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Investigation of the frequency of left ventricular hypertrophy in hypertensive patients by the hospital anxiety and depression scale

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Abstract

The development of left ventricular hypertrophy (LVH) in patients with hypertension increases the frequency of ventricular arrhythmias and sudden death. It is known that psychiatric conditions such as anxiety and depression are associated with hypertension (HT) and other cardiovascular diseases. HT patients were assessed using the Hospital Anxiety and Depression Scale (HADS). Sociodemographic and laboratory data of the patients were recorded. LVH was seen in 34 of 103 patients, and LVH was not seen in 69 patients. The mean age was 47.8 years in the group with LVH and 49.5 in the group with non-LVH. According to the results of our study, there was no significant difference between the LVH group and the Non-LVH group in terms of age, gender, BMI, smoking, loneliness status, and EF. The most striking result of our study was that the HADS score was significantly higher in the group with LVH than in the group with Non-LVH ($p<0.001$). The glucose level was significantly higher in the group with LVH than in the group with non-LVH ($p<0.001$). Patients should be aware of adverse cardiac outcomes according to their anxiety level, and precautions should be taken while performing mental health interventions and treatments. In order to better explain the causes and pathophysiology that may lead to LVH in anxiety disorders and depression, more comprehensive and molecular-level investigations are required.

Keywords: Hypertension, anxiety, depression

Introduction

Hypertension (HT) is a prevalent disease that poses a considerable risk of cardiovascular death, coronary heart disease, and stroke. Left ventricular hypertrophy (LVH) may increase the risk of ventricular arrhythmias and sudden cardiac death due to HT [1]. The importance of mental and social factors in cardiovascular diseases has received extensive attention over the years. Given that anxiety generates symptoms and reactions that can increase blood pressure, potential relationships between anxiety and HT are being investigated [2].

It is thought that anxiety disorders seen in patients with HT could possibly be the determinant of the development of HT [3]. Earlier studies have shown that anxiety is related to nighttime and early morning HT and impaired circadian rhythm [4,5]. Elevated left ventricular mass index has been observed more frequently in some HT patients with anxiety disorders [6]. The effect of anxiety on the pathogenesis and LVH of HT is not entirely clear. Anxiety can cause high blood pressure and LVH by changing the basal autonomic nervous system and stress state [7-10].

In this study, we evaluated the level of anxiety evaluated by the hospital anxiety depression scale (HADS) in stage 1 HT (systolic 140-159 mmHg diastolic 90-99 mmHg) patients diagnosed with

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ambulatory electrocardiographic (Holter) monitoring according to the diagnostic criteria of the International Society of HT (ISH) global HT practice guidelines [11]. We investigated the rates of LVH echocardiographically, thus showing that LVH is more common in patients with high levels of anxiety and depression. We aim to reduce cardiac mortality and morbidity with early treatment of these diseases by keeping these diseases at the forefront of the clinic.

Material and Methods

Study design

This study is cross-sectional and descriptive. This study was endorsed by Local Ethics Committee (Decision Date: 25/20/2022, Meeting Number: 7, Decision Number: 2022/7-56). Signed informed consent was gathered from the participants for this study. The study was executed in compliance with the Declaration of Helsinki. In this study, a total of 145 patients, who were admitted to the Cardiology outpatient clinic between March 2022 and November 2022, were monitored with Holter monitoring and were in the stage 1 hypertension class according to the ISH global HT practice guidelines, were scanned from the hospital archive records, and 103 of these patients were included in the study. The HADS scale was applied to these patients. Turkish reliability and validity study of the form was published by Aydemir et al. [12]. According to HADS, 0-7 was defined as normal, 8-10 as borderline anxiety, and 11 and above was defined as anxiety.

Echocardiographic examination was performed by two cardiologists. Transthoracic echocardiographic examinations (GE Vivid 7 or Vivid E9, GE Vingmed, Horten, Norway; ALOKA Prosound F75, Tokyo, Japan) were performed with a 3-6 MHz phase transducer in all cases. Standard two-dimensional echocardiography with Doppler examination was performed and measurements were obtained according to the guidelines of the American Society of Echocardiography. Left ventricular mass was measured by echocardiography and left ventricular mass index (LVMI) was calculated. LVH was defined as LVMI >115 g/m2 in men and >95 g/m² in women. Left ventricular systolic functions were recorded in the echocardiography reports of the patients. Among the patients screened in the archive, patients with secondary conditions that could lead to LVH were excluded from the study (e.g., aortic stenosis, chronic renal failure, aortic coarctation, myocarditis, severe heart valve insufficiency, aortic and mitral valve insufficiency, hypertrophic cardiomyopathy, severe coronary artery disease, those with severe valvular insufficiency, those with heart failure with ejection fraction (EF)<40, and athletes as cardiac hypertrophy may occur). The included patients were evaluated according to their LVH echocardiography report notes and were recorded as present (LVH+) or absent (LVH-). Sociodemographic data, duration of HT, smoking status, loneliness status, biochemistry, hemogram parameters, systolic echocardiographic functions, LVH presence, and body mass index (BMI) were documented. We compared the patients grouped according to HADS score according to the presence of LVH.

Statistical analysis

All data analysis was accomplished on the Mac version of SPSS version 26.0. Number (n) and percentage (%) were used for nominal data. Continuous data with a normal distribution were shown as mean±standard deviation (M±SD). Among the continuous data, those that did not fit the normal distribution were shown with the median (minimum-maximum). Nominal data were compared with the chi-square test. Continuous data with normal distribution were compared with the independent samples t-test and those not with the Mann-Whitney U test.

Results

In our study, LVH was seen in 34 of 103 patients, and LVH was not present in 69 patients. The comparison of sociodemographic and clinical features of the LVH group and the non-LVH group is shown in Table 1. The mean age was 47.8 years in the group with LVH and 49.5 in the group with non-LVH. According to the results of our study, there was no significant difference between the LVH group and the Non-LVH group in terms of age, gender, BMI, smoking, duration of HT, loneliness status, and EF. The most striking result of our study was that the HADS score was significantly higher in the group with LVH than in the group with Non-LVH ($p<0.001$). The average HADS score was 10 in the group with LVH, and the average HADS score was 6 in the non-LVH group. The comparison of the laboratory parameters of the LVH group and the non-LVH group is shown in Table 2. There was no significant difference between the groups regarding hemoglobin, albumin, leukocytes, platelets, AST, ALT, creatinine, sodium, potassium, and the lipid panel. However, the mean fasting blood glucose value in the group with LVH was found to be 132 mg/dl. The glucose level was significantly higher in the group with LVH than in the group with non-LVH ($p<0.001$).

Table 1. Comparison of Sociodemographic Features and Echocardiographic Parameters of Patients with HT According to LVH

	LVH(n=34) M±SD or n (%) or Median (Min-Max)	Non-LVH(n=69) M±SD or n (%) or Median (Min-Max)	P
Age	47.88±8.93	49.52±9.26	0.395[1]
Gender			0.236[2]
Female	19 (55.9)	30 (43.5)	
Male	15 (44.1)	39 (56.5)	
Smoking	11 (32.4)	26 (37.7)	0.596[2]
BMI, kg/m²	27 (18-37)	25 (20-36)	0.128[3]
HADS	10 (3-14)	6 (2-9)	<0.001[3]
Duration of HT, years	6.24±2.34	7.21±3.44	0.366[1]
Loneliness	6 (17.6)	6 (8.7)	0.183[2]
LVEF, %	55 (45-60)	55 (40-55)	0.185[3]
DM	11 (17.5)	8 (20)	0.846[2]

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HT, hypertension; LVH, left ventricular hypertrophy; BMI, body mass index; HADS, Hospital anxiety and depression scale; LVEF, left ventricular ejection fraction; DM, diabetes mellitus. [1]Independent t test was used. [2]Chi-square test was used. [3]Mann-Whitney U test was used. p<0.05 was accepted as statistically significant

Table 2. Comparison of Laboratory Parameters of Patients with HT According to LVH

	LVH(n=34) M±SD or Median (Min-Max)	Non-LVH(n=69) M±SD or Median (Min-Max)	P
Hemoglobin, mg/dL	12.8 (9.5-17.2)	13.6 (11.1-17.2)	0.386[2]
Albumin, mg/dL	3.8 (2.8-4.6)	3.8 (2.7-4.4)	0.189[2]
WBC, 10^3/µL	6.42 (3.13-9.66)	6.52 (3.53-12.42)	0.241[2]
Platelet, 10^3/µL	236.65±99.57	245.05±105.77	0.700[1]
Glucose, mg/dL	132 (68-452)	102 (53-220)	<0.001[2]
ALT, IU/L	15 (9-29)	13 (9-35)	0.800[2]
AST, IU/L	19.5 (11-44)	20 (11-44)	0.565[2]
Creatinine, mg/dL	0.8 (0.4-1.3)	0.86 (0.4-1.5)	0.189[2]
Na, mEq/L	138 (125-147)	138 (128-148)	0.543[2]
K, mEq/L	3.7 (3.2-4.5)	3.7 (3.2-4.5)	0.348[2]
LDL-C, mg/dL	120 (63-185)	105 (54-190)	0.175[2]
HDL-C, mg/dL	34 (21-65)	35 (24-75)	0.608[2]

HT, hypertension; LVH, left ventricular hypertrophy; ALT, alanine transaminase; AST, aspartate transaminase; Na, sodium; K, potasium; WBC, white blood cell; LDL-C, low-density cholesterol; HDL-C, high-density cholesterol. [1]Independent t test was used. [2]Mann-Whitney U test was used. p <0.05 was accepted as statistically significant

The comparison of males and females is shown in Table 3. Accordingly, there was no difference in sociodemographic and clinical parameters between males and females.

Table 3. Comparison of Sociodemographic Features and Echocardiographic Parameters of Patients with HT According to Gender

	Female (n=49) M±SD or n (%) or Median (Min-Max)	Male (n=54) M±SD or n (%) or Median (Min-Max)	P
Age	47.84±9.26	50.02±9.01	0.228[1]
Smoking	18 (36.7)	19 (35.2)	0.870[2]
BMI, kg/m²	25 (18-37)	26 (20-36)	0.900[3]
HADS	7 (2-12)	7 (3-14)	0.352[3]
Loneliness	6 (12.2)	6 (11.1)	0.858[2]
LVH	19 (38.8)	15 (27.8)	0.236[3]

HT, hypertension; BMI, body mass index; HADS, Hospital anxiety and depression scale; LVH, left ventricular hypertrophy. [1]Independent t test was used. [2]Chi-square test was used. [3]Mann-Whitney U test was used. p<0.05 was accepted as statistically significant.

The comparison of those with and without HADS scores above 7 is shown in Table 4. Accordingly, BMI was significantly higher in patients with HADS score above 7 (p=0.048). LVH was more common in patients with HADS scores above 7 (p<0.001).

Table 4. Comparison of Sociodemographic Features and Echocardiographic Parameters of Patients with HT According to HADS Score

	HADS Score ≥7 (n=63) M±SD or n (%) or Median (Min-Max)	HADS Score >7(n=40) M±SD or n (%) or Median (Min-Max)	P
Age	49.11±9.16	48.78±9.23	0.8571
Gender			0.4252
Female	28 (44.4)	21 (52.5)	
Male	35 (55.6)	19 (47.5)	
Smoking	23 (36.5)	14 (35)	0.8762
BMI, kg/m²	25 (20-36)	27 (18-37)	0.0483
LVH	16 (25.4)	18 (45)	<0.0012
Loneliness	6 (9.5)	6 (15)	0.3992

HT, hypertension; HADS, Hospital anxiety and depression scale; BMI, body mass index; LVH, left ventricular hypertrophy. 1Independent t test was used. 2Chi-square test was used. 3Mann-Whitney U test was used. p<0.05 was accepted as statistically significant

Discussion

The main result of our study is that LVH is more likely to occur in patients with HT who have a higher level of anxiety according to the HADS score. The present study is the first one in the field to examine the degree of anxiety and LVH with approved psychometric scales. The results align with studies on anxiety and HT.

There is a complex relationship between physical well-being and mental health. Anxiety and depression are proven to have various implications for physical functions. Since the autonomic nervous system regulates the cardiovascular system, emotional states can profoundly affect the cardiovascular system, including blood pressure [13].

Many researchers have studied psychological factors that may lead to HT [14]. Rutledge and Hogan found that people who experience psychological stress develop HT at a rate of 8% more often than those who experience minimal stress [3]. Myocardial hypertrophy and decreased diastolic dysfunction are frequently found in patients with HT. This result indicates the relationship between anxiety and myocardial hypertrophy in patients with HT. HT is also a significant risk factor for the development of cardiovascular disease. Considering that anxiety triggers responses and symptoms that can increase blood pressure, scientists have begun to explore its possible connection.

Various surveys around the world investigate the relationship between anxiety disorders and HT, and conflicting results have been obtained [15]. In a research focusing exclusively on men in

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Hong Kong, anxiety was associated with HT, but depression was not associated with HT [13]. In a study conducted in the USA, the relationship of HT with generalized anxiety disorder, major depressive disorder, and comorbid conditions was determined [16]. In a study conducted with a group of Danish patients with anxiety, a higher rate of HT was found in the anxious group compared to the general Danish population [17]. On the other hand, Wei et al. found that 12% of patients with HT had anxiety symptoms [2].

Conversely, Hildrum et al. reported an inverse relationship between high anxiety and HT in their cross-sectional and prospective study. In a study conducted on adult men in New York, Friedman et al. investigated the relationship between multiple psychological diseases and HT. They found no difference between regular or slightly elevated HT [18,19].

However, exactly how anxiety causes LVH is not clear. In 2014, De Gui Kong et al. investigated the relationship between anxiety disorder and plasma adrenomedullin and LVH and found that plasma adrenomedullin level was higher in those with anxiety. Adrenomedullin is a biologically active peptide regulated by normal body fluids and electrolytes. In addition, this molecule plays a role in heart and kidney functions, vascular cell proliferation, and cardiac remodeling. Therefore, this molecule is thought to play a role in the pathophysiology of LVH, as shown in a previous study [20].

A study by Ma LL et al. in 2008 showed that the 24-hour circadian rhythm is disrupted in anxiety disorder and HT. It was found that the systolic pressure drop that usually occurs at night in these patients disappeared by 84% and was not observed in diastolic pressure by more than 33%. High blood pressure at night can lead to increased cardiovascular load and peripheral vascular resistance, myocardial cell volume, and an increase in the synthesis of myocardial fibrous proteins and LVH [5]. As another reason, in a study by Esler et al. in 2000, it was thought that anxiety disorders cause dysfunction in the autonomic nervous system, especially by activating the sympathetic system in the chronic process, causing an increase in catecholamine release and, as a result, an increase in plasma adrenomedullin levels with deterioration of myocardial fibroblast secretion compensation [21].

In 2022, Ling Zhu et al. found an association between anxiety with increased LVH and transmural repolarization distribution. In their subgroup analysis, they found that anxiety was more common in males and that anxiety in males was associated with LVH. They attributed this to biological stress response differences. In another study, male salivary cortisol levels were found to be higher than that of females during psychological stress [22].

In this study, there was no significant difference between the genders regarding LVH. However, since there are studies in the literature in which LVH is seen more frequently in males than in females, it has given contradictory results with the literature. In addition, we could not find any difference between the sexes regarding HADS score, smoking status, BMI, and loneliness status, regardless of LVH.

The present study has several limitations. First of all, in the study of a single center, a single scale was used instead of serial scales for anxiety-depression scoring. Since our study's small sample size makes it challenging to generalize the results. It is not clear whether LVH is seen as secondary to the inflammatory process in diabetes since the glucose ratios in the patient group in the LVH group are significantly higher than in the Non-LVH group. It is known that diabetes also causes LVH. However, it is yet to be known whether the laboratory fasting glucose level obtained from the archive records of the patients was taken at the total 8-hour fasting or postprandial time, so this difference was thought to be insignificant. Therefore, there is a need for studies with larger samples in which non-diabetic patients are selected in terms of study design. One of the limitations of our study is that some laboratory findings, such as thyroid function tests, which are necessary for secondary causes that may affect the results in terms of LVH, were not performed.

Conclusion

In conclusion, we detected LVH more frequently in HT patients with high anxiety levels. In addition, although the duration of the disease was similar, HADS scores were found to be significantly higher in the group with LVH. Therefore, patients should be aware of adverse cardiac outcomes according to their anxiety level, and precautions should be taken while performing mental health interventions and treatments. In order to better explain the causes and pathophysiology that may lead to LVH in anxiety disorders and depression, more comprehensive and molecular-level investigations are required.

Patient informed consent :
Signed informed consent was gathered from the participants for this study.

Conflict of interests
The authors declare that there is no conflict of interest in the study.

Financial Disclosure
The authors declare that they have received no financial support for the study.

Ethical approval
This study was endorsed by Adıyaman University Non-Interventional Clinical Researches Ethics Committee (Decision Date: 25/20/2022, Meeting Number: 7, Decision Number: 2022/7-56).

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