Ischemic Changes in Hypertensive Patients with Left Ventricular Hypertrophy

الثغرات اللافقارية في مرضى ارتفاع ضغط الدم مع تضخم البطين الأيسر

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Abstract:

This study aimed to Evaluate forty five hypertensive patients with left ventricular hypertrophy were included for enrollment in a clinical research was conducted in the Al-Ryada hospital and Aden international German hospital in Aden governorate with informed consent was obtained from each participant, where analyzed in a cross-sectional manner to compare the characteristics of those with left ventricular hypertrophy and ischemia evoked on a hand grip and treadmills exercise test, in patients with no previous ischemic attacks. Group I, n=8, versus those with a normal exercise stress tests group II, n=37. There were no differences in age, sex, severity, and duration of hypertension between the two groups, but group I patients were significantly more overweight. Blood pressure at peak treadmills exercise was higher in group I despite shorter exercise duration, although resting pressures were similar. Group I patients had evidence of more pronounced cardiac enlargement and left ventricular hypertrophy by both electrocardiograph and echocardiograph criteria (LVM P < 0.001 and ischemic changes respectively <0.001), but both groups had equally good systolic function and similar degrees of mild diastolic dysfunction. Analysis of electrocardiograph showed a significantly greater propensity to increase heart rates in group I. This study provide a conclusion that, hypertensive patients with LVH, overweight and

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attained higher systolic pressure at treadmills exercise, despite a shorter duration, have propensity for higher risk of ischemic attacks at stress. Further study and assessment is needed in such risk patients.

Keywords: *primary atrial hypertension, Left ventricular hypertrophy*

الملخص:

هدفت الدراسة إلى تقييم وجود خمسة وأربعين من مرضى ارتفاع ضغط الدم مع تضخم البطين الأيسر تم إدراجهم للتسجيل في بحث سريري أجري في مستشفى الريادة ومستشفى عدن الألماني الدولي في محافظة عدن بموافقة مستنيرة تم الحصول عليها من كل مشارك ، حيث تم تحليلها في طريقة مستعرضة لمقارنة تلك الخصائص مع تضخم البطين الأيسر ونقص التروية تثار على اختبار قبضة اليد والممارسة تمارين ، في المرضى الذين يعانون من أي هجمات نقص ترويه السابقة. المجموعة الأولى ، ن = ٨ ، مقابل تلك مع اختبارات الإجهاد ممارسة عادية المجموعة الثانية ، ن = ٣٧. لم تكن هناك اختلافات في العمر والجنس وشدة ومدة ارتفاع ضغط الدم بين المجموعتين ، ولكن المرضى من المجموعة الأولى كانوا يعانون زيادة كبيرة في الوزن. كان ضغط الدم عند ممارسة تمارين الذروة أعلى في المجموعة الأولى بالرغم من مدة التمرين القصيرة ، على الرغم من أن الضغوط الباقية كانت متشابهة. كان لدى مرضى المجموعة الأولى دليلاً على تضخم القلب بشكل أكثر وضوحا وتضخم البطين الأيسر من خلال كل من مخطط القلب الكهربائي ومخطط ضربات القلب (LVM P <0.001 وتغيرات إقفارية على التوالي <٠.٠٠١) ، لكن كلا المجموعتين كان لهما وظيفة انقباضية جيدة على قدم المساواة ودرجات متشابهة من الخلل الوظيفي الانبساطي. أظهر تحليل مخطط القلب الكهربائي ميلًا أكبر بشكل ملحوظ لزيادة معدلات ضربات القلب في المجموعة الأولى. توصلت هذه الدراسة إلى استنتاج مفاده أن مرضى ارتفاع ضغط الدم الذين يعانون من انخفاض ضغط الدم وفرط الوزن والضغط الانقباضي الأعلى في تمرينات الدوس ، على الرغم من مدة أقصر ، لديهم ميل لزيادة خطر الإصابة بنوبات نقص ترويه في التوتر. هناك حاجة إلى مزيد من الدراسة والتقييم في هؤلاء المرضى المعرضين للخطر.

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Introduction:

In one of the very early reports from the Framingham Heart Study, the term "factors of risk" was introduced to identify those very specific clinical abnormalities that conferred increased risk for premature cardiovascular morbidity and mortality associated with coronary heart disease. (1) Included among those initial cardiovascular risk factors were: hypertension, left ventricular hypertrophy (LVH), and hypercholesterolemia. (2)

Many experimental and clinical studies have demonstrated that there is impaired left ventricular coronary blood flow and flow reserve in hypertension.(3) and those studies have been directed to determine underlying mechanisms that explain the risk associated with LVH even without co-existing atherosclerotic coronary arterial disease.(4) Among these risk phenomena that are associated with the development of LVH are ischemia, fibrosis, apoptosis, and long-standing dietary salt excess and inflammatory factors. (5)

LVH is an independent predictor of risk in the general population, in essential hypertension, and in coronary artery disease, (6) and was one of the first three factors of risk originally identified to predisposing the patient to premature morbidity and mortality resulting from coronary heart disease. (7) Morphological and physiological changes in the vasculature have been described in the evolution and maintenance of hypertension.(8) Hypertension-induced vascular dysfunction may present itself as a contributing, or consequential factor,(9) to vascular remodeling caused by chronically elevated systemic arterial blood pressure. (10)

Other than age, LVH is the most potent predictor of adverse cardiovascular outcomes in the hypertensive population, (11) and is an independent risk factor for coronary heart disease, sudden death, heart failure and stroke. (12) Although directly related to systolic blood pressure, other factors including age, sex, race, body mass index and stimulation of the renin-angiotensin-aldosterone and sympathetic nervous systems play an important role in the pathogenesis of LVH involving changes in myocardial tissue architecture. (13) The principal structural adaptation of the heart to an increased pressure load is LVH, (14) essentially producing an increase in wall thickness at the expense of chamber volume, (15) although increases in arterial pressure may be sustained by enhanced left ventricular contractility with little or no LVH, therefore, it cannot be regarded as a benign compensatory mechanism, (16) for its presence carries a risk of mortality that is independent of blood pressure and other risk factors. (17)

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As already suggested, in ischemic heart disease a number of functional changes are related to the increased oxygen demand based on the magnitude of the increased ventricular wall tension, (18) additionally, there is altered auto regulatory reserve and, of course, the impaired coronary hemodynamics associated with the extent of the LVH (19) Diagnostic modalities are under investigation to add to improved management for the patient with hypertension having LVH. (20) Hence, after more than 50 years, knowledge is becoming available to explain the risk associated with LVH and to direct clinical management to reduce its associated risk. (21)

Methods:

The study comprised 45 hypertensive patients (40 men, 5 women; mean age, 52±10 years; range, 43 to 62) with mild to moderate essential hypertension evaluated in the Al - Ryada hospital and Aden international German hospital in Aden governorate. All patients underwent Echocardiographic examination with system (Sonos 1000). Complete M-mode, two-dimensional, and Doppler echocardiographic studies were obtained. Left ventricular mass (LVM) was estimated by Penn equation (LV Mass (g) 0.8{1.04[([LVEDD + IVSd + PWd]3 LVEDD3)]} + 0.6) Quantitative analysis of M-mode echocardiograms provided the following parameters, thus allowing the assessment of left ventricular anatomy and function: end-diastolic (LVDD) and end-systolic diameters (LVSD), interventricular septum (IVSD) and posterior wall (PWD) of left ventricle thickness in diastole at three sequential levels. Fractional shortening (FS was used as an index of systolic function. The proportion of blood that is pumped from the left
ventricle with each heart beat was assessed by ejection fraction (EF) and the diastolic function was assessed by calculating the early diastolic filling velocity (E wave), late diastolic filling velocity (A wave), and E-to-A ratio (<1 being indicative of diastolic dysfunction). Blood pressure measured as the mean of three readings taken 2 minutes apart after 10 minutes of rest. Handgrip simple exercise tests were performed using a hand gripper and measurements of blood pressure were taken at peak of effort. Treadmill exercise test last about 15-20 minutes and patients asked to be not having a heavy meal within one hour of the test as well to stop taken beta blocker. Patients putting on treadmill and the

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exercise started at every easy pace and gradually made more strenuously by increasing the speed and incline of the treadmill. Blood pressure measured from time to time and ECG tracing are made and Standard 12-lead electrocardiograms were recorded. Sokolow index, duration of P wave , QRS complex, axis and total voltage were the collected data were fed to the computer using the statistical package for social science (SPSS) .Statistical analysis were done by ANOVA for measures within groups and linear analysis presented as mean ± SD . Difference were considered to be significant if P< .05.

Results:

Among the 8 patients (group I) the mean age was 54 years (SD = ± 8) 2 of them were female, (group II) were 37 patients with mean age 52 years (SD = ± 10) 3 of them were female. Table I, displays both groups were similar for age, smoking, duration of hypertension, but group I were more overweight than patients of group II.

In Table II, all patients had similar clinic blood pressure at rest, although patients of (group I) had a tendency to higher systolic blood pressure during exercise by handgrip and did increase significantly at peak exercise during treadmill testing.

Table III, revealed the echocardiographic data of LV anatomy and function. There was no difference in posterior wall and IVS thickness in diastole, but patients of (group I) had greater LVDD as well as LVM. All patients had normal systolic function with an ejection fraction >50%, but all patients had some degree of diastolic dysfunction with an E-A ratio <1.

Analysis of the electrographic data on Table IV revealed that there was significant higher ischemic changes on exercise (group I) versus (group II); as well Sokolow index was observed with significant criteria in group I.

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Table I: Data characteristics of the two studied groups

Data	Group I (n=8)	Group II (n=37)	Statistical significance
Age (Years)	54±8	52±10	P < 0.06
Sex (M/F)	6/2	34/3	P < 0. 1
Weight (kg)	100±18	85±13	P < 0.001**
Height (cm)	174±8	173±9	P < 0.8
Smoking (%)	25	21	P < 0.49
Duration HTN (Years)	11±3	9±2	P < 0.01

Table II: Blood Pressure Measurements

DATA	Group I (n=8)	Group II (n=37)	Statistical significance
REST			
SPB (mm Hg)	157±19	159±13	P < 0.050
DBP (mm Hg)	104±9	104±6	P < 0.07
Handgrip			
SPB (mm Hg)	198 ±16	167±21	P < 0.05*
DBP (mm Hg)	110±9	116 ±8	P < 0.06
Treadmill testing			
SBP (mm Hg)	221±4	195±12	P < 0.001 **

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Table III: Echocardiographic Data

DATA	Statistical significance	Group II (n=37)	Group I (n=8)
IVSD	0.12± 0.1	0.11±0.3	P < 0.6
PLVWD	13.0±2.0	11.7 ±1.0	P < 0.06
LVDD	53.5±3.0	50.8±4.0	P < 0.05*
LVM	177. 4± 38	152.6±11	P < 0.001**
FS %	39 ±4	37±6	P < 0.72
EF %	66.5±4.0	62.9±6.0	P < 0.091
E-A Ratio	0.86± 0.18	0.91±0.11	P < 0.49*

Table IV: Electrocardiographic Data

DATA	Group I (n=8)	Group II (n=37)	Statistical significance
Heart Rate (Exercise)	121±19	97±21	p<0.05*
Sokolow index mm	34±6	21.7±6	p<0.05*
Resting ECG			
Ischemic changes	87.6±19	65.7±9	p<0.08
Exercise ECG			
Ischemic changes	99.3 ± 17	73.8±6	p<0.001 **

(*) Significant (**) Highly significant

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Discussion:

The clinical end points of hypertensive heart disease identified above can be explained by structural, functional, and endothelial pathophysiological mechanisms that alter coronary hemodynamics and ventricular function. Among the structural alterations associated with hypertension are changes in coronary arterial flow that involve ventricular wall compression, luminal obstruction, and, of course, the increased wall thickening of the hypertensive arteriole that diminish the wall lumen ratio , that phrased by Hector. (22)

The purpose of this study was to compare a number of anatomic and functional characteristics between two groups of hypertensive patients with LVH, classified according to the presence (group I) or absence (group II) of myocardial ischemia changes at stress, so that ischemic changes would be attributable to the particular characteristics accompanying LVH in these patients. Our data indicate that the two groups did not differ in terms of age, sex, severity and duration of hypertension or smoking habits, but the group I patients, who had more pronounced LVH, were significantly more overweight Although, maximal systolic BP attained by effort tended to be higher in group I, and the systolic BP attained at peak exercise on the treadmill was significantly higher. A significant relation between LV and systolic blood pressure.

Our study revealed that, group I patients; those with myocardial ischemia changes at stress had a significantly greater degree of LVH by both ECG and echocardiographic criteria. Bosy et al reported about that, changes in the architecture of the hypertrophied heart exacerbate the imbalance between energy expenditure and energy production.(23) As well, Dirk et al reported in his study that, changes include an increase in the distance between capillaries, resulting in under perfusion and diminished diffusion of oxygen, as well as a decrease in coronary reserve. (24) The greater LVM of these patients was associated with overall more cardiac enlargement. However, the thickening of various cardiac structures, such as the interventricular septum and posterior wall, was no different between groups

Two different patterns of LVH (eccentric and concentric) were described several years ago and depend on the type of hemodynamic load, that what demonstrated by Akintunde et al, (25) and Richard et al reported in their study that , Concentric hypertrophy, with symmetrical thickening of the LV wall but no enlargement of the chambers, was considered to be typical of hypertension. (26) This was evidently the pattern of our patients. However, the patients of group I had concentric hypertrophy pattern, which would

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appear to represent a more advanced or severe stage of hypertensive LVH. Because the apical area of left ventricle supplies most of the force for contraction, it would also have the greatest energy demand and hence sustain a more pronounced deficit under conditions of relative hypo perfusion, which might explain the ischemic changes under stress in these patients. In the resting state, our patients exhibited essentially normal fractional shortening and ejection fraction, indicating normal overall systolic capacity (although some degree of diastolic dysfunction was present in all of them, as indicated by an E-A wave ratio <1). However, under stress, such patients have been described to exhibit impaired LV functional reserve with a lesser increase in ejection fraction , that where proved by Barry and Walter in their study about preserved ejection fraction .(27) Edward revealed that ,Interestingly, other studies in hypertensive with angina and normal coronary angiograms have suggested that reduced coronary reserve is also the result of LVH, (28) whereas others suggested that it may be a characteristic of the hypertensive state per se and not necessarily correlated with LV mass, that reported by Frohlich (29)

An important consequence of LVH is electrophysiological instability for Indeed. André showed that, several studies have documented the association between LVH and cardiac arrhythmias including complex arrhythmias and runs of ventricular tachycardia, which probably account for the long-known higher incidence of sudden death in such patients. (30)

Conclusions:

Left ventricular hypertrophy is a potent independent predictor of cardiovascular events particularly in association with hypertension were are both a supplementary factor of risk for ischemic changes mainly for obesity and elevated systolic blood pressure which were predominant among the sample of our study.

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