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Impact of Hyperuricemia on Left Ventircular Function in Hypertensive Patients Presented to Ain Shams University Hospitals

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Abstract

Background: The association between serum uric acid and cardiovascular diseases has long been recognized.

Aim of Study: The aim of study was to assess the left ventircular function in hypertensive patients with or without hyperuricemia.

Patients and Methods: This prospective, comparative, cross sectional study involved 300 patients coming to cardiology the clinic for clinical assessment. The study was performed in the Cardiology Department, Faculty of Medicine, Ain Shams University Hospital at the during period from July 2022 to March 2023.

Results: The results of the study showed a significant difference in the tie index among the three groups (Hypertension with Hyperuricemia, Hypertension without Hyperuricemia, and Control). The median tie index was highest in the Hypertension with Hyperuricemia group (0.46) followed by the Hypertension without Hyperuricemia group (0.42) and was lowest in the control group (0.37). The difference in tie index between the groups was statistically significant with a *p*-value of <.001.

Conclusion: This study revealed that hyperuricemia is widespread in our study population with systemic hypertension and both are positively correlated. Hyperuricemia was associated with LVH. Thus, the study recommends a repetitive evaluation of serum UA in all hypertensive patients. As it suggests that hyperuricemia is an early marker of increased left ventricular mass that can be used to identify a hypertensive population with cardiac TOD.

Key Words: *Uric acid – Uricemia – Hypertension – Echocardiography – Left ventricular function.*

Introduction

THE association between serum uric acid and cardiovascular diseases has long been recognized **[1]**. However, it has not been definitively established whether serum uric acid is merely a marker for risk or a causative agent in cardiovascular diseases, or whether treatment targeting serum uric acid levels affects outcomes **[1]**.

The relationships between hyperuricemia and other cardiovascular risk factors including hypertension, hyperinsulinemia, reduced physical activity, increased body mass index (BMI), and decreased high density lipoprotein cholesterol (HDL) are demonstrated in many clinical studies **[2,3]**. Factors which are related to hyperuricemia are associated with the established risk factors for many cardiovascular diseases. The independence of uric acid association from other confounding factors has remained controversial **[4]**.

Although the mechanisms by which uric acid may play a pathogenetic role in cardiovascular diseases is unclear, hyperuricemia is associated with deleterious effects on endothelial function, oxidative metabolism, platelet adhesiveness, and aggregation **[5]**.

Although the echocardiographic examination is usually recommended as a second-line study in the evaluation of hypertensive patients, it is one of most commonly used imaging modality and has given insights into pathophysiology and clinical implications in patients with hypertension. It can detect anatomical and functional changes easily in a real-time, quick, and reproducible manner **[6]**.

The contributory effect of hyperuricemia to myocardial impairment produced by hypertension has not been clarified yet. In this respect, this study is designed to assess the LV systolic and diastolic functions in patients with hypertension with or without hyperuricemia. Tissue Doppler imaging will be used for detailed analysis.

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Aim of study:

The aim of study was to assess the left ventircular function in hypertensive patients with or without hyperuricemia.

Patients and Methods

This prospective, comparative, cross sectional study involved 300 patients coming to cardiology clinic for clinical assessment. The study was performed in the Cardiology Department, Faculty of Medicine, Ain Shams University Hospital at the period from July 2022 to March 2023.

Inclusion criteria:

The study included 300 patients that were divided into three groups: Group A: Consisted of 125 hypertensive patients with hyperuricemia. Group B: Consisted of 125 hypertensive patients without hyperuricemia. Group C: Consisted of 50 age and sex matched healthy control participants.

The control participants had normal physical examination, normotensive, normal transthoracic echocardiography and with no cardiovascular or any other organ system disease.

Arterial hypertension is defined as a BP values ›¯ 140mmHg and/or diastolic BP values ›¯ 90mmHg on repeated measurements or receiving antihypertensive treatment **[7]**.

Hyperuricemia is defined as serum uric acid level ›¯ 7mg/dL (in men) or ›¯ 6.0mg/dL (in women) **[8]**.

Exclusion criteria:

Patients with any of the following criteria were excluded from this study: History of diabetes mellitus (Diabetes mellitus was defined according to the recommendation of 2019 American Diabetes Association as fasting plasma glucose ›¯ 126mg/dl or random plasma glucose ›¯ 200mg/dl or HbA1c concentration of ›¯ 6.5% or receiving treatment of diabetes) **[9]**. History of Heart failure with reduced left ventricular systolic function, known Ischemic heart disease, significant valvular heart disease, chronic obstructive heart disease (COPD) (The criteria for symptoms of COPD were chronic cough, dyspnea (on exertion, progressive over years) and expectoration with lifelong smoking history), obesity. (The body mass index (BMI) was defined as a person's weight in kilograms divided by the square of the person's height in meters (Kg/m^2). An adult who had a BMI of 30 or higher was considered as obese **[10]**, Age >60, left ventricular hypertrophy due to other causes: valve disease, hypertrophic cardiomyopathy (HOCM), and congenital heart disease, patients on uricosuric drugs that is either primary such as allopurinol, sulfinpyrazone, probenecid and colchicine or secondary uricosuric drugs such as fenofibrate, adrenocorticotrophic hormone and cortisone and hypertensive patients on hydrocholorothiazide therapy.

Methods:

Ethical considerations: The study was conducted after approval of the protocol by the Local Research Committee & the Studies Committee as well as the Research Ethics Committee.

Complete assessment was done for each patient including:

Full history taking: Personal history: Name, age, sex, residence and occupation. Past history: History of previous medical illness including cardiovascular risk factors e.g. hypertension, diabetes mellitus, smoking and their durations, history of previous surgical intervention and anesthesia and history of previous medications.Present history: Patient's first complaint, onset of the disease, current medications, duration of the disease and its progression.

Clinical examination: General examination including body mass index, heart rate and blood pressure.

Laboratory work-up:

Venous blood samples were collected, under aseptic condition, 10cm from each patient and healthy control subject.

Serum samples were arranged for the following tests: Serum uric acid, serum creatinine, lipid profile including: Total serum cholesterol, triglycerides, low-density lipoprotein cholesterol (LDL) and high-density lipoprotein cholesterol (HDL) and fasting blood glucose - HbA1c

Serum uric acid levels were determined with end point enzymatic PAP (Uricase-PAP) method on automated chemistry analyzer (Flexor EL 200, Elitech, Paris, France) using Elitec AUML-0250 kits.

Transthoracic echocardiographic examination: All participants underwent complete transthoracic echocardiographic studies including two-dimensional (2D), color flow, and spectral Doppler as well as TDI with a GE Vivid 7 system (GE-Vingmed Ultrasound AS, Horten, Norway) using a 2.5-3.5-MHz transducerwith simultaneous ECG signal recorded. All examinations were performed with the patients in the standard left lateral position. 2D images were acquired with a frame rate between

40 and 60/s, obtained during breath hold and saved in cine-loop format from three consecutive beats. Gray-scale images and conventional blood-pool Doppler data were acquired in standard planes from parasternal and apical windows **[11]**.

A comprehensive echocardiographic study to all patients including:

2DE-giuded m mode approach measurments:left ventricular (LV) systolic function (ejection fraction), aortic annulus (Ao) and left atrium diameter (LA) were measured.

Cardiac chamber quantifications: Left atrial (LA) volume index (LAVI) and LV mass (LVMI) were measured.

Doppler measurments:

1- PW Doppler measurments: A sample volume (length of the sample volume was 5 or 10mm) was placed at the tips of the mitral leaflets to get the left ventricular inflow wave forms from the apical four chamber view. The following parameters were obtained: Peak Transmitral E-wave velocity, A-wave velocity and E/A ratio **[12]**.

2- Tissue Doppler measurements were performed Guided by the two-dimensional four-chamber view, a sample volume (length of the sample volume was 5 or 10mm) was placed on the mitral lateral annulus. The following parameters were obtained: Peak systolic (S'), peak early (E') and late (A') diastolic annular velocities, E'/A' ratio, isovolumetric relaxation time (IVRT), isovolumetric contraction time (IVCT), left ventricular ejection time (LVET) and we calculated Tie index by the formula (IVCT+IVRT)/ET.

For assessment of diastolic function, ECAVI recommends using four variables (mitral annular E´ velocity, average E/E´ ratio, peak of tricuspid regurge (TR) velocity and left atrium (LA) volume index.

Statistical analysis: The collected data were coded, tabulated, and statistically analyzed using IBM SPSS statistics (Statistical Package for Social Sciences) software version 28.0, IBM Corp., Chicago, USA, 2021. Quantitative data tested for normality using Shapiro-Wilk test, then described as mean ± SD (standard deviation) as well as minimum and maximum of the range, and then compared using one way ANOVA test. Qualitative data described as number and percentage and compared using Chi square test as well as Fisher's exact test for variables with small expected numbers. Spearman correlation coefficient was used to correlate between both uric acid, tie index and other parameters. The level of significance was taken at p-value <.050 was significant, otherwise was non-significant.

Results

The study was conducted on hypertensive patients who were attending the outpatient clinic of Ain Shams University Hospital during the period from June 2022 to March 2023 the study included 300 participants who were divided into three groups: Group A: Consisted of 125 hypertensive patients with hyperuricemia. Group B: Consisted of 125 hypertensive patients without hyperuricemia. Group C: Consisted of 50 healthy control participants.

The results of the table show that there was no significant difference in age among the three groups. However, there was a significant difference in gender distribution, with a higher percentage of males in the group with hypertension without hyperuricemia (52.0%) compared to the group with hypertension and hyperuricemia (36.8%) and the control group (44.0%).

The Uric acid levels were significantly higher in the Hypertension with Hyperuricemia group (7.65±0.61mg/dl) compared to the Hypertension without Hyperuricemia (4.49 ±0.53mg/dl) and Control (4.16±0.52mg/dl) groups (p<.001 for both). The Serum creatinine levels were not significantly different between the three groups (p=.136). The Fasting Blood Glucose levels were also not significantly different between the three groups (p=.061).

The results show that there were significant differences in most of the variables studied among the groups. The IVS thickness diastole was significantly higher in the hypertension with hyperuricemia group compared to the hypertension without hyperuricemia and control groups (p<.001 and p=.019, respectively). Similarly, the LV posterior wall thickness diastole was significantly higher in the hypertension with hyperuricemia group compared to the hypertension without hyperuricemia and control groups (p<.001 and p=.473, respectively).

The diastolic function was normal 28.3%, while 48.7% had grade I diastolic dysfunction, and 23.0% had grade II diastolic dysfunction. No participant was diagnosed with grade III diastolic dysfunction.

The results of the study showed a significant difference in the tie index among the three groups

(Hypertension with Hyperuricemia, Hypertension without Hyperuricemia, and Control). The median tie index was highest in the Hypertension with Hyperuricemia group (0.46) followed by the Hypertension without Hyperuricemia group (0.42) and was lowest in the control group (0.37). The difference in tie index between the groups was statistically significant with a *p*-value of <.001.

The results of the study showed a significant difference in left ventricular mass index (LVMI) among the three groups (Hypertension with Hyperuricemia, Hypertension without Hyperuricemia, and Control). The LVMI was highest in the Hypertension with Hyperuricemia group (118.80 ±24.92) followed by the Hypertension without Hyperuricemia group (107.98 ±14.95) and was lowest in the control group (77.38 ± 13.76). The difference in LVMI between the groups was statistically significant with a *p*-value of <.001.

The results indicate a strong positive correlation between uric acid levels and IVS thickness diastole (*r*-value=0.917), LV end diastolic diameter (*r*-value=0.532), LV posterior wall thickness diastole (*r*-value=0.508), left atrium internal diameter (*r*-value=0.720), peak E velocity (*r*-value=0.424), peak TR velocity (*r*-value=0.348), LA volume index (*r*-value=0.626), Tie index (*r*-value=0.840), LV mass index (*r*-value=0.652), E/E" (*r*-value= 0.455), and IVRT (*r*-value=0.695). These correlations are all statistically significant (*p*-value <.001).

Table (1): Demographic characteristics of the three groups.

Variables	Groups						*p*-value
	Hypertension with Hyperuricemia (n=125)		Hypertension without Hyperuricemia (n=125)		Control (n=50)		
Age (years): Mean ± SD.	43.44±3.38		42.29±2.70		41.24±2.34		0.050
Gender:							
Male	46	36.8%	65	52.0%	22	44.0%	<0.001*
Female	79	63.2%	60	48.0%	28	56.0%	

SD: Standard deviation. *p-value significant if <.05.

Table (2): Laboratory data of the three groups.

Variables Mean ± SD.	Groups			*p*-value
	Hypertension with Hyperuricemia (n=125)	Hypertension without Hyperuricemia (n=125)	Control (n=50)	
Uric acid (mg/dl):	7.65±0.61	4.49±0.53	4.16±0.52	<.001*
p-value$_1$		<.001*	<.001*	
p-value$_2$			.002*	
Serum creatinine (mg/dl)	0.96±0.16	0.94±0.65	0.81 ±0.08	.136
Triglyceride (mg/dl):	125.27± 15.48	91.44±9.42	74.80± 11.92	<.001*
p-value$_1$		<.001*	<.001*	
p-value$_2$			<.001*	
Cholesterol (mg/dl):	253.53±33.90	137.02±25.16	131.42±22.00	<.001*
p-value$_1$		<.001*	<.001*	
p-value$_2$			.503	
LDL (mg/dl):	107.26±9.68	83.46± 13.21	66.94±7.56	<.001*
p-value$_1$		<.001*	<.001*	
p-value$_2$			<.001*	
HDL (mg/dl):	43.62 ±3.74	50.00±4.42	58.74±4.26	<.001*
p-value$_1$		<.001*	<.001*	
p-value$_2$			<.001*	
Fasting blood glucose	109.47±3.59	105.55± 13.84	105.84± 10.39	.061
HbA1 c:	5.72±0.35	4.05±0.58	4.69±0.73	<.001*
p-value$_1$		<.001*	<.001*	
p-value$_2$			<.001*	

SD: Standard deviation.
*p-value significant if <.05.
p-value$_1$: Comparison with Hypertension with Hyperuricemia group.
p-value$_2$: Comparison with Hypertension without Hyperuricemia group.

The table shows the relation between LV Tei index and diastolic function, categorized as normal diastolic function, grade I diastolic dysfunction, and grade II diastolic dysfunction. The results indicate that there is a significant difference in LV Tei index values among the three groups (p<.001). The median LV Tei index value increases from 0.38 in normal diastolic function group to 0.49 in grade II diastolic dysfunction group.

Table (3): Echocardiographic data of the three groups.

Variables	Groups			*p*-value
	Hypertension with Hyperuricemia (n=125)	Hypertension without Hyperuricemia (n=125)	Control (n=50)	
IVS thickness diastole (mm): *p*-value [1] *p*-value [2]	13.62±1.09	12.38±0.09 .019*	9.45±0.27 <.001 * .001 *	<.001 *
LV end diastolic diameter (mm)	50.22±2.66	47.78±1.99	48.84±3.59	.064
LV Posterior wall thickness diastole(mm): *p*-value [1] *p*-value [2]	10.96±0.35	10.78±0.35 .473	8.46±0.42 <.001 * <.001 *	.003 *
Left atrium internal diameter (mm): *p*-value [1] *p*-value [2]	43.62±2.68	40.62±2.18 .001 *	35.64±3.59 .009* .002*	<.001 *
Ejection fraction (%)	68.08±3.50	68.30±3.30	69.96±2.98	.096
Peak E velocity (cm/s): *p*-value [1] *p*-value [2]	66.85±5.07	71.23±1.98 <.001 *	75.77±3.84 <.001 * <.001 *	<.001 *
Peak A velocity (cm/s)	61.98 ±3.50	61.20±3.30	58.76±2.98	.094
E/A ratio: *p*-value [1] *p*-value [2]	1.09±0.37	1.24±0.35 .003*	1.32±0.28 .001 * .001 *	.001 *
Peak of TR Velocity (m/s): *p*-value [1] *p*-value [2]	2.88±0.64	2.58±0.54 .001 *	2.18±0.58 .001 * .001 *	.001 *
LA Volume index (ml/m ^{2}): *p*-value [1] *p*-value [2]	29.41±3.25	24.67±2.77 <.001 *	19.18±6.89 <.001 * <.001 *	<.001 *
E'(cm/s): *p*-value [1] *p*-value [2]	8.57±0.93	9.37±1.49 <.001 *	10.38±0.85 <.001 * <.001 *	<.001 *
E/E': *p*-value [1] *p*-value [2]	8.45±0.94	8.27±1.45 .002*	8.18±0.85 .001 * <.001 *	.001 *
S`(cm/s): *p*-value [1] *p*-value [2]	8.86±0.94	9.63±1.49 <.001 *	11.05±1.20 <.001 * <.001 *	<.001 *
IVRT (ms): *p*-value [1] *p*-value [2]	94.03±8.42	77.15±8.57 <.001 *	55.48±4.64 <.001 * <.001 *	<.001 *
Ejection time (ms)	279.96± 18.49	281.14± 18.54	282.08±21.46	.153

Ivs thickness: Interventircular septum.
LV end diastole: Left ventricle.
Peak E: Early diastolic mitral inflow velocity.
Peak A: Late diastolic mitral inflow velocity.
Peak TR: tricuspid regurge.
E' : Early diastolic myocardial peak velocity lateral mitral annulas.
s' : Peak systolic myocardial velocity at lateral annulus.
IVRT: Tissue doppler derived isovolumetric relaxation time

SD: Standard deviation.
*p-value significant if <.05.
p-value [1]: Comparison with Hypertension with Hyperuricemia group.
p-value [2]: Comparison with Hypertension without Hyperuricemia group.

Table (4): Correlation of serum uric acid with echo parameters.

Variables	Uric acid (mg/dl)
IVS thickness diastole (mm):	
r-value	.917
p-value	<.001 *
LV end diastolic diameter (mm):	
r-value	.532
p-value	<.001 *
LV Posterior wall thickness diastole (mm):	
r-value	.508
p-value	<.001 *
Left atrium internal diameter (mm):	
r-value	.720
p-value	<.001 *
Ejection fraction (%):	
r-value	–.169
p-value	.003*
Peak E velocity (cm/s):	
r-value	.424
p-value	<.001 *
Peak A velocity (cm/s):	
r-value	.031
p-value	.594
E/A:	
r-value	.089
p-value	.123
Peak of TR Velocity m/s:	
r-value	.348
p-value	<.001 *
LA Volume index (ml/m ^{2}):	
r-value	.626
p-value	<.001 *
E' (cm/s):	
r-value	–.318
p-value	.001 *
E/E":	
r-value	.455
p-value	<.001 *
S`(cm/s):	
r-value	–.457
p-value	.009*
IVRT/ms:	
r-value	.695
p-value	<.001 *
Ejection time (ms):	
r-value	–.086
p-value	.139
Tie index:	
r-value	.840
p-value	<.001 *
LVmass index (gm/m ^{2}):	
r-value	.652
p-value	<.001 *

Table (5): LV Tei index relation with diastolic function.

Variables	Groups			*p*-value
	Normal diastolic function	Grade I diastolic dysfunction	Grade II diastolic dysfunction	
LV Tei index Median (IQR)	0.38 (0.38-0.39)	0.43 (0.43-0.44)	0.49 (0.49-0.51)	<0.001 *

*p-value significant if <.05.

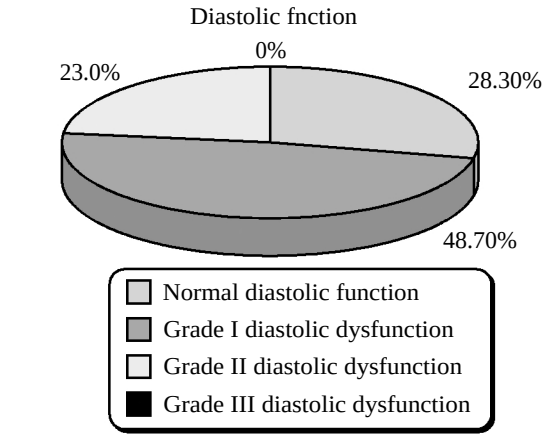

Fig. (1): Diastolic function distribution of the total study participants.

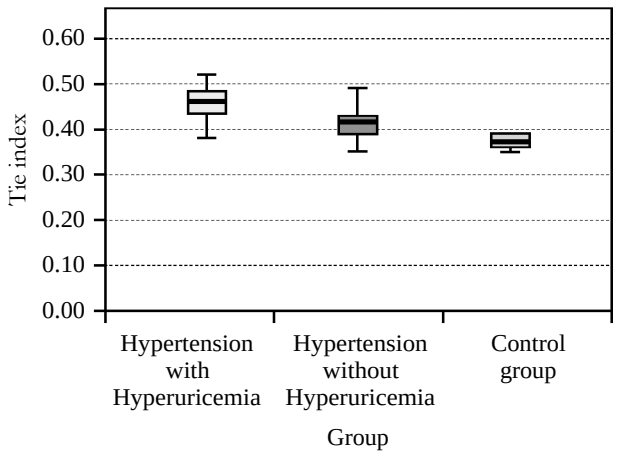

Fig. (2): Tie index of the three groups.

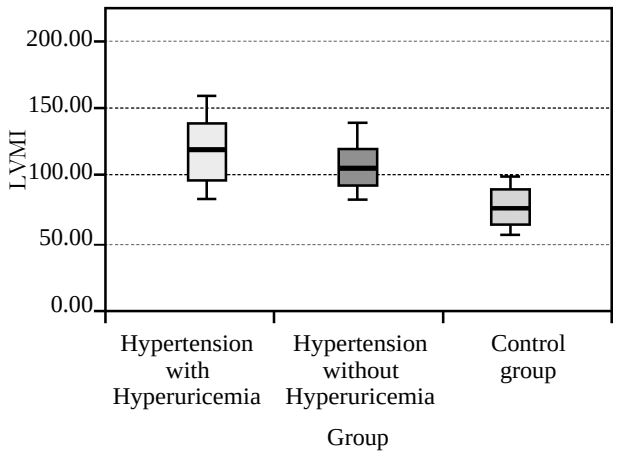

Fig. (2): LVMI of the three groups.

Discussion

In recent years, SUA has become an important parameter to consider when assessing cardiovascular risk. Indeed, sUA is associated with insulin resistance, hypertension, diabetes mellitus, dyslipidemia, obesity, metabolic syndrome, renal dysfunction, and hypothyroidism, which may explain enhanced cardiovascular (CV) risk among hyperuricemic individual [13].

Regarding the demographic data and medical history there was no significant difference in age among the three groups. However, there was a significant difference in gender distribution, with a higher percentage of males in the group with hypertension without hyperuricemia (52.0%) compared to the group with hypertension and hyperuricemia (36.8%) and the control group (44.0%).

Our results were supported by study of Adewuya et al., [14], as they reported that a cross-sectional study was performed on 271 (178 females, 93 males) patients with systemic hypertension. Two hundred and seventy-one healthy age and sex matched non-hypertensive persons obliged as controls. The mean age of the cases was 60.8 ± 12.3 years. There were 92 males (33.9%) and 179 females (66.1%). The controls were of similar sociodemographic characteristics. There was no statistically significant difference in the mean ages of the cases and controls (p=.811).

In the study in our hands, the Uric acid levels were significantly higher in the Hypertension with Hyperuricemia group (7.65 ±0.61mg/dl) compared to the Hypertension without Hyperuricemia (4.49 ±0.53mg/dl) and Control (4.16 ±0.52mg/dl) groups (p<.001 for both). The Serum creatinine levels were not significantly different between the three groups (p=.136). The Fasting Blood Glucose levels were also not significantly different between the three groups (p=.06).

Our results were supported by study of Adewuya et al., [14], as they reported that the mean SUA was significantly higher in the cases (371 ± 125 μmol/L) than in the controls (269± 101.4 μmol/L; p<.001). The Triglyceride and LDL levels were significantly higher in cases than control.

This association between elevated SUA and hypertensives was similar to the findings by Poudel et al., [15] who carried out a cross sectional study of hypertensive patients in Nepal and found that elevated SUA was more common in hypertensive patients when compared to the normotensive patients (28.8% versus 13.7% respectively; p<.001).

However, in the study of Tavil et al., [16], lipid parameters, plasma creatinine and blood urea levels were not different between the groups.

In a clinical study, an increase in uric acid levels >5.5mg/dL was found in 90% of hypertensive adolescent subjects, while uric acid levels were significantly lower in controls [17]. Furthermore, reduction of uric acid normalized blood pressure in 66% of hyperuricemic adolescents with hypertension as compared to 3% in the control individuals [18].

The present study showed that the IVS thickness diastole was significantly higher in the hypertension with hyperuricemia group compared to the hypertension without hyperuricemia and control groups (p<.001 and p=.019, respectively). Similarly, the LV posterior wall thickness diastole was significantly higher in the hypertension with hyperuricemia group compared to the hypertension without hyperuricemia and control groups (p<.001 and p=.473, respectively).

There was a significant difference in the tie index among the three groups (Hypertension with Hyperuricemia, Hypertension without Hyperuricemia, and Control). The median tie index was highest in the Hypertension with Hyperuricemia group (0.46±0.0) followed by the Hypertension without Hyperuricemia group (0.42 ±0.04) and was lowest in the control group (0.37±0.06).

Our results were supported by study of Adewuya et al., [14], as they reported that left ventricular mass index (LVMI) was found to be significantly higher in the hypertensive cases than the controls, and in the hypertensive hyperuricemic cases than the nonhyperuricemic cases respectively (p=<.001). LVMI (β=.382; p=<.001).

Furthermore, Fang et al., [19], stated that four age- and sex-matched groups were studied: I: healthy controls, HT - HU- (n = 40); II: HT - HU + (n = 40); III: HT+ HU- (n = 40); IV: HT+ HU + (n = 44). Despite LV diameters, LV volumes and EF were similar among groups (all p>.05).

Our results showed that regarding correlation between serum uric acid and various echocardiographic parameters. The results indicate that there is a statistically significant strong positive correlation between uric acid levels and IVS thickness diastole (r-value=0.917), LV end diastolic diameter (r-value=0.532), LV posterior wall thickness diastole (r-value=0.508), left atrium internal diameter (r-value=0.720), peak E velocity (r-value=0.424), peak TR velocity (r-value=0.348), LA volume

index (*r*-value=0.626), Tie index (*r*-value=0.840), LV mass index (*r*-value=0.652), E/E" (*r*-value= 0.455), and IVRT (*r*-value=0.695). These correlations are all statistically significant (*p*-value <.001).

On the other hand, the LV Tei index has a weak negative correlation with ejection fraction (*r*=–0.187, *p*=.001), E' (*r*=–0.180, *p*=.002) and S' (*r*=–0.158, *p*=.006). There was no significant correlation between LV Tei index and peak A velocity (*r*=0.073, *p*=.207), E/A ratio (*r*=0.033, *p*=.563), and ejection time (*r*=–0.048, *p*=.412).

The median LV Tei index value increases from 0.38 in normal diastolic function group to 0.49 in grade II diastolic dysfunction group.

Our results were supported by study of Adewuya et al., **[14]**, as they reported that there is a positive linear association between SUA ranges and LVMI (*r*=0.221, *p*=<.001). Adjusting for effects of multiple confounders like age, BMI, gender, exercise, lipids, diabetes, duration of hypertension and the class of BP made the correlation stronger (*r*=0.334, *p*=.001) meaning other factors apart from SUA affects the development of LVH and its different geometric patterns.

On the other hand, Campo et al. **[20]** found that hyperuricemia was not an independent marker of LVH and Tsioufis et al. **[21]** assessing the relationship between SUA and markers of target organ damage (TOD) such as LVH and microalbuminuria in 842 nondiabetic hypertensive patients reported that increased SUA levels were associated with microalbuminuria but not with LVH.

Furthermore, Tavil et al., **[16]**, revealed that correlations between the SUA and LV function parameters are shown. After correction for age and BMI, SUA was significantly correlated with IVRT, E/A ratio, Em/Am ratio, deceleration time.

Moreover, Visco et al., **[22]**, stated that SUA correlated positively with LVMi, as well as body size, metabolism, and kidney function. In a multivariate analysis, SUA confirmed the independent association with LVMi. Also, levels of SUA >5.6mg/dl are associated with larger cardiac size.

Conclusion:

This study reveals that hyperuricemia is widespread in our study population with systemic hypertension and both are positively correlated. Hyperuricemia was associated with LVH. Thus, the study recommends a repetitive evaluation of serum UA in all hypertensive patients. As it suggests that hyperuricemia is an early marker of increased left ventricular mass that can be used to identify a hypertensive population with cardiac TOD.

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تأثير فرط حمض بوليك الدم على وظيفة البطين الأيسر لدى مرضى ارتفاع ضغط الدم بمستشفيات جامعة عين شمس

فى هذه الدراسة المقطعية المستعرضة، تم فحص ٣٠٠ شخص وتم تقسيمهم إلى ٣ مجموعات كالتالى :

مجموعة أ وعددهم ١٢٥ وهم المرضى الذين يعانون من ارتفاع ضغط الدم المصابين بفرط حمض بوليك الدم، ومجموعة ب وعددهم ١٢٥ وهم المرضى الذين يعانون من ارتفاع ضغط الدم بدون فرط حمض بوليك الدم، ومجموعة ج وعددهم ٥٠ مشاركاً صحياً لا يعانى من أى شئ. وقد تم جمع الحالات من عيادات مستشفيات جامعة عين شمس ومعامل الموجات فوق الصوتية على القلب. وتم أخذ الموافقة من جميع المشاركين قبل الدخول فى الدراسة بعد شرح الغرض من الدراسة واجراءاتها . وخضع المشاركون فى الدراسة لمعايير الاشتمال والاستبعاد التالية:

أ- معايير الاشتمال بالدراسة:

- مرضى ضغط الدم المرتفع يعتمد التشخيص على تكرار قياس الضغط الانقباضى بحيث تساوى أو تزيد قيمة عن ١٤٠ مليمتر زئبقى و /أو ضغط الدم الانبساطى بحيث تساوى أو تزيد قيمة عن ٩٠ مليمتر زئبقى أو تاريخ علاجى بأدوية علاج ضغط الدم المرتفع.
- كفاءة عضلة القلب للبطين الأيسر أكثر من ٥٥٪.

ب- معايير الاستبعاد بالدراسة:

تم استثناء الحالات التى تعانى من : تاريخ مرض السكرى، قصور الشرايين التاجية، فشل عضلة القلب الاحتقانى، أمراض صمامات القلب المتقدمة، داء الأنسداد الرئوى المزمن ، بدانة ، العمر أكثر من ٦٠، تضخم البطين الأيسر الناتج عن أى سبب مرض الصمام، تضخم عضلة القلب، وأمراض القلب الخلقية. أو يتناول المرضى أى أدوية لأى من هذه الحالات. علاوة على ذلك، سيتم استبعاد الأشخاص الذين يتناولون أدوية لخفض حمض البوليك.

طرق الفحص :قد تم أخذ تاريخ مرضى مفصل من كل الحالات، مع إجراء الفحص بالموجات فوق الصوتية على القلب، والتى تشمل ثنائى الأبعاد وتدفق الألوان والدوبلر الطيفى بالإضافة إلى تصوير دوبلر الأنسجة. وقد تم أخذ الأبعاد المتعلقة بنهاية الانبساط ونهاية الانقباض للبطين الأيسر، سمك الحاجز ما بين البطينين والجدار الخلفى للبطين الايسر المتعلقة بنهاية الانبساط، كفاءة عضلة القلب، أبعاد الشريان الأورطى الصاعد، قطر الأذين الأيسر، كسر طرد البطين الأيسر ومؤشر كتلة البطين الأيسر ومؤشر حجم الأذين الايسر ومؤشر اداء عضلة القلب.

الاستنتاج :فى هذه الدراسة، خلصنا إلى أن هناك ارتباطاً وثيقاً بين ارتفاع نسبة حمض البوليك فى الدم وارتفاع ضغط الدم الشريانى، وأن له آثراً سلبياً على وظيفة عضلة القلب الانبساطية، والذى تم الكشف عنه بحساب مؤشر اداء عضلة القلب وذلك باستخدام الموجات فوق الصوتية على القلب فى مرضى ارتفاع ضغط الدم المرتفع.