Med. J. Cairo Univ., Vol. 90, No. 4, June: 1251-1259, 2022
www.medicaljournalofcairouniversity.net

Association of Postural Hypotension and Echocardiographic Parameters in Chronic Heart Failure Patients

GEHAN A. ELMOGHAZY, M.Sc.*; ESSAM M. MAHFOUZ, M.D.** and MAHMOUD M. ELRAYES, M.D.**

The Department of Cardiovascular Medicine, National Heart Institute and Faculty of Medicine, Mansoura University***

Abstract

Background: Orthostatic hypotension (OH) is a frequent concomitant disorder and a common co-morbidity in heart failure (HF). It has been associated with increased risk of mortality and CV morbidity. Prevalent OH may precede the incidence of heart failure (HF) and atrial fibrillation (AF).

Aim of Study: To assess postural hypotension in chronic heart failure patients and its association with clinical manifestations and echocardiographic parameters.

Patients and Methods: The study included 100 patients who presented to National Heart Institute with symptoms and signs of chronic heart failure from November 2018 to November 2019 and were subjected to thorough history taking, full examination for symptoms and signs of heart failure, 12 lead ECG, transthoracic echocardiography and 6MWT evaluation. Patients of the study were classified to two groups; Group A with OH and group B without OH.

Results: One hundred patients hospitalized for HF [mean age: 58.64±12.91 years; 55% women] were examined with conventional echocardiograms and active-standing test. Associations of cardiac remodelling parameters with the difference between supine and standing (after 3min) systolic/diastolic BP were examined. A total of 33 patients (33%) met conventional OH criteria; i.e. systolic/diastolic BP decreases by ≥20/10mmHg. In comparison between the two groups (Group A with OH and group B without OH), OH group was associated with greater left atrial volume [$p \leq 0.001$], and greater left ventricular mass [$p=0.001$].

Conclusion: It was clear that OH is associated with the presence of structural cardiac changes such as increased LVM and increased left atrial volume. These findings suggest that autonomic dysfunction promotes cardiac remodelling in HF.

Key Words: *Chronic heart failure – Orthostatic hypotension.*

Introduction

HEART failure (HF) is a global public health problem affecting an estimated 37.7 million people worldwide, with frequent hospitalizations, impaired quality of life, and shortened life expectancy **[1]**.

Orthostatic hypotension (OH) is a sign of cardiovascular (CV) autonomic dysfunction, which is typically caused by an impaired circulatory adaptation to the reduction of central blood volume that occurs in standing position **[2]**.

It has also been demonstrated that prevalent OH may precede the development of heart failure (HF) and atrial fibrillation (AF) **[3]**.

OH has been associated with increased risk of mortality and CV morbidity **[4]**.

The mechanisms behind these associations remain elusive, but studies have recently demonstrated that the presence of OH among middle-aged adults predicts the development of left ventricular hypertrophy (LVH) independently of traditional risk factors such as hypertension **[5]**.

Higher diurnal BP variability and supine (nocturnal) hypertension, both present in OH, may provoke intermittent bouts of increased afterload, leading to permanent end-organ damage, such as left ventricular hypertrophy, thereby paving the way to left ventricular diastolic dysfunction, increased risk of congestive heart failure, and myocardial ischemia **[2]**.

The prevalence of OH in patients with HF remains uncertain. Reported data vary from 8% to as much as 83% among elderly patients hospitalized due to HF exacerbation **[6]**. Moreover, data on relations between impaired orthostatic homeostasis and presence of structural cardiac abnormalities in patients diagnosed with HF are very sparse **[7]**.

In this study, we examined a consecutive series of patients hospitalized for HF with BP measurements during active standing and standard echocardiography. Orthostatic BP changes will be studied in relation to echocardiographic parameters of cardiac remodelling.

Correspondence to: Dr. Gehan A. Elmoghazy, The Department of Cardiovascular Medicine, National Heart Institute

Material and Methods

Patients:

The study included 100 patients who presented to National Heart Institute at Giza Governorate, Egypt with symptoms and signs of chronic heart failure and were subjected to thorough history taking, full examination for symptoms and signs of heart failure, 12 lead ECG, transthoracic echocardiography and 6MWT evaluation. Patients of the study were classified to two groups; Group A with OH and group B without OH. This study was carried out over a year from November 2018 to November 2019. Approval was granted from the University Institutional Review Board.

Inclusion criteria:

All patients admitted to our department presented by chronic heart failure either newly diagnosed or exacerbated chronic HF [heart failure with preserved ejection fraction (HFpEF), heart failure with reduced ejection fraction (HFrEF) & heart failure with mid-range ejection fraction (HFmrEF)]. The studied patients were divided according to BP assessment (supine and after 3minutes of standing) into two groups, group A included 33 patients with OH and group B included 67 patients without OH.

Exclusion criteria:

1- Inability to deliver oral or written consent.
2- Cerebrovascular stroke (recent and old).
3- Extra-pyramidal affection.

Methods:

Data was collected through:

- *Medical history focusing on:*

1- Age, sex, special habits (smoking).
2- History of ischemic heart disease or MI.
3- Diabetes mellitus (DM) defined as either self-reported diagnosis of type 2 diabetes or use of anti-diabetic medication or fasting plasma glucose (FPG) >7mmol/L **[8]**.
4- Hypertension defined as either systolic BP (SBP) ≥ 140 mmHg and/or diastolic BP (DBP) ≥90mmHg **[9]**.
5- History of medications (Diuretics, ACEIs, ARBs, beta blockers).
6- HF symptoms (dyspnea "NYHA classification", paroxysmal nocturnal dyspnea (PND), ankle swelling and fatigue).

- *Clinical Examination focusing on:*

1- Orthostatic change in blood pressure; i.e. systolic/diastolic BP decreases by ≥20/10mmHg (A manual or electronic cuff is typically used to record supine and after 3 minutes standing BP) **[10]**.
2- Signs of heart failure [pulmonary crackles, edema, gallop (S3 heart sound)].
3- Body mass index (BMI) was calculated as kilograms per square meter.
4- Body surface area was calculated according to the DuBois formula **[11]**.
5- 6-minute walk test (6MWT).

- *Electrocardiography (ECG):*

12 lead ECG was done for all patients in our study groups to detect the heart rate, atrial fibrillation (AF), any conduction abnormalities, any ST segment deviation, Q waves of previous STEMI and R wave progression.

- *Echocardiography:*

Conventional transthoracic echocardiography was performed to all patients using Philips [IE33 with a 1-5 MHz transducer (S5-1)]. Measurements were done according to the recommendations of the American Society of Echocardiography. For evaluation of:

1- Ejection fraction (EF%).
2- Interventricular septal diastolic diameter (IVSDd) (mm/m^2).
3- Left ventricular internal diastolic dimension (LVIDd) (mm/m^2).
4- Right ventricular internal diastolic dimension (RVIDd) (mm/m^2).
5- Posterior wall diastolic diameter (PWDd) (mm/m^2).
6- Left atrial (LA) volume (mm/m^2).
7- Right atrial (RA) volume (mm/m^2).
8- Left ventricular mass index (LVMI) (g/m^2).
9- Left Ventricular Diastolic Function (septal E/é ratio).

Statistical analysis:

Data were analyzed using the Statistical Package of Social Science (SPSS) program for Windows (Standard version 24). The normality of data was first tested with one-sample Kolmogorov-Smirnov test. Qualitative data were described using number and percent. Association between categorical variables was tested using Chi-square test while Fischer exact test was used when expected cell count less than 5. Continuous variables were presented as mean ± SD (standard deviation) for parametric data and median (min-max) for non-parametric data. The two groups were compared with Student *t*-test for parametric data and Mann

Whitney test for non-parametric data. Pearson correlation (parametric) and Spearman correlation (non parametric) were used to correlate continuous data.

Level of significance:

For all above mentioned statistical tests done, the threshold of significance is fixed at 5% level (p-value). The results were considered Significant when the probability value is less than 5% ($p<0.05$). The smaller the p-value obtained, the more significant are the results.

Results

Table (1): Baseline characteristics of the study population (n=100).

Demographic data	Total (n=100)
Age/years:	
Mean ± SD	58.64± 12.91
Min-Max	14-82
Sex:	
Male	45 (45%)
Female	55 (55%)
BMI:	
Mean ± SD	30.56±7.05
Smoking	42 (42%)
Hypertension	57 (57%)
DM	47 (47%)
IHD	53 (53%)
NYHA class:	
I	15 (15%)
II	32 (32%)
III	35 (35%)
IV	18 (18%)
PND	67 (67%)
HF signs	71 (71%)
Chest crackles	37 (37%)
Lower limb edema	58 (58%)
Gallop (S3)	59 (59%)
6MWT(m)	170 (50-550)
Median (Min-Max)	
Postural hypotension	33 (33%)
Drug therapy	96 (96%)
Diuretics	66 (66%)
MRAs	45 (45%)
ACEIs (or) ARBs	85 (85%)
Beta blockers	82 (82%)

The present study was carried out on 100 chronic heart failure patients. The mean age was 58.64 ± 12.91 years, 45 % were males, 55% were females and 33% met conventional OH criteria.

As regard the medical history, 47% were diagnosed with DM, 57% had arterial hypertension, 42% were smokers and 53% had ischemic heart disease.

As regard the clinical presentation, the majority of patients presented with NYHA class II & III (32 & 35%), 67% presented with PND, 37% had chest crackles, 58% had lower limb edema and 59% had gallop (S3) with mean 6MWT distance of 170 (50:550) m.

Revision of patient's medication history revealed that 96% were taking guidelines directed medical treatment for heart failure, 85% were on angiotensin converting enzyme inhibitors or angiotensin receptor blockers (ACEIs/ARBs), 66% were on diuretics, 45% were on mineralocorticoids receptors antagonists (MRAs) and 82% were on beta blockers.

Table (2) shows that patients with OH presented with more advanced HF symptoms & signs compared to patients without OH with statistically significant difference (p-value >0.05).

Table (3) shows that the majority of patients taking ACEIs (or) ARBs were in group B, 61 patients (91%) in group B compared to 24 (72.7%) in group A wit p-value 0.016.

Table (4) shows that AF was more common in patients with OH with statistically significant value (p-value was 0.001).

Table (5) shows that Patients with OH had worse LV systolic function, greater LV & RV dimensions, greater LA & RA volumes, increased LVMI & systolic PAP and diastolic dysfunction with statistically significant difference between the two groups (p-value was <0.05).

Table (2): Comparative analysis between the two groups (Group A with OH and group B without OH) as regard the clinical examination.

Variables	Postural hypotension (n=33)	No Postural hypotension (n=67)	Test of significance	*p*-value
NYHA class:				
I	0 (0%)	15 (22.4%)	χ^2=39.17	≤0.001 *
II	4 (12.1%)	28 (41.8%)		
III	13 (39.4%)	22 (32.8%)		
IV	16 (48.5%)	2 (3.0%)		
PND:				
Positive	33 (100%)	34 (50.7%)	χ^2=24.26	≤0.001 *
Negative	0 (0%)	33 (49.3%)		
HF signs:				
Positive	28 (84.8%)	43 (64.2%)	χ^2=4.59	0.032*
Negative	5 (15.2%)	24 (35.8%)		
Chest crackles:				
Positive	22 (66.7%)	15 (22.4%)	χ^2=18.59	≤0.001 *
Negative	11 (33.3%)	52 (77.6%)		
Lower limb edema:				
Positive	24 (72.7%)	34 (50.7%)	χ^2=4.38	0.036*
Negative	9 (27.3%)	33 (49.3%)		
Gallop:				
Yes	28 (84.8%)	31 (46.3%)	χ^2=13.60	≤0.001 *
No	5 (15.2%)	36 (53.7%)		
6MWT(m):				
Median (Min-Max)	80 (50-400)	200 (50-550)	Z=4.21	≤0.001 *

χ^2: Chi square test. Z: Mann Whitney test. *Significant $p \leq 0.05$.

Table (3): Comparative analysis between the two groups (Group A with OH and group B without OH) as regard the drug therapy.

Drug therapy	Postural hypotension (n=33)	No Postural hypotension (n=67)	Test of significance	*p*-value
Drug therapy:				
Yes	31 (93.9%)	65 (97.0%)	FET	0.597
No	2 (6.1%)	2 (3.0%)		
Diuretics	24 (72.7%)	42 (62.7%)	χ^2_2=0.993	0.319
MRAs	14 (42.4%)	31 (46.3%)	χ^2_2=0.132	0.716
ACEIs (or) ARBs	24 (72.7%)	61 (91.0%)	χ^2_2=5.82	0.016*
Beta blockers	24 (72.7%)	58 (86.6%)	χ^2=2.87	0.090

FET: Fischer exact test.

Table (4): Comparative analysis between the two groups (Group A with OH and group B without OH) as regard ECG.

ECG	Postural hypotension (n=33)	No Postural hypotension (n=67)	Test of significance	*p*-value
Atrial fibrillation	14 (42.4%)	9 (13.4%)	χ^2_2=10.49	0.001 *
Ischemic changes	22 (66.7%)	42 (62.7%)	χ^2_2=0.152	0.697
Pathological Q waves	7 (21.2%)	22 (32.8%)	χ^2_2=1.45	0.228
ST segment wave changes	19 (57.6%)	46 (68.7%)	χ^2_2=1.19	0.275
Conduction abnormalities	9 (27.3%)	10 (14.9%)	χ^2_2=2.19	0.139
LBBB	4 (12.1%)	4 (6.0%)	χ^2=1.14	0.286
RBBB	0 (0%)	1 (1.5%)	FET	1.00
IVCD	5 (15.2%)	5 (7.5%)	χ^2_2=1.45	0.228
Poor R wave progression	16 (48.5%)	22 (32.8%)	χ^2=2.29	0.130

Table (5): Comparative analysis between two groups (Group A with postural hypotension and group B without postural hypotension) as regard ECHO data.

Variables	Postural hypotension (n=33)	No Postural hypotension (n=67)	Test of significance	*p*-value
EF%: Mean ± SD	26.99±9.79	39.75±11.77	5.37	≤0.001*
RWMA	27 (81.8%)	54 (80.6%)	0.021	0.884
IVSDd (mm): Mean ± SD	10.12±2.47	10.42±2.67	0.532	0.596
PWDd (mm): Mean ± SD	9.23±3.19	9.81±2.70	0.943	0.348
LVIDd (mm): Mean ± SD	66.97±11.85	56.19±9.09	5.03	≤0.001*
RV basal diameter: Mean ± SD	50.96±11.30	38.64±11.28	5.12	≤0.001*
RV mid diameter: Mean ± SD	38.49±12.10	29.19±8.24	4.52	≤0.001*
RV longitudinal diameter (mm)	80.33±12.00	71.43±9.92	3.93	≤0.001*
LA volume (ml/m ^{2}): Median (Min-Max)	62.3 (28.1-123.1)	35.6 (12-79)	5.46	≤0.001*
RA volume (ml/m ^{2}): Median (Min-Max)	50.7 (15.58-127)	23 (10-75)	4.43	≤0.001*
LVMI (g/m ^{2}): Median (Min-Max)	146.1 (88.11-310.4)	124.8 (62.4-316.8)	3.19	0.001*
Diastolic function (Septal E/É)	16.25 (8.1-45.5)	11.81 (4.83-29.29)	2.49	0.013*
SPAP (mm Hg): Mean ± SD	41.88±16.75	35.38±13.15	2.12	0.037*

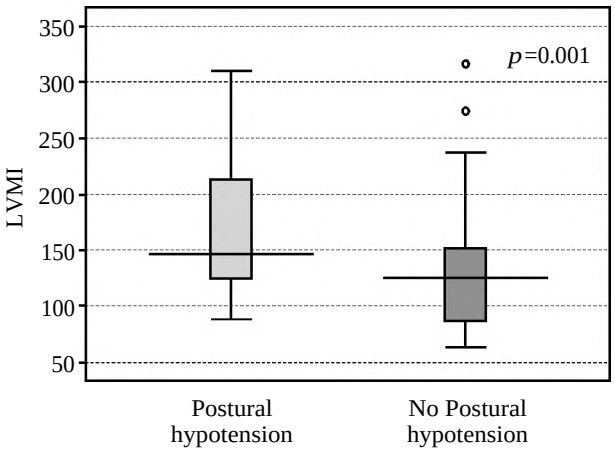

Fig. (1): Box plot for median LVMI (g/m ^{2}) among postural hypotension and non-postural hypotension groups.

Discussion

In the current study population, heart failure was more prevalent in middle ages with mean age ± SD (58.64±12.91), mean BMI was (30.56±7.05), more common in females (55%) than males (45%). There was high prevalence of many co-morbidities including diabetes mellitus in (47%) of patients, hypertension (57%) and ischemic heart disease (53%).

Scholfield et al., and Al-Omary et al., studies showed that increased age is common in heart failure patients with mean age 76.3 years, elevated incidence of heart failure in male patients more than females (50-75%). Increased incidence of diabetes mellitus reaching (28-31%), hypertension (40-50%) and ischemic heart disease (45-50%). **[12]**.

The younger age of our patients with heart failure than the other studies may be related to more severe causes of heart failure as ischemic heart disease and cardiomyopathy that may affect more patients in their middle age, and also may be related to higher mortality in our patients with HF due noncompliance to guidelines documented treatment or other HF interventional therapy. Also, the small sample size of the study may have a role.

In the current study, smoking is common in patients with heart failure representing (42%) of patients.

Al Faleh et al., study showed that patients with heart failure had history of smoking in (17.9%) of patients **[13]**.

In the current study, 33% of all heart failure patients met conventional OH criteria (systolic/diastolic BP decreases by ›_20/10mmHg).

Fedorowski et al., study demonstrated that the proportion of individuals who meet the diagnostic criteria of OH increased with chronic conditions associated with increased risk of CV events, such as heart failure. OH prevalence in heart failure was (8%-83%) **[14]**.

In the current study population, the most common clinical presentations were dyspnea with NYHA functional class II & III affecting 32 & 35% of patients, 67% of patients presented with PND, 37% had chest crackles, 58% had lower limb edema and 59% had gallop (S3).

Abdellah et al study is consistent with our results showing that dyspnea was the most prevalent clinical presentation in (91.7%) of patients followed by lower limb edema (54.2%) **[15]**.

Karaye et al., and Alhabib et al., studies demonstrated that electrocardiographic changes are prevalent in heart failure patients about (98.2%). The most common arrhythmia was atrial fibrillation found in (15.9%) of patients, wide QRS complexes in (15%) of patients **[16,17]**.

In the current study groups, electrocardiographic changes in patients with heart failure were common. Atrial fibrillation was described in (23%) of patients, 64% had ischemic changes, 29% had pathological Q waves, 65% had ST segment & T wave changes, 38% had poor R progression and 19% had conduction abnormalities in the form of LBBB, RBBB & IVCD (8, 1 & 10%; respectively).

In the current study, patients were divided into two groups, orthostatic and non-orthostatic group, with no statistically significant difference as regard the age and sex in contrast to Zimmermann et al., study which showed that orthostatic hypotension increased with aging **[18]**.

The non-significant correlation between OH & age in the current study in contrast to other studies may be related to the small sample size and the limited variability in the age in the study group.

In the current study, OH patients presented with more advanced HF symptoms & signs, including (NYHA class, PND, chest crackles, lower limb edema, gallop & 6MWT).

Gorelik et al., study demonstrated that higher NYHA grade and especially non-ischemic etiology of HF predicted the appearance of OH **[19]**. On the opposite side, Potocka-Plazak et al., study showed that the severity of HF according to NYHA functional class was not associated with the presence of OH **[20]**. The contradictions between the studies in the associations between OH and HF severity may be related to higher prevalence of multiple chronic comorbidities and polypharmacy.

In the current study, the majority of patients (91%) in the orthostatic group were taking ACEIs (or) ARBs revealing that there is positive correlation between OH and ACEIs (or) ARBs. This is in agreement with Gorelik et al., study which demonstrated that the addition of captopril to chronic treatment with diuretics in HF patients was associated with development of OH **[6]**.

The possible mechanism of increased risk of OH with ACEIs (or) ARBs is blockade of aldosterone secretion by ACE inhibition with subsequent decreased sodium retention and intravascular volume. Furthermore, at upright posture, the compensatory rise of systemic vascular resistance, which accompanies peripheral pooling, is blocked with ACE inhibitors as stated by Gorelik et al., study **[6]**.

In the current study atrial fibrillation was more common among OH patients. This may be explained by several explanations linking OH and cardiovascular diseases, including AF. First, OH may provoke intermittent ischemic bouts and increase afterload leading to LVH. As a result of postural BP swings and a tendency towards supine (and nocturnal) hypertension, OH can lead to LVH which is regarded one of the main structural determinants of AF. Moreover, an altered autonomic tone, in response to a decrease in BP on standing, may directly modulate the activity of the sinus node and lead to episodes of paroxysmal AF **[14, 21]**.

Min et al study confirmed that OH may predict a high risk of developing a series of cardiovascular diseases, including AF **[22]**, and Fedorowski et al study demonstrated that the presence of OH predicts the long-term incidence of AF in middle-aged predominantly asymptomatic individuals, independently of conventional risk factors, but this association seems to be limited to hypertensive individuals **[23]**.

In the current study, we have demonstrated that prevalent OH may lead to the development of structural and functional changes in the heart, as

it was associated with greater LVMI especially in atrial fibrillation patients.

It is well known that higher diurnal BP variability and nocturnal hypertension, both present in OH correlate with target-organ damage, specifically with LVH. The mechanism of LVH development may not only involve the supine afterload enhancement but also a periodic vasoconstrictor surge in response to orthostatic BP instability. Thus, the up regulated neuroendocrine mechanisms such as sympathetic, renin-angiotensin, and endothelin systems may ultimately provoke compensatory increase of LVM through their direct action on the heart or, indirectly, through their action on the vasculature as stated by Magnusson et al., study [24].

In the current study, OH was associated with impaired diastolic function (Septal E/É). This in agreement with Magnusson et al., study which showed that OH predicts development of diastolic dysfunction in patients without diabetes **[24]**.

The mechanism behind this association may be attributed to the abnormal catecholamine response in patients with OH due to the autonomic failure syndrome, where OH depends on a deficient sympathetic reflex vasoconstriction due to suppressed catecholamine release. Impaired beta-adrenergic signaling is well established in diastolic dysfunction. Moreover, diastolic dysfunction has been associated with reduced chronotropic, systolic, and lusitropic reserve, findings that further stress a possible role of impaired beta-adrenergic signaling. Finally, OH has been associated with increased arterial stiffness and since earlier studies have implicated an association between increased arterial stiffness and higher prevalence of diastolic dysfunction, reduced arterial compliance might mediate the increased risk of diastolic dysfunction in OH patients seen in our study **[24]**.

In the current study, OH patients had greater RV size. In the opposite side, Magnusson et al., study demonstrated smaller RV size among OH patients which is considered more logical than our finding. In patients with OH the venous return mechanisms are impaired, and the RV preload during daytime (in upright position) may be distinctly reduced. It should be noted that the echocardiographic measurements were performed in supine position, and, accordingly, the orthostatic RV preload reduction could not influence these results. Thus, the decreased RV volume in OH is rather a chronic state, which is independent of body position [25].

The greater RV size in OH patients may be related to the presence of more advanced cases of bi-ventricular failure, and our findings of greater RA volume and pulmonary hypertension in the same cases may enforce this theory.

In the current study, OH was associated with increased left atrial volume. This is in agreement to Ali et al., study and may be related to prevalent AF among OH patients **[7]**.

In the current study, OH was associated with poorer LV systolic function & dilated LV internal dimension. Data on relation between OH & LV systolic function are scares except for Ali et al., study which observed that LVEF was inversely associated with LVM, and this may need more dedicated studies **[7]**.

Conclusion:

Orthostatic hypotension (OH) is associated with the presence of structural cardiac changes such as increased LVM and increased left atrial volume. These findings suggest that autonomic dysfunction promotes cardiac remodelling in HF.

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العلاقة بين انخفاض ضغط الدم الوضعى ومؤشرات تخطيط صدى القلب فى مرضى قصور القلب المزمن

يعتبر قصور القلب (HF) مشكلة صحية عامة عالمية تؤثر على ما يقدر بـ ٣٧.٧ مليون شخص فى جميع أنحاء العالم، مع التأثير على ممارسة المريض لحياته اليومية، تكرار دخول المستشفى، ونقص العمر المتوقع.

هبوط القلب هو متلازمة سريرية تتميز بالأعراض النموذجية (مثل ضيق التنفس وتورم الكاحل والتعب) التى قد تكون مصحوبة بعلامات (مثل ارتفاع ضغط الوريدى الوداجى والإرتشاح الرئوى) الناجمة عن خلل بنائى أو وظيفى فى القلب، مما يؤدى إلى انخفاض فى النتاج القلبى و/أو ارتفاع الضغوط داخل القلب أثناء الراحة أو أثناء الإجهاد.

قصور القلب يعُد سبب رئيسى لوفيات القلب والأوعية الدموية فى جميع أنحاء العالم.

هناك علاقة وثيقة بين التغييرات التى تحدث فى ضغط الدم الإنقباضى (SBP) ومضاعفات ووفيات أمراض القلب والأوعية الدموية فى مرضى قصور القلب المزمن (HF).

انخفاض ضغط الدم الوضعى (OH) يتميز بانخفاض غير طبيعى فى ضغط الدم (BP) فى وضع الوقوف. وهو علامة على خلل وظيفى فى القلب والأوعية الدموية (CV)، والذى يحدث عادة نتيجة قدرة الدورة الدموية على التكيف مع انخفاض حجم الدم المركزى الذى يحدث فى وضع الوقوف.

انخفاض ضغط الدم الوضعى (OH) كثيراً ما يصيب كبار السن والمرضى الذين يعانون من أمراض عصبية أو مرض السكرى أو ارتفاع ضغط الدم. تتراوح نسبة انتشاره من ٥٪ فى المرضى <٥٠ سنة من العمر إلى ٣٠٪ فى >٧٠ سنة من العمر.

انخفاض ضغط الدم الوضعى (OH) يزيد من خطر الإصابة بالأمراض القلبية الوعائية (CVD) ومعدل الوفيات. يختلف انتشار OH فى المرضى الذين يعانون HF من ٨٪ إلى ما يصل إلى ٨٣٪ بين المرضى كبار السن.

يرتبط الإنخفاض الإنتصابى فى ضغط الدم الإنقباضى (SBP) بين مرضى قصور القلب المزمن (HF) كبار السن مع وجود تغييرات هيكلية فى القلب مثل زيادة كتلة البطين الأيسر(LVM) وزيادة حجم الأذين الأيسر. هذه النتائج تشير إلى أن الخلل اللاإرادى يعزز إعادة تشكيل القلب فى مرضى قصور القلب المزمن (HF).

الهدف من الدراسة : الهدف من هذه الدراسة هو تقييم انخفاض ضغط الدم الوضعى لدى مرضى قصور القلب المزمن وارتباطه بالأعراض الإكلينيكية مؤشرات تخطيط صدى القلب.

خطوات الدراسة : شملت الدراسة مئة مريضاً تم ادخالهم بأعراض وعلامات هبوط القلب المزمن بقسم أمراض القلب والأوعية الدموية بمستشفى معهد القلب القومى.

تم أخذ التاريخ المرضى الكامل وكذلك الفحص السريرى الشامل خاصة علامات ضعف عضلة القلب وقياس ضغط الدم والتغير الانتصابى فى قياس ضغط الدم.

تم عمل رسم القلب الكهربائى والموجات فوق صوتية على عضلة القلب.

النتائج : المرضى الذين يعانون من هبوط القلب المزمن كانوا أصغر سناً من أى دراسات أخرى.

انتشار انخفاض ضغط الدم الوضعى بين مرضى هبوط القلب المزمن بنسبة تصل إلى ٣٣٪.

لوحظ زيادة نسبة انتشار الذبذبة الأذينية بين مرضى انخفاض ضغط الدم الوضعى بنسبة تصل إلى ٤٢٪ فيما وجدت نسبة انتشارها فى باقى المرضى ١٤.٣٪.

لوحظ وجود علاقة طردية بين انخفاض ضغط الدم الوضعى وحدة أعراض هبوط القلب.

لوحظ أن وجود انخفاض ضغط الدم الوضعى يتزامن مع وجود تغيرات بناتية فى عضلة القلب كزيادة كتلة البطين الأيسر وزيادة حجم الأذين الأيسر مما يدعم فرضية أن خلل الجهاز اللاارادى يعزز إعادة التشكيل البنائى للقلب فى مرضى هبوط القلب المزمن.