IOSR Journal of Dental and Medical Sciences (IOSR-JDMS)
e-ISSN: 2279-0853, p-ISSN: 2279-0861.Volume 21, Issue 6 Ser.5 (June. 2022), PP 48-52
www.iosrjournals.org

"Wall motion abnormalities among hypertensive patients"

Dr. Md. Younus Ali[1], Dr. Md. Mahfuzul Islam[2], Dr. A. K. M. Rezwanul Islam[3], Dr. Koushik Bhowmick[4], Dr. Md. Abdur Rob Rayhan[5], Dr. Sadequl Islam[6], Dr. Maskurul Alam[7]

[1]*Assistant Professor, Department of Cardiology, TMSS Medical College and Rafatullah Community Hospital, Bogura, Bangladesh.*
[2]*Associate Professor, Department of Cardiology, TMSS Medical College and Rafatullah Community Hospital, Bogura, Bangladesh.*
[3]*Consultant, Cardiology, Shaheed Ziaur Rahman Medical College & Hospital, Bogura, Bangladesh.*
[4]*Consultant, Department of Cardiology, Mymensingh Medical College Hospital, Mymensingh, Bangladesh.*
[5]*Registrar, Department of Cardiology, TMSS Medical College and Rafatullah Community Hospital, Bogura, Bangladesh.*
[6]*Assistant Registrar, Department of Cardiology, TMSS Medical College and Rafatullah Community Hospital, Bogura, Bangladesh.*
[7]*Assistant Registrar, Department of Cardiology, TMSS Medical College and Rafatullah Community Hospital, Bogura, Bangladesh.*
***Corresponding Author:** Dr. Md. Younus Ali, Assistant Professor, Department of Cardiology, TMSS Medical College and Rafatullah Community Hospital, Bogura, Bangladesh*

Abstract
***Background:** Cardiac wall motion abnormalities (WMA) are basically kinetic alterations in the cardiac wall motion during the cardiac cycle with the effect on cardiac function. Wall motion abnormalities can be categorized according to their degree and their distribution pattern whether they are global or segmental and whether they can be attributed to a coronary territory or follow a non-coronary distribution.*
***Aim of the study:** The aim of this study was to assess the patterns of wall motion abnormalities among hypertensive patients.*
***Methods:** This prospective observational study was conducted in the department of Cardiology, TMC & RCH, Bogura, Bangladesh from January 2020 to January 2021. In total 56 hypertensive patients were included as the study subjects for this study. This study was approved by the ethical committee of the mentioned hospital. A predesigned questioner was used in data collection. All data were collected, processed and analyzed by using MS Office and SPSS version 23 programs as per need.*
***Results:** In this study 21% of the patients were with segmental wall motion abnormalities, 20% were with global dysfunction and 59% were fully free from any type of WMA. Among patient with global dysfunctions, 82% were with mild and 9% were with moderate-to-severe dysfunction whereas this ratio was 58:25 in segmental abnormality patients. More than 50% segmental abnormality patients were with inferior wall dysfunction, antero-septal dysfunction, posterior septal dysfunction, LV base dysfunction and LV mid-cavity dysfunction and LV apical dysfunction separately. In echocardiographic assessment we found among segmental WMA patients, 91% were with LV hypertrophy whereas 46% were with aortic fibro-calcification. On the other hand, in global dysfunction patients, 92% were with LV hypertrophy whereas 57% were with aortic fibro-calcification.*
***Conclusion:** Majority of the participants were found free from any type of wall motion abnormality. Wall motion abnormalities were associated with greater, Cornell voltage-duration product and anatomic left ventricular mass, male gender, and microangiopathy independently of overt CHD. For getting more specific findings we would like to recommend for conducting similar more studies with larger sized samples in several places.*

***Keywords:** Wall motion abnormalities, Hypertension, Global, Segmental, Echocardiography*

Date of Submission: 06-06-2022 Date of Acceptance: 21-06-2022

I. Introduction

Cardiac wall motion abnormalities (WMA) are basically kinetic alterations in the cardiac wall motion during the cardiac cycle with the effect on cardiac function. Coronary heart disease (CHD) is one of the leading causes of mortality as well as morbidity among hypertensive patients. [1] Myocardial infarction (MI) or severe ischemia are the most common causes of left ventricular wall motion abnormalities, [2] which can reduce the

LV pump function. [3,4] Information regarding the prevalence and correlation of WM abnormalities in ambulatory high-risk hypertensive patients are limited. [5] Hypertension (HTN) is a major determinant of left ventricular hypertrophy [1] and is associated with the increased risk of left ventricular systolic dysfunction. [6] Global left ventricular ejection fraction (EF), a measure of left ventricular chamber function highly useful as an indicator of left ventricular systolic dysfunction which can be normal despite segmental wall motion abnormalities (WMA), especially when EF is measured from linear echocardiographic left ventricular dimensions at mid-cavity level, [7] or from single-plane contrast ventriculograms. Two-dimensional echocardiography allows semiquantitative evaluation of wall motion abnormalities [8,9] which are patho-physiologically associated with coronary heart disease (CHD). [4] For measuring wall motion abnormalities, wall motion score index (WMSI) is a simple method to quantify regional as well as global systolic function [10], has been shown to be an accurate estimate of LVEF [11]. WMSI (wall motion score index) is an independent predictor of outcomes following STEMI [12], as well as non-STEMI [13]. Wall motion score index does not require a high frame-rate or a very high image quality, which is the case for strain imaging by two-dimensional speckle tracking [14].

II. Methodology

This prospective observational study was conducted in the department of Cardiology, TMSS Medical College (TMC) and Rafatullah Community Hospital (RCH), Bogura, Bangladesh from January 2020 to January 2021. In total 56 hypertensive patients were included as the study subjects for this study. This study was approved by the ethical committee of the mentioned hospital. As per the inclusion criteria of this study, only patients between the age from 30 to 80 years with hypertension were included. On the other hand, as per the exclusion criteria, patients with AV or intraventricular conduction disturbances, or congenital heart disease, cardiomyopathy, rheumatic heart disease, patients on permanent pacemaker (PPM), right ventricular hypertrophy, using drugs like verapamil and patients who did not consent for the study were excluded. All necessary data regarding demographic as well clinical status, risk factors, comorbidities, histories and echocardiographic findings were recorded and analyzed as per necessity. A predesigned questioner was used in data collection. All data were collected, processed and analyzed by using MS Office and SPSS version 23 programs as per need.

III. Result

In this study, among total 56 participants, 57% were male whereas the rest 43% were female. So male participants were dominating in number and the male-female ratio was 1.3:1. Among all the participants of this study, 16%, 18%, 5% and 21% were with Diabetes, CHD, MI and clinical CVD respectively as the comorbidities and/or risk factors. The mean (±SD) systolic BP (mm Hg), diastolic BP (mm Hg) and heart rate (bpm) were found as 175±45, 99±34 and 79±17 respectively. On the other hand, the mean (±SD) total cholesterol, mg/dL and HDL cholesterol, mg/dL of the participants were 234±27, 63±56 respectively. In analyzing the frequencies of wall motion abnormalities (WMA) among participants we observed that, 21% of the patients were with segmental wall motion abnormalities, 20% were with global dysfunction and 59% were fully free from any type of WMA. Among patient with global dysfunctions, 82% were with mild and 9% were with moderate-to-severe dysfunction whereas this ratio was 58:25 in segmental abnormality patients. Besides these, more than 50% segmental abnormality patients were with inferior wall dysfunction, antero-septal dysfunction, posterior septal dysfunction, LV base dysfunction and LV mid-cavity dysfunction and LV apical dysfunction. In echocardiographic assessment we found among segmental wall motion abnormalities group patients, 91% were with LV hypertrophy whereas 46% were with aortic fibro-calcification. On the other hand, in global dysfunction group patients, 92% were with LV hypertrophy whereas 57% were with aortic fibro-calcification.

Table 1: Demographic and clinical status of participants (N=56)

Demographic status	
Age in year (Mean ±SD)	63±84
Male (%)	57
Female (%)	43
BMI, kg/m2	26.92±3.82
Comorbidities & risk factors	
Diabetes (%)	16
CHD (%)	18
MI (%)	5

Clinical CVD (%)	21
BP and heart rate distribution (Mean ±SD)	
Systolic BP, mm Hg	175±45
Diastolic BP, mm Hg	99±34
Heart rate, bpm	79±17
Lipid profile distribution (Mean ±SD)	
Total Cholesterol, mg/dL	234±27
HDL Cholesterol, mg/dL	63±56

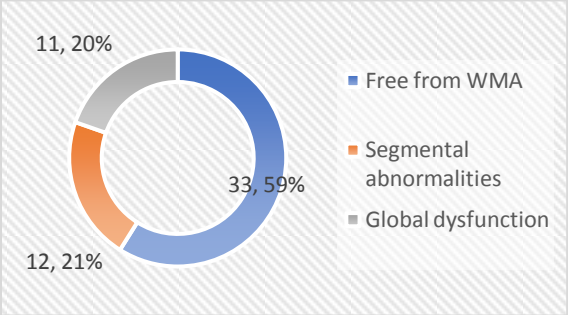

Figure 1: Frequencies of wall motion abnormalities (WMA) among participants (N=56)

Table 2: Severity and location of wall motion abnormalities among participants (N=56)

Characteristics	Segmental abnormalities (n=12)		Global dysfunction (n=11)	
	Frequency	%	Frequency	%
Mild dysfunction	7	58%	9	82%
Moderate-to-severe dysfunction	3	25%	1	9%
Inferior wall dysfunction	9	75%	0	0
Antero-septal dysfunction	8	67%	0	0
Anterior wall dysfunction	4	33%	0	0
Lateral wall dysfunction	3	25%	0	0
Posterior wall dysfunction	5	42%	0	0
Posterior septal dysfunction	7	58%	0	0
LV base dysfunction	8	67%	0	0
LV mid-cavity dysfunction	9	75%	0	0
LV apical dysfunction	8	67%	0	0

Table 3: Echocardiographic correlation between segmental and global wall motion abnormalities participants (N=56)

Characteristics	Segmental abnormalities (n=12)	Global dysfunction (n=11)	P value
LV hypertrophy (%)	91%	92%	
Aortic fibro-calcification (%)	46%	57%	
LV septum (cm)	1.15±0.18	1.16±0.88	0.970
LV posterior wall (cm)	1.12±0.14	1.06±0.13	0.300
LV diastolic diameter (cm)	5.9±0.52	5.9±0.47	1.000
LV mass (g)	269.12±64.77	283.51±57.29	0.580
Left atrium (cm)	4.33±0.67	4.41±0.59	0.765
Aortic root diameter (cm)	3.68±0.51	3.72±0.48	0.849
Doppler-derived EF (%)	44.32±18.71	43.81±21.57	0.952
WM score-derived EF (%)	48.43±7.78	44.37±4.97	0.155
Cardiac index, L/min	2.54±0.71	2.45±0.69	0.761

IV. Discussion

The aim of this study was to assess the patterns of wall motion abnormalities among hypertensive patients. In our study, in echocardiographic assessment we found among segmental wall motion abnormalities group patients, 91% were with LV hypertrophy whereas 46% were with aortic fibro-calcification. On the other hand, in global dysfunction group patients, 92% were with LV hypertrophy whereas 57% were with aortic fibro-calcification. The 12.5% prevalence of LV wall motion abnormalities (WMA) in LIFE echo sub-study patients is nearly 3 times higher than the 4.3% reported among hypertensive adult patients in the Cardiovascular Health Study. [5] One most potential explanation is the selection of subjects with ECG left ventricular hypertrophy for LIFE, in view of the known association of higher left ventricular (LV) mass with CHD, [15,16] myocardial infarction (MI), [17] and larger myocardial infarction size. [18] In analyzing the frequencies of wall motion abnormalities (WMA) among participants we observed that, 21% of the patients were with segmental wall motion abnormalities, 20% were with global dysfunction and 59% were fully free from any type of WMA. Among patient with global dysfunctions, 82% were with mild and 9% were with moderate-to-severe dysfunction whereas this ratio was 58:25 in segmental abnormality patients. Besides these, more than 50% segmental abnormality patients were with inferior wall dysfunction, anterior septal dysfunction, posterior septal dysfunction, LV base dysfunction and LV mid-cavity dysfunction and LV apical dysfunction. Segmental wall motion abnormalities can be related to subclinical coronary artery disease. [4] Up to one third of acute MI (Myocardial infarctions) can be clinically silent; [19,20] up to 30% of acute MI (Myocardial infarctions) never manifest diagnostic Q waves; a diagnostic Q wave disappears in 10% to 30% of Q-wave infarctions [21] or is not diagnosed 2 years after an acute MI (Myocardial infarction). [22] Therefore, silent ischemia or MI (Myocardial infarction) or chronic ischemia or hibernating myocardium may cause wall motion abnormalities (WMA) at rest. Although mild hypokinesis may be a normal variant, [23] hypokinesis is strongly associated with significant CHD. [4]

Limitation of the study:

This was a single centered study with a small sized sample. So, findings of this study may not reflect the exact scenario of the whole country.

V. Conclusion & Recommendation

Through echocardiography wall motion abnormalities are usually found in about one eighth of patients with moderately severe hypertension as manifested by blood pressure level and presence of ECG-LV hypertrophy. Although the exclusion of patients with recent myocardial infarction or stroke, heart failure, or clinical need for beta-blocker or ACE-inhibitor therapy. Wall motion abnormalities were associated with greater Cornell voltage-duration product and anatomic left ventricular mass, male gender, and microangiopathy independently of overt CHD. For getting more specific findings we would like to recommend for conducting similar more studies with larger sized samples in several places.

Funding: *No funding sources*
Conflict of interest: *None declared*
Ethical approval: *The study was approved by the Institutional Ethics Committee.*

References

[1]. The sixth report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure. Arch Intern Med. 1997;157:2413–2446. (Correction. 1998;158:573.)
[2]. Horowitz RS, Morganroth J, Parrotto C, Chen CC, Soffer J, Pauletto FJ. Immediate diagnosis of acute myocardial infarction by two- dimensional echocardiography. Circulation. 1982;65:323–329.
[3]. Nishimura RA, Tajik AJ, Shub C, Miller FA Jr, Ilstrup DM, Harrison CE. Role of two-dimensional echocardiography in the prediction of in-hospital complications after acute myocardial infarction. J Am Coll Cardiol. 1984;4:1080 –1087.
[4]. Lewis SJ, Sawada SG, Ryan T, Segar DS, Armstrong WF, Feigenbaum H. Segmental wall motion abnormalities in the absence of clinically documented myocardial infarction: clinical significance and evidence of hibernating myocardium. Am Heart J. 1991;121:1088 –1094.
[5]. Gardin JM, Siscovick D, Anton-Culver H, Lynch JC, Smith VE, Klopfenstein HS, Bommer WJ, Fried L, O'Leary D, Manolio TA. Sex, age, and disease affect echocardiographic left ventricular mass and sys- tolic function in the free-living elderly. The Cardiovascular Health Study. Circulation. 1995;91:1739 –1748.
[6]. Devereux RB, Bella JN, Palmieri V, Oberman A, Kitzman DW, Hopkins PN, Rao DC, Morgan D, Paranicas M, Fishman D, Arnett DK. Left ventricular systolic dysfunction in a biracial sample of hypertensive adults: The Hypertension Genetic Epidemiology Network (HyperGEN) Study. Hypertension. 2001;38:417– 423.
[7]. Teichholz LE, Kreulen T, Herman MV, Gorlin R. Problems in echocar- diographic volume determinations: echocardiographic-angiographic cor- relations in the presence of absence of asynergy. Am J Cardiol. 1976; 37:7–11.
[8]. Kisslo JA, Robertson D, Gilbert BW, von Ramm O, Behar VS. A comparison of real-time, two dimensional echocardiography and cinean- giography in detecting left ventricular asynergy. Circulation. 1977;55: 134 –141.

[9]. Freeman AP, Giles RW, Walsh WF, Fisher R, Murray IP, Wilcken DE. Regional left ventricular wall motion assessment: comparison of two- dimensional echocardiography and radionuclide angiography with contrast angiography in healed myocardial infarction. Am J Cardiol. 1985;56:8 –12.
[10]. R.M. Lang, L.P. Badano, V. Mor-Avi, J. Afilalo, A. Armstrong, L. Ernande, 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, Eur. Heart J. 16 (3) (2015) 233–271.
[11]. J.H. McGowan, J.G. Cleland, Reliability of reporting left ventricular systolic function by echocardiography: a systematic review of 3 methods, Am. Heart J. 146 (3) (2003) 388–397.
[12]. J.E. Møller, G.S. Hillis, J.K. Oh, G.S. Reeder, B.J. Gersh, P.A. Pellikka, Wall motion score index and ejection fraction for risk stratification after acute myocardial infarction, Am. Heart J. 151 (2) (2006) 419–425.
[13]. C. Eek, B. Grenne, H. Brunvand, S. Aakhus, K. Endresen, P.K. Hol, et al., Strain echocardiography and wall motion score index predicts final infarct size in patients with non-ST-segment-elevation myocardial infarction, Circ. Cardiovasc. Imaging 3 (2) (2010) 187–194.
[14]. M. Dandel, H. Lehmkuhl, C. Knosalla, N. Suramelashvili, R. Hetzer, Strain and strain rate imaging by echocardiography - basic concepts and clinical applicability, Curr. Cardiol. Rev. 5 (2) (2009) 133–148.
[15]. Pech HJ, Witte J, Romaniuk R, Parsi RA, Porstmann W. Left ventricular mass in coronary artery disease without hypertension. Haemodynamic and angiocardiographic study. Br Heart J. 1974;36:362–367.
[16]. Strauer BE. Ventricular function and coronary hemodynamics in hyper- tensive heart disease. Am J Cardiol. 1979;44:999 –1006.
[17]. Yotsukura M, Suzuki J, Yamaguchi T, Sasaki K, Koide Y, Mizuno H, Yoshino H, Ishikawa K. Prognosis following acute myocardial infarction in patients with ECG evidence of left ventricular hypertrophy prior to infarction. J Electrocardiol. 1998;31:91–99.
[18]. Koyanagi S, Eastham C, Marcus ML. Effects of chronic hypertension and left ventricular hypertrophy on the incidence of sudden cardiac death after coronary artery occlusion in conscious dogs. Circulation. 1982;65: 1192–1197.
[19]. Margolis JR, Kannel WS, Feinleib M, Dawber TR, McNamara PM. Clinical features of unrecognized myocardial infarction—silent and symptomatic. Eighteen year follow-up: the Framingham study. Am J Cardiol. 1973;32:1–7.
[20]. Medalie JH, Goldbourt U. Unrecognized myocardial infarction: five-year incidence, mortality, and risk factors. Ann Intern Med. 1976;84:526 –531.
[21]. Wasserman AG, Bren GB, Ross AM, Richardson DW, Hutchinson RG, Rios JC. Prognostic implications of diagnostic Q waves after myocardial infarction. Circulation. 1982;65:1451–1455.
[22]. Bergovec M, Zigman M, Prpic H, Mihatov S, Vukosavic D. Global and regional parameters of left ventricular performance in healthy subjects during rest and exercise assessed by radionuclide ventriculography. Int J Card Imaging. 1993;9:39 – 48.
[23]. Haendchen RV, Wyatt HL, Maurer G, Zwehl W, Bear M, Meerbaum S, Corday E. Quantitation of regional cardiac function by two-dimensional echocardiography. I. Patterns of contraction in the normal left ventricle. Circulation. 1983;67:1234 –1245.

Dr. Md. Younus Ali, et. al. "Wall motion abnormalities among hypertensive patients." *IOSR Journal of Dental and Medical Sciences (IOSR-JDMS)*, 21(06), 2022, pp. 48-52.