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RESEARCH ARTICLE

Impact of asymptomatic hypertension on left and right ventricular functions by echocardiography

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ABSTRACT

Background: Hypertension generally has no symptoms and is called the silent killer as the first warning presentation may itself be a cardiac failure or myocardial infarction, which is often massive and fatal. Hypertension many times remains asymptomatic for several years and is detected on regular health check-ups. **Aims and Objective:** The objective of this study is to study the cardiac changes in both right and left ventricles by echocardiography in asymptomatic hypertensive participants. **Materials and Methods:** Twenty-five asymptomatic cases and 29 normotensive controls (total 54 participants) were studied. All the participants were male aged between 20 years and 60 years and above. Clinical evaluation was done by determining their age, systolic and diastolic blood pressures, heart rate, and symptoms. Cardiac structural and functional changes were studied by echocardiogram. Cardiac output, stroke volume, left ventricular (LV) mass, right ventricular thickness, LV systolic and diastolic dimensions and functions, fractional fiber shortening, and ejection fraction were studied in both cases and controls. **Results:** There was a change in LV mass in asymptomatic hypertensive participants when compared with control participants. There was a statistically significant change in LV mass measured in grams (weight) showing $P < 0.001$ (0.00 vs. 0.00) in asymptomatic cases and healthy normotensive controls, and the LV posterior wall also showed a statistically significant change with $P < 0.001$ (0.008 vs. 0.009) in asymptomatic cases and normotensive controls. **Conclusions:** There is increased cardiac mortality and morbidity in asymptomatic hypertensive participants in whom the cardiac damage is concealed, compared with symptomatic hypertensives who consult a physician as soon as symptoms are felt.

KEY WORDS: Asymptomatic Hypertension; Ventricular Functions; Echocardiography

INTRODUCTION

The WHO defines health as a state of complete physical, mental, and social well–being, and merely the absence of disease or illness. The disease can be communicable such as flu and AIDS and non-communicable such as hypertension and diabetes. Hypertension or high blood pressure is a condition where blood pressure levels are elevated. If repeated checks on blood pressure show that it is ≥140/90 mmHg, it can be labeled as hypertension. In adults, normal blood pressure is at 120/80 mmHg, here 120 mmHg is the systolic blood pressure, and 80 mmHg is the diastolic blood pressure.[1] As the systolic and diastolic pressures increase, the heart has to pump the blood against resistance, due to which both heart and blood vessels will be damaged and lead to heart as well as systemic diseases. Hypertension is now gaining more importance because of its increased incidence in general population; even the younger people are affected by it. Many studies[2-4] have been done and are being done on hypertension because of its risk factors and

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are especially dangerous because it often has no warning symptoms and has deleterious effect on heart as well as on other organs.

In hypertension, the heart has to change its functional anatomy and must work hard to compensate and supply the blood to various organs. This results in muscle thickening, which is called hypertrophy.[5] This thickened vessels pump inefficiently, and over time the force of their contractions weakens. The heart muscles then have difficulty relaxing and filling the heart with blood. Hypertension is the common cause of ventricular hypertrophy,[6] which is also called remodeling, is helpful to maintain normal heart functions for a short period but irreversible damage and later the heart begins to fail.

Ventricular hypertrophy can be easily detected on electrocardiogram (ECG) and echocardiogram. Detecting changes in the right ventricle are difficult due to its location. Left ventricular (LV) hypertrophy can alone be a predictive factor for cardiovascular morbidity.

Previously, many studies were done to study the changes in heart anatomy and functions in hypertension by echocardiography. Other analogous studies were done invasively and non-invasively. Many authors[7-9] had worked on similar kind of subject of hypertension and heart functions in controls and asymptomatic hypertensive participants and concluded that LV systolic functions were normal, but the stroke volume (SV), ejection fraction (EjF), cardiac output (CO), and cardiac index increased in hypertensives.

Hypertension generally has no symptoms and is called the silent killer as the first warning presentation may itself be a cardiac failure or myocardial infarction, which is often massive and fatal. Hypertension many times remains asymptomatic for several years and is detected on regular health check-ups.[10] Studying cardiac changes in both the right ventricle and left ventricle by echocardiography in asymptomatic hypertensive participants, comparing with normotensives can be helpful to detect the cardiac changes and effective functions in early stages and starting treatment, which can improve cardiac functions and decrease morbidity and mortality caused by undetected, and therefore, untreated hypertension.

MATERIALS AND METHODS

Design

This is an observational case–control study.

Duration

The study duration was during February 2017–December 2018.

Setting

Department of Physiology, Gandhi Medical College, Secunderabad, Telangana.

Place of Study

The study was conducted in the Department of Physiology, Gandhi Medical College, Secunderabad, Telangana.

Type of Study

This is an observational case–control study.

Sample Collection

Echocardiography method by the same cardiology intern both in hypertensive and normotensive participants by the same machine. Apical four-chamber view was done to assess the right ventricular (RV) functions and its measurements; apical two-chamber view was used to assess LV functions; and parasternal long axis and short axis were used to detect other muscular structures and functions.

Sampling Methods

SV = (LV diastolic diameter [LViDd])3–(LV systolic diameter [LViDs])3

$$\text{EjF} = \frac{(\text{LVIDd})3 - (\text{LVIDs})3}{(\text{LVIDd})3}$$

Fractional fiber shortening = (LVEDD – LVESD)/LVEDD
FFS% = (EDD–ESD)/EDD × 100
CO = SV × Heart rate
*LVID is LV internal diameter
*d is Diastole *s is Systole
*EDD is End-diastolic diameter
*ESD is End-systolic diameter.

Inclusion Criteria

Male hypertensive participants aged 20–60 years and above with normal heart rate and with systolic blood pressure >120 mmHg and diastolic blood pressure >80 mmHg.

Exclusion Criteria

Participants with symptoms such as headache, blurring of vision, giddiness, epistaxis, discomfort in chest, chest pain, shortness of breath, any other urinary problems, or diabetes were excluded from the study.

Statistical Methods

The results obtained were tabulated and analyzed using the appropriate statistical program.

RESULTS

Result Analysis

Twenty-five asymptomatic hypertensive participants were compared with 29 controls who's blood pressure were within the normal level, and all the study participants satisfied the inclusion and exclusion criteria were taken up for the study.

Age

The age of controls ranged from 21 to 85 years, with a mean of 45.4 ± 29.90 given in Table 1. The age ranged from 20 to 65 years, with a mean of 46.4 ± 23.60 in asymptomatic hypertensive table.

Heart Rate

In both participants, that is in hypertensive and controlled heart rate was within the normal ranges that is 60–98 beats/min, and mean in controls was between 79.38 ± 23.32 and in hypertensive it was and in hypertensive it was 79.92 ± 19.89.

Blood Pressure

The normal participants had systolic blood pressure ranging from 110 to 170 mmHg with a mean of 128.28 ± 30.27, and diastolic blood pressure ranging from 70 to 90 mmHg and the mean was 79.59 ± 97.02. The systolic blood pressure in hypertensive participants ranged from 100 to 180 mmHg, with a mean of 149.12 ± 38.78, and diastolic pressure ranged from 80 to 120 mmHg, with a mean of 96.32 ± 15.732.

Table 1: Comparison between the groups

Character	Normal participants	Hypertensive participants	*P*
	Mean±2SD	Mean±2SD	
Age	45.4±29.90	46.4±23.60	>0.05
Heart rate	79.38±23.32	79.92±19.89	>0.05
Systolic BP	128.28±30.27	149.12±38.78	<0.001
Diastolic BP	79.59±97.02	96.32±15.732	<0.001
LV diastolic diameter	4.4±0.927	4.372±0.8534	>0.05
LV systolic diameter	2.83±0.762	2.84±0.962	>0.05
IV w	1.04±0.291	1.02±0.246	>0.05
Right ventricular wall	3.468±0.832	3.468±1.06	>0.05
LV mass	131.31±74.70	170.6±74.71	<0.05
LV Pw	0.8. ±0.259	0.978±0.237	<0.05
IVSD	0.981±0.381	1.10±0.739	>0.05
EJ%	64.45±18.17	63±23.66	>0.05
FFS%	34.86±12.25	35.96±15.10	>0.05
Stroke volume	59.92±52.05	55.87±50.56	>0.05
Cardiac output	5.0±3.35	4.9±3.39	>0.05

BP: Blood pressure, LV: Left ventricular

Echocardiographic Evaluation

LV diastolic diameter in normal ranged from 3 to 5.2 with a mean of 4.4 ± 0.927 and in hypertensive ranged from 3.5 to 5.2 with a mean of 4.372 ± 0.8534.

LV systolic diameter in normal ranged from 2.1 to 3.4 with a mean of 2.83 ± 0.762 and in hypertensive participants ranged from 2.3 to 3.4 with a mean 2.84 ± 0.962.

IV wall in normotensive participants ranged from 0.8 to 1.3 with a mean of 1.04 ± 0.291, and in study participants, it ranged from 0.8 to 1.3 with a mean of 1.02 ± 0.246.

RV wall controls RV wall size ranged from 2.4 to 4 with an average mean of 3.468 ± 0.832 and in hypertensive ranged from 2.5 to 4 with a mean of 3.468 ± 1.06.

LV mass in the controls is ranged from 86 to 154 mg with a mean of 131.31 ± 74.70 and in asymptomatic ranged from 94 to 241 mg with a mean of (170.6 ± 74.71).

Wall thickness the posterior wall of the left ventricle in normal participants, ranged from 0.7 to 1.1 and mean of 0.8 ± 0.259 and in hypertensive it ranged from 0.7 to 1.1 with a mean of 0.978 ± 0.237.

Ventricular septal thickness in normal ranged from 0.72 to 1.5 with a mean of 0.981 ± 0.381, and in hypertensive, it ranged from 0.8 to 1.9 with a mean of 1.10 ± 0.739.

EJ% in normal ranged from 38 to 78% with a mean of 64.45 ± 18.17 and in hypertensive it ranged from 43 to 79% with a mean of (63 ± 23.66).

FFS% in controls ranged from 23 to 46% with a mean of 34.86 ± 2.25, and in study participants ranged from 24 to 47% with a mean of 35.96 ± 15.10.

SV in controls ranged from 7 to 101 ml with a mean of 59.92 ± 52.05 and in asymptomatic ranged from 9.1 to 121 ml with a mean of (55.87 ± 50.56).

CO ranged from 1.5 to 8 l in normal with a mean of 5.0 ± 3.35 and ranged from 2.2 to 9 l with a mean of 4.9 ± 3.39 in asymptomatic hypertensive participants.

DISCUSSION

An endeavor was made to study non-invasively by echocardiogram on 25 asymptomatic hypertensive participants comparing their ages, blood pressure, heart rate, and other echocardiography findings with normotensive participants taken as controls. Hypertension can be due to idiopathic or secondary cause where there is a considerable effect on the heart, and it has to undergo certain changes to

maintain mundane functions to preserve the milieu interior. Incremented cardiac mass associated with hypertension is the result of structural adaptation by the heart to function customarily at high pressure. The echocardiographic study revealed that in 25 asymptomatic hypertensive patients compared with 29 normotensives, all the parameters of both the ventricular functions such as SV, CO, EjF, and fractional fiber minimizing were in mundane ranges. The parameters that were highly consequential among the study participants compared to controls were systolic and diastolic blood pressures and the left ventricular mass and posterior left ventricular wall. The left ventricular mass ranged from 86 to 154 gms in normotensives, with a mean of 131.31 + 74.7 when compared to asymptomatic hypertensives whose left ventricular mass ranged from 94 to 241 gms, with a mean of 170 + 74.7 which gave a consequential value of $P < 0.001$. The other parameters were posterior wall diameter that ranged from 0.7 to 1.1, with a mean of 0.886–0.2596 in controls as compared with range of 0.7–1.1 and a mean of 0.978–0.2378 with $P < 0.001$ in study participants. The other parameters which were highly paramount with $P < 0.001$ were systolic and diastolic blood pressures, but these values were not considered since the study itself was to optically discern the difference hypertensives and normotensives. However, the transmutations in the right ventricle did not show much in hypertensives when calculated for RV wall thickness at diastole.

Several studies[11-13] revealed that in hypertensive participants, there was consequential vicissitude in RV wall thickness, hypertrophy in interventricular septum, and transmutes in diastolic functions. Both the paramount changes, especially left ventricular mass and posterior wall diameter be token that there is paramount left ventricular hypertrophy in asymptomatic hypertensive subjects compared to normotensives. These data can be explained by compensatory mechanisms by the heart. Many studies[14-16] attested that there is consequential left ventricular hypertrophy, transmutations in ventricular and afterload, and other systolic and diastolic functions. The information suggests that when left ventricular pressure raise, left ventricular hypertrophy occurs as a categorical adaptive quantification that accommodates to keep the force of contraction of the heart muscle within the mundane range.[17,18] This results from the categorical adaptive pattern of concentric hypertrophy, in compensated phase, this increases left ventricular wall thickness but keeps the cavity size unchanged, which had been detected by echocardiography in this study; the ventricular hypertrophy first commences as a physiological hypertrophy to supply the injuctive authorization and later becomes pathological change and damaged irreversible. This kind of study can estimate myocardial damage in early stages and starting treatment with anti-hypertensive drugs can stop irreversible damage to the heart.

Incremented cardiac mortality and morbidity in asymptomatic hypertensive participants is observed in whom the cardiac damage is concealed, compared with symptomatic hypertensives who consult a medico when symptoms show up.

Limitations of ECG

Not all heart problems will show up on an ECG. False positives and false negatives are common among clinical tests. False-negative is probably the biggest concern with ECG. For some heart patients, the ECG may be entirely normal, and yet their conditions should be reflected in the ECG.

CONCLUSIONS

There is increased cardiac mortality and morbidity in asymptomatic hypertensive participants in whom the cardiac damage is concealed, compared with symptomatic hypertensives who consult a physician as soon as symptoms are felt.

REFERENCES

1. Alicandri C, Fouad FM, Tarazi RC, Bravo EL, Greenstreet RL. Sympathetic contribution to the cardiac response to stress in hypertension. Hypertension 1983;5:147-54.
2. Pearson AC, Gudipati C, Nagelhout D, Sear J, Cohen JD, Labovitz AJ, *et al.* Echocardiographic evaluation of cardiac structure and function in elderly subjects with isolated systolic hypertension. J Am Coll Cardiol 1991;17:422-30.
3. Phillips BJ. RDMS: Manual of Echocardiography Techniques. Philadelphia, PA: WB Saunders Company; 1980. p. 139-40.
4. Francis CM, Caruana L, Kearney P, Love M, Sutherland GR, Starkey IR, *et al.* Open access echocardiography in management of heart failure in the community. BMJ 1995;310:634-6.
5. Davies M, Hobbs F, Davis R, Kenkre J, Roalfe AK, Hare R, *et al.* Prevalence of left-ventricular systolic dysfunction and heart failure in the echocardiographic heart of England screening study: A population based study. Lancet 2001;358:439-44.
6. De Simone G. Left ventricular chamber and wall mechanism. J Hypertens1999;17:1001-106.
7. Ganau A, Devereux RB, Roman MJ, de Simone G, Pickering TG, Saba PS, *et al.* Patterns of left ventricular hypertrophy and geometric remodeling in essential hypertension. J Am Coll Cardiol 1992;19:1550-8.
8. Drescher E, Austenat J, Günther KH, Purfürst WD, Hujer W. Echocardiography in hypertension. Dimensions and mass of the left ventricle. Comparison with controls and sportsmen. G Ital Cardiol 1980;10:843-50.
9. Dunn FG, Chandraratna P, deCarvalho JG, Basta LL, Frohlich ED. Pathophysiologic assessment of hypertensive heart disease with echocardiography. Am J Cardiol 1977;39:789-95.
10. Forlich ED. Physiological consideration of left ventricula hypertrophy. Am J Med 1893;75:12-8.
11. Feigenbum H. Echocardiography. 3rd ed. Philadelphia, PA: Lea and Febiger; 1981.
12. Messerli FH. Clinical determinants and consequences of left ventricular hypertrophy. Am J Med 1983;75:51-6.
13. Abi-Samra F, Fouad FM, Tarazi RC. Determinants of left ventricular hypertrophy and function in hypertensive patients.

An echocardiographic study. Am J Med 1983;75:26-33.
14. Bhatt JV. Impact of asymptomatic hypertension on left ventricular functions. Indian J Physiol Pharmacol 2003;47:171-8.
15. JNC Joint National Committee on Prevention on Prevention, Detections. Evaluation and Treatments of High Blood Pressure, 7th Report. Bethesda, MD: National Heart, Lung, and Blood Institute, US; 2003.
16. Kirkendall WM, Burton AC, Epstein FH, Freis ED. Recommendations for human blood pressure determination by sphygmomanometers. Circulation 1967;36:980-8.
17. McFarland TM, Alam M, Goldstein S, Pickard SD, Stein PD. Echocardiographic diagnosis of left ventricular hypertrophy. Circulation 1978;57:1140-4.
18. Rao MS. Pathogenesis and consequences of essential hypertension. J Indian Med Assoc 2003;101:251-3.

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