, while E/E' above fifteen indicates excessive left atrial pressure. (9).

Assessment of LV mass index and RWT: The relative wall thickness (RWT) has been computed utilizing the formula: (two × posterior wall thickness) / LV interior diameter. The left ventricular mass (LVM) has been determined utilizing the M-mode-derived cubed approach & indexed to height^2.7(Ht^2.7) to account for body habitus; left ventricular hypertrophy has been defined as LVM/Ht^2.7  $\geq$  fifty-one grams per meter^2.7 for men and  $\geq$  49.5 grams per meter^2.7 for women (17).

# 3. RESULTS

Comparative analysis between the three groups regarding age demonstrated that the mean age of group A was 35.15 years  $\pm 11.18$  whereas the mean age of group B was 44.55 years  $\pm 10.93$ , and in group C was 46.35 years  $\pm 7.37$ . A statistically significant variance has been observed among the three groups as regard age (P equal to 0.0016).

Comparative analysis between the three groups according to sex revealed that males were 10(50%) in group A, and 8(40%) in groups B and C, while females were 10(50%) in group A and 12(60%) in groups B and C. A statistically insignificant difference has been observed between the three groups according to sex. (P-value 1.0).

<b>Table 1.</b> Comparative between the three groups according to MPI by	PW doppler.
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Crown	MPI by PWD			Develve
Group	Mean	±	SD	P-value
Group A	0.41	±	0.02	
Group B	0.44	±	0.03	0.0010
Group C	0.48	±	0.08	

In group A: MPI by PWD (mean  $\pm$  SD) was 0.41 $\pm$  0.02. In group B: MPI by PWD (mean  $\pm$  SD) was 0.44  $\pm$  0.03. In group C: MPI by PWD (mean  $\pm$  SD) was 0.48 $\pm$  0.08. MPI by PWD variance between the three groups was extremely significant (P value = 0.0010).

**Table 2.** comparative analysis between the three groups regarding MPI by TDI.

Cwann	MPI by TD	I	<u> </u>	D scales
Group	Mean	±	SD	P-value
Group A	0.47	±	0.02	
Group B	0.44	±	0.03	< 0.0001
Group C	0.55	±	0.12	

In group A: MPI by TDI (mean  $\pm$  SD) was  $0.47\pm0.02$ . In group B: MPI by TDI (mean  $\pm$  SD) was  $0.44\pm0.03$ . In group C: MPI by TDI (mean  $\pm$  SD) was  $0.55\pm0.12$ . MPI by TDI variance between the three groups was extremely significant (P below 0.0001).

**Table 3.** comparative between the three groups regarding S wave by TDI.

Cwann	S wave by T	DI	<u> </u>	P-value
Group	Mean	±	SD	r-value
Group A	12.4	±	1.46	
Group B	10.1	±	1.23	< 0.0001
Group C	9.3	±	2.08	

In group A: S wave by TDI (mean  $\pm$  SD) was 12.4 $\pm$  1.46. In group B: S wave by TDI (mean  $\pm$  SD) was 10.1  $\pm$  1.23. In group C: S wave by TDI (mean  $\pm$  SD) was 9.3  $\pm$  2.08. S wave variance between the three groups was extremely significant (P below 0.0001).

**Table 4.** Comparative analysis between the 3 groups as regard MV E/A

Cusum	E/A			D scales
Group	Mean	±	SD	P-value
Group A	1.35	±	0.13	
Group B	0.95	±	0.22	< 0.0001
Group C	0.83	±	0.20	

Comparison between the three groups according to MV E/A: When comparing E/A ratio between the three group we found: In group A: MV E/A (mean  $\pm$  SD) was  $1.35\pm0.13$ . In group B: MV E/A (mean  $\pm$  SD) was  $0.95\pm0.22$  In group C: MV E/A (mean  $\pm$  SD) was  $0.83\pm0.20$ 

**Table 5.** Comparison between the three groups regarding MV E/E'

Cwann	E/E'			
Group	Mean	±	SD	P-value
Group A	5.32	±	1.09	

Group B	10.11	±	4.71	< 0.0001
Group C	13.29	±	3.92	

Comparison between the three groups according to MV E/E': MV E/E' ratio revealed the following: In group A: MV E/E' (mean  $\pm$  SD) was 5.32 $\pm$  1.09. In group B: MV E/E' (mean  $\pm$  SD) was 10.11  $\pm$  4.72. In group C: MV E/E' (mean  $\pm$  SD) was 13.29  $\pm$  3.92. E/ E' variance among the two groups was extremely significant (P below 0.0001).

**Table 6.** Comparative analysis between the three groups as regard IVRT

	IVRT		<u> </u>	D value
Group	Mean	±	SD	P-value
Group A	74.65	±	4.91	
Group B	90.45	±	25.78	< 0.0001
Group C	100.55	±	24.91	

In group A: IVRT (mean  $\pm$  SD) was 74.65 $\pm$ 4.91ms. In group B: IVRT (mean  $\pm$  SD) was 90.45 $\pm$  25.78ms. In group C: IVRT (mean  $\pm$  SD) was 100.55 $\pm$ 24.91ms. IVRT variance between the three groups was extremely statistically significant (P below 0.0001).

**Table 7.** Comparison between the three groups as regard LVMI to Ht^2.7

LVMI to Ht^2.7		^2.7		Decelor
Group	Mean	±	SD	P-value
Group A	24.45	±	9.28	
Group B	40.91	±	8.40	< 0.0001
Group C	41.78	±	10.57	

Comparison between the three groups according to LV mass indexed to Ht^2.7: When LV mass indexed to Ht^2.7: In group A: LVMI to Ht^2.7 (mean  $\pm$  SD) was 24.45 $\pm$  9.28 g/m^2.7. In group B: LVMI to Ht^2.7 (mean  $\pm$  SD) was 40.91  $\pm$  8.40 g/m^2.7. In group C: LVMI to Ht^2.7 (mean  $\pm$  SD) was 41.78  $\pm$  10.57 g/m^2.7.

**Table 8.** Comparison between the three groups as regard RWT

	LV RWT		<u> </u>	P-value
Group	Mean	±	SD	r-value
Group A	0.36	±	0.03	
Group B	0.38	±	0.08	< 0.0001
Group C	0.48	±	0.09	

Comparison between the three groups according to LV Relative Wall Thickness. Comparison of LV RWT revealed: In group A: LV RWT (mean  $\pm$  SD) was 0.36  $\pm$ 0.03. In group B: LV RWT (mean  $\pm$  SD) was 0.38 $\pm$ 0.08. In group C: LV RWT (mean  $\pm$  SD) was 0.48 $\pm$ 0.09. LV RWT variance between the three groups was extremely statistically significant (P below 0.0001).

# 4. DISCUSSION

MetS is a multifaceted condition with significant socioeconomic implications, seen as a global epidemic. MetS is characterized via a constellation of interrelated factors that significantly elevate the risk of coronary heart disease (CHD), various types of cardiovascular atherosclerotic diseases (CVD), & type 2 diabetes mellitus (DMT2). The primary components include dyslipidemia (raised triglycerides and reduced high-density lipoproteins (HDL)), impaired glucose homeostasis, and increased arterial blood pressure (BP), with abdominal obesity and/or IR emerging as central manifestations of the syndrome. Lately, other illnesses like chronic prothrombotic & proinflammatory states, non-alcoholic fatty hepatic illness, and sleep apnea have been incorporated into the syndrome, complicating its description further. Despite the numerous components and clinical ramifications of MetS, a broadly agreed pathogenic mechanism and precisely defined diagnostic criteria remain elusive. Moreover, there remains contention on whether this entity signifies a distinct syndrome or serves as a proxy for a confluence of risk factors that predispose the individual to specific vulnerabilities. A significant emerging characteristic of Met S is its rising frequency in young adulthood and childhood, together with the potential future implications for the global health burden it may impose (18).

The measurement of cardiac chamber dimensions and functionality is fundamental to cardiac imaging, with echocardiography being the predominant noninvasive technique due to its distinctive capacity to deliver real-time visuals of the pulsating cardiac, along with its accessibility and portability (19). Thus, it serves as an effective instrument for assessing left ventricular function and structure in metabolic syndrome.

Historically, cardiac failure has predominantly been viewed through the lens of systolic failure characterized by diminished EF; however, there is an increasing recognition that diastolic dysfunction, which has been comparatively overlooked, may also significantly contribute to the condition. Approximately fifty percent of patients exhibiting symptoms and signs of cardiac failure possess a normal left ventricular EF, yet demonstrate an anomaly in the diastolic characteristics of the LV, with a prevalent history of hypertension among these cases (20). LV diastolic impairment typically precedes systolic dysfunction (21). marked by atypical relaxation throughout the initial phases (22).

Diastolic dysfunction can be evaluated using TDI, which facilitates the measurement of cardiac diastolic and systolic velocities in the myocardium (23).

# LV Systolic Function;

All cases involved in this research illustrate normal left ventricular systolic function as evaluated by EF-M mode method and ejection fraction- Simpson method but when using MPI (Tei index) either by PWD method or TDI method there was statically significant different between three groups with higher values in patients with metabolic syndrome.

As regard MPI by PWD (mean  $\pm$  SD): In group(A) was  $0.41\pm0.02$  and in group (B) was  $0.44\pm0.03$  while in group (C) MPI by PWD was  $0.48\pm0.08$  (P value =0.0010). Results for MPI by TDI were  $0.47\pm0.02$  for group (A),  $0.44\pm0.0$  for group (B) and  $0.55\pm0.12$  for group (C) (P value <0.0001).

This came in agreement with Sreenivasa, et al., 2014 who observed that Met S is a potent predictor of subclinical myocardial dysfunction in subjects free of clinically apparent cardiac illness. The study included 50 patients with MetS and 30 in control group who were assessed by echocardiography to calculate MPI (Tei index). The mean LVMPI using traditional PWD imaging was  $0.63 \pm 0.08$  in cases and  $0.48 \pm 0.05$  in controls. The mean LVMPI measured via traditional p-wave Doppler technique was considerably elevated in cases than controls (p below 0.0001) (24).

As regard S wave (Vs global) by TDI: In group (A) S wave by TDI (mean  $\pm$  SD) was 12.4 $\pm$  1.46 and in group (B) was 10.1  $\pm$  1.23 and in group (C) S wave by TDI was 9.3  $\pm$  2.08.

This aligns partially with Lisa et al., 2007. Although the LVEF was comparable between the 3 groups, the TDI-derived septal S wave, but not global S wave, (determines of longitudinal systolic myocardial contractility) has been significantly decreased in the Met S group in comparison with the normal group (P equal to 0.006) (25).

# **Left Ventricular Diastolic Function**;

A statically significant variance between the 3 groups as regard diastolic function by different echocardiographic parameters. Persons with MetS were more liable for diastolic dysfunction.

As regard E/A: (mean  $\pm$  SD): In group(A) was 1.35 $\pm$  0.13 and in group (B) was 0.95  $\pm$  0. 22while in group (C) E/A was 0.83  $\pm$  0.20 (P value < 0.0001). When comparing MV E/ E': (mean  $\pm$  SD): In group(A) was 5.32 $\pm$  1.09and in group (B) was 10.11  $\pm$  4.72 while in group (C) MV E/ E' was 13.29  $\pm$  3.92 (P value < 0.0001). As regard IVRT: (mean  $\pm$  SD): In group(A) was 74.65 $\pm$ 4.91 ms and in group (B) was 90.45 $\pm$  25.78 ms while in group (C) IVRT was 100.55 $\pm$ 24.91 ms (P below 0.0001).

The results align with Lisa et al. (2007), which demonstrate that assessments of left ventricular diastolic performance deteriorated progressively from the absence of metabolic syndrome to the pre-MetS and MetS groups, demonstrating a decline in diastolic function with the escalating burden of MetS. This research consisted of 607 subjects, aged twenty-one and older meeting study criteria and divided in three groups normal, metabolic and pre-metabolic. The research's results demonstrated a gradual decline in the E/A ratio from the Absent group to the pre-metabolic syndrome group and finally to the Met-S group, mostly due to an increase in A-wave velocity; the DT and IVRT have been significantly prolonged in the MetS group. The TDI-derived Veseptal and Ve global values decreased significantly in both the MetS and pre-MetS groups compared to & absence group (P-value equal 0.0002 for all). The data indicate a steady deterioration in left ventricular relaxation corresponding to an increase in the number of MetS criteria. The occurrence of left ventricular diastolic dysfunction, as measured by PWD- and TDI-derived indices, varied from seven to nine percent in the Absent group to seventeen to eighteen percent in the Pre-MetS group, and twenty-nine to thirty-five percent in the MetS group. In the Pre-MetS cohort, the odds ratio for identifying LV diastolic dysfunction was 2.6 (CI of 95%: 1.2-5.6, P-value equal 0.01) via PWD and 2.2 (ninety-five percent confidence interval: 1.1–4.5, P equal to 0.03) via TDI; in the MetS cohort, the OR was 5.2 (ninetyfive percent confidence interval: 2.4–11.4, P below 0.0001) and 5.5 (CI of 95%: 2.7–11.3, P below 0.0001), correspondingly (25).

# Left Ventricular Mass and Relative Wall Thickeness:

In this study LV mass has been calculated from this equation: LV mass= 0.8 (1.04 ((LVIDD + PWTD + IVSTD)3- (LVIDD)3)) + 0,6 gram, then indexed to height2.7 (LVM/Ht2.7). It was found that there is increasing in LVM/ Ht2.7 and RWT in MetS and pre-metabolic than normal persons. As regard LVM/Ht2.7 (mean  $\pm$  SD) in group(A)24.45 $\pm$ 9.28, while it was40.91 $\pm$  8.40 in group(B) and 41.78 $\pm$ 10.57 in group(C) P value < 0.0001.

When comparing RWT in the three groups we found extremely statically significant relationship between MetS and increase RWT values, (mean  $\pm$  SD) in group(A)0.36  $\pm$ 0.03, while it was0.38 $\pm$ 0.08 in group(B) and 0.48 $\pm$ 0.09 in group(C) while P value < 0.0001.

This outcome came in agreement with LA Ferrara, et al., 2007. research included 707 subjects; 153 (21.6%) of the study population were found to have MetS. The study found that subjects with MetS had a greater LV mass and, accordingly, a greater occurrence of left ventricular hypertrophy LVH (83/153 subjects with MetS, 54.2% (mean±SD) 41.47±11.3 vs 141/554 subjects without MetS, 25.4%; (mean±SD) 49.07±12.0 (P below 0.001).

With regard to the geometrical pattern of the LV, concentric hypertrophy has been present in 5.0 percent of the non-MetS group and in 8.5 percent of the MetS group, eccentric hypertrophy in 20.4 & 45.7 percent correspondingly.

Furthermore, 8.3 percent of non-MetS group and 4.4 percent of MetS had concentric remodelling. The variances in the occurrence of the geometrical patterns of the LV among subjects with and with no MetS

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were statistically significant, RWT (Mean±SD) was (37.37±7.7) for non-MetS and (39.87±7.7) for MetS group (P<0.001), (26).

In Mahmoud, et al., 2009, who studied 160 persons (aged 46±1 years (mean ±SD), fifty-three percent male) had 2-D echocardiography and TDI and assessment for MetS to test the hypothesis that the heart functional and structural anomalies of the MetS are independent of BMI, although LV mass was higher with MetS (Mean±SD) 164±7 in Met Svs 149±4 in non-MetS (p below 0.05), a statistically insignificant variance has been observed between 2 groups when LV mass has been indexed either to height or height, the LVM/height 2.7 (Mean±SD) was 39±2 in MetS vs 37±1 in non-MetS (p non-significant), this may be due to age, sex and BMI was similar in the two groups (27).

These results were not only restricted to adults but also in children. Alkholy et al. (2016) discovered that left ventricular mass index in obese kids was considerably elevated in comparison with the control group, with even greater significance shown in obese children with metabolic syndrome than their non-MetS counterparts. A strong positive association has been observed between LVMI and indices of MetS, as well as with indicators of IR. This research involved eighty-two obese youngsters. Their average age was 10.2  $\pm$  2.8 years; the participants comprised 25 obese children with MetS, seventy-five obese kids with no MetS, and forty healthy age- and sex-matched kids have been utilized as a control group. All children underwent clinical evaluation and got an echocardiographic examination (Doppler, 2-D, M-mode, and tissue Doppler echocardiography) along with laboratory assessment. Results. The left ventricular mass index (LVMI) has been elevated in the obese group relative to the control group (p below 0.001). A notable positive connection existed between LVMI and serum leptin levels relative to WC, BMI, fasting insulin, fasting glucose, homeostatic model assessment for IR, triglycerides, and LDL among all obese kids, particularly within the MetS group. A significant negative association has been observed among both left ventricular mass index and serum leptin levels in respect to HDL (28).

Hypertension is identified as a prevalent risk factor for the development of left ventricular hypertrophy (LVH) and increased left ventricular mass. Al Naggar and Al-Daydamony (2015) examined the impact of metabolic syndrome components, excluding hypertension, on left ventricular mass index (LVMI). Their study revealed that even in the absence of hypertension, patients with MetS exhibited significantly greater left ventricular wall thickness, elevated LV mass and mass index, and an elevated frequency of LVH compared to control subjects. Their research comprised fifty MetS cases without hypertension and fifty healthy individuals. Unlike our research, there was insignificant variance among both groups concerning certain indices of diastolic function, like the E/A ratio (29).

The research conducted by Wang et al. in 2015 involved 1,733 cases with metabolic syndrome and 2,373 cases with hypertension without MetS. The study revealed that left ventricular mass and left ventricular mass index have been elevated in the Metabolic Syndrome group than the non-Metabolic Syndrome group. The left ventricular mass index, left ventricular mass, interventricular septum, and posterior wall have been elevated with the increased prevalence of metabolic syndrome illnesses. MetS correlated with an elevated risk of LVH (unadjusted OR 1.38; ninety-five percent confidence interval 1.21-1.57); following adjusting for age, sex, and BP, the adjusted OR was 1.39 (ninety-five percent confidence CI 1.22-1.59). MetS has been correlated with an elevated risk of eccentric hypertrophy in both female and male cases. MetS has been only related to an elevated risk of concentric hypertrophy in female cases, but MetS wasn't correlated with concentric remodeling (30).

It can now be stated that MetS may result in both diastolic dysfunction and left ventricular hypertrophy. Is diastolic dysfunction dependent on left ventricular hypertrophy or not? Ayalon et al. (2014) aimed to investigate whether preclinical left ventricular diastolic dysfunction can manifest independently of left ventricular hypertrophy in metabolic syndrome. Ninety consecutive volunteers with Metabolic Syndrome (MetS) and no cardiovascular illness have been recruited (mean age 46 years, 78 percent female), along with twenty-six controls (no risk factors for MetS; mean age forty-three years, sixty-five percent female). Participants received echocardiography utilizing TDI. In age- and sex-adjusted studies, MetS correlated with increased left atrial (LA) diameter, elevated LV mass, diminished E/A ratio, and reduced mean e' (P below 0.001 for all). These relationships remained significant following additional adjustments for BP, antihypertensive medication usage, and BMI. Upon controlling for left ventricular mass, mitral stenosis

remained independently correlated with increased left atrial diameter, diminished E/A ratio, and reduced mean e' (P-value not more than 0.01 for all). In conclusion, multiple sclerosis was linked to preclinical left ventricular diastolic dysfunction, irrespective of left ventricular mass, as indicated by increased left atrial diameter, diminished E/A ratio, and reduced mean e'. This indicates that MetS may result in diastolic dysfunction through processes that are not reliant on hypertrophy. Variations in diastolic function were more evident at younger ages (31).

# 5. CONCLUSION

Individuals with metabolic syndrome (MetS) and preserved LV ejection fraction often exhibit subclinical abnormalities in both diastolic and, to a lesser extent, systolic function. MetS is also related with increased left ventricle mass, higher occurrence of LV hypertrophy, and greater relative wall thickness. The degree of left ventricle dysfunction and hypertrophy relates with the number of MetS criteria present. Echocardiographic methods differ in diagnostic accuracy, with MPI by TDI proving superior to MPI by PWD for early detection of systolic dysfunction, while MV E/E′ and IVRT are more reliable for evaluating diastolic dysfunction.

#### REFERENCES

- 1. Olufadi R, Byrne CD. Clinical and laboratory diagnosis of the metabolic syndrome. J Clin Pathol. 2008;61(6):697-706.
- 2. Lakka HM, Laaksonen DE, Lakka TA, Niskanen LK, Kumpusalo E, et al. The metabolic syndrome and total and cardiovascular disease mortality in middle-aged men. JAMA. 2002;288(21):2709-16.
- 3. Grundy SM, Cleeman JI, Daniels SR, Donato KA, Eckel RH, et al. Diagnosis and management of the metabolic syndrome: an American Heart Association/National Heart, Lung, and Blood Institute Scientific Statement. Circulation. 2005;112(17):2735-52.
- 4. Gami AS, Witt BJ, Howard DE, Erwin PJ, Gami LA, et al. Metabolic syndrome and risk of incident cardiovascular events and death: a systematic review and meta-analysis of longitudinal studies. J Am Coll Cardiol. 2007;49(4):403-14.
- 5. Giovannucci E. Metabolic syndrome, hyperinsulinemia, and colon cancer: a review. Am J Clin Nutr. 2007;86(3 Suppl):s836-42.
- 6. Devereux RB, Koren MJ, de Simone G, Roman MJ, Laragh JH. Left ventricular mass as a measure of preclinical hypertensive disease. Am J Hypertens. 1992;5(3):175-81.
- 7. De Simone G, Palmieri V, Bella JN, Celentano A, Hong Y, Oberman A, et al. Association of left ventricular hypertrophy with metabolic risk factors: The HyperGEN study. J Hypertens. 2002;20(2):323-31.
- 8. Jensen MD, Haymond MW, Rizza RA, Cryer PE, Miles JM. Influence of body fat distribution on free fatty acid metabolism in obesity. J Clin Invest. 1989;83(4):1168-73.
- 9. Leeson P, Augustine D, Mitchell ARJ, Becher H. Echocardiography. In: Oxford Specialist Handbooks in Cardiology. Oxford: Oxford University Press; 2012. p. 251.
- 10. Lang RM, Badano LP, Mor-Avi V, Afilalo J, Armstrong A, et al. Recommendations for cardiac chamber quantification by echocardiography in adults: An update from the American Society of Echocardiography and the European Association of Cardiovascular Imaging. J Am Soc Echocardiogr. 2015;28(1):1-39.
- 11. Lopes L, Joao I, Vinhas H. Evaluation of systolic and systo-diastolic function: the Tei index in acute myocardial infarction treated with acute reperfusion therapy—early and late evaluation. Rev Port Cardiol. 2007;26(7-8):649-56.
- 12. Otto CM. Textbook of Clinical Echocardiography. 5th ed. Philadelphia: Elsevier; 2013. p. 175.
- 13. Tei C, Ling LH, Hodge DO. New index of combined systolic diastolic myocardial performance: a simple and reproducible measure of cardiac function—a study in normals and dilated cardiomyopathy. J Cardiol. 1995;26(6):357-66.
- 14. Yu CM, Sanderson JE, Marwick TH, Oh JK. Tissue Doppler imaging: A new prognosticator of cardiovascular diseases. J Am Coll Cardiol. 2007;49(19):1903-14.

- 15. Armstrong WF, Ryan T, Feigenbaum H. Feigenbaum's Echocardiography. 7th ed. Philadelphia: Wolters Kluwer Health/Lippincott Williams & Wilkins; 2010. p. 143-54.
- 16. Alam M, Wardell J, Andersson E, Samad BA, Nordlander R. Characteristics of mitral and tricuspid annular velocities determined by pulsed wave Doppler tissue imaging in healthy subjects. J Am Soc Echocardiogr. 1999;12(7):618-28.
- 17. De Simone G, Daniels SR, Devereux RB, Meyer RA, Roman MJ, et al. Left ventricular mass and body size in normotensive children and adults: assessment of allometric relations and impact of overweight. J Am Coll Cardiol. 1992;20(5):1251–60.
- 18. Kassi E, Pervanidou P, Kaltsas G, Chrousos G. Metabolic syndrome: definitions and controversies. BMC medicine. 2011 May 5;9(1):48.
- 19. Lang RM, Bierig M, Devereux RB, Flachskampf FA, Foster E, Pellikka PA, Picard MH, Roman MJ, Seward J, Shanewise JS, Solomon SD. Recommendations for chamber quantification: a report from the American Society of Echocardiography's Guidelines and Standards Committee and the Chamber Quantification Writing Group, developed in conjunction with the European Association of Echocardiography, a branch of the European Society of Cardiology. Journal of the American society of echocardiography. 2005 Dec 1;18(12):1440-63.
- 20. Redfield MM. Understanding "diastolic heart failure". N Engl J Med. 2004;350(19):1930-1.
- 21. Grossman W, McLaurin LP. Diastolic properties of the left ventricle. Ann Intern Med. 1976;84(3):316–26.
- 22. Ishida Y, Meisner JS, Tsujioka K, Gallo JI, Yoran C, et al. Left ventricular filling dynamics: influence of left ventricular relaxation and left atrial pressure. Circulation. 1986;74(1):187–96.
- 23. Sohn DW, Chai IH, Lee DJ, Kim HC, Kim HS, et al. Assessment of mitral annulus velocity by Doppler tissue imaging in the evaluation of left ventricular diastolic function. J Am Coll Cardiol. 1997;30(2):474–80.
- 24. Sreenivasa Kumar ML, Rajasekhar D, Vanajakshamma V, Latheef K. Impact of metabolic syndrome on global left ventricular function as evaluated by the myocardial performance index. J Saudi Heart Assoc. 2014;26(3):145–51.
- 25. de las Fuentes L, Brown AL, Mathews SJ, Waggoner AD, Soto PF, et al. Metabolic syndrome is associated with abnormal left ventricular diastolic function independent of left ventricular mass. Eur Heart J. 2007;28(5):553–9. doi:10.1093/eurheartj/ehl526
- 26. Ferrara LA, Cardoni O, Mancini M, Zanchetti A. Metabolic syndrome and left ventricular hypertrophy in a general population. J Hum Hypertens. 2007;21(10):795–801.
- 27. Mahmoud A, Almuntaser I, Brown A, King G, Crean P, et al. Left ventricular structural and functional changes in the metabolic syndrome. J Cardiometab Syndr. 2019;4:81–8.
- 28. Alkholy UM, Ahmed IA, Karam NA, Ali YF, Yosry A. Assessment of left ventricular mass index could predict metabolic syndrome in obese children. J Saudi Heart Assoc. 2016;28(2):102–22.
- 29. Al Naggar Y, Al-Daydamony MM. Left ventricular hypertrophy in metabolic syndrome patients without hypertension. Int J Sci Res. 2016;4(9):1–5.
- 30. Wang SX, Song KX, Guo XY, Xue H, Chen JZ, et al. The association of metabolic syndrome with left ventricular mass and geometry in community-based hypertensive patients among Han Chinese. J Res Med Sci. 2015;20(10):963–8.
- 31. Ayalon N, Gopal DM, Mooney DM, Simonetti JS, Grossman JR, et al. Preclinical left ventricular diastolic dysfunction in metabolic syndrome. Am J Cardiol. 2014;114(6):838–42.