Kardiologia Polska 2016; 74, 11: 1339–1345; DOI: 10.5603/KP.a2016.0061 ISSN 0022–9032

ARTYKUŁ ORYGINALNY / *ORIGINAL ARTICLE*

Assessment of myocardial performance index in hypertensive patients with or without hyperuricaemia

Cengiz Basar[1], Feyzullah Besli[2], Hakan Ozhan[3], Yasin Turker[3], Osman Kayapinar[3], Mesut Kecebas[4]

[1]Department of Cardiology, Duzce Ataturk State Hospital, Duzce, Turkey
[2]Department of Cardiology, Harran University School of Medicine Hospital, Sanliurfa, Turkey
[3]Department of Cardiology, Duzce University School of Medicine Hospital, Duzce, Turkey
[4]Department of Cardiology, Mudanya Ataturk State Hospital, Bursa, Turkey

Abstract

Background: Myocardial performance index (MPI) is impaired in patients with hypertension. Uric acid is biologically active and can stimulate oxidative stress, endothelial dysfunction, inflammation, and vasoconstriction. Hyperuricaemia may provide a negative contribution to impaired MPI in hypertension.

Aim: The study was designed to assess the MPI in hypertensive patients with or without hyperuricaemia.

Methods: A total of 96 consecutive hypertensive patients were divided into two groups according to levels of serum uric acid (SUA); 49 normouricaemic patients (defined as SUA < 7.0 mg/dL in men and < 6.0 mg/dL in women) and 47 hyperuricaemic patients. SUA levels and other biochemistry parameters were determined by a standard analytical technique. All patients were evaluated by two-dimensional and Doppler echocardiography.

Results: The two groups were similar according to age, body mass index, and smoking status. Mean MPI value (0.498 ± 0.06 vs. 0.410 ± 0.05, $p < 0.001$) was significantly higher in the hyperuricaemic group than the normouricaemic individuals and positively correlated with the mean value of SUA levels ($r = 0.51$, $p < 0.001$).

Conclusions: Our study demonstrated that high SUA levels were significantly associated with impaired MPI in hypertensive patients. SUA may suggest a valuable laboratory finding in assessing the risk of developing subclinical impaired left ventricular global function.

Key words: hypertension, serum uric acid, myocardial performance index

Kardiol Pol 2016; 74, 11: 1339–1345

INTRODUCTION

Hyperuricaemia is common in subjects with cardiovascular disease but is not generally considered a true risk factor [1]. Epidemiological studies have found that uric acid can independently predict the development of hypertension, stroke, and heart failure [1, 2]. Serum uric acid (SUA) levels may induce left ventricular (LV) hypertrophy in hypertensive patients [3].

The myocardial performance index (MPI) has been described by Tei et al. [4]. The MPI is a powerful index that provides the evaluation of systolic and diastolic functions at the same time [4, 5].

Left ventricular systolic and diastolic functions are impaired in patients with hypertension [6]. MPI also increases in hypertensive patients without LV hypertrophy [7]. The impact of SUA on MPI in hypertensive patients has not previously

Address for correspondence:
Dr Feyzullah Besli, Department of Cardiology, Harran University School of Medicine Hospital, Sanliurfa, Turkey, e-mail: feyzullahbesli@gmail.com
Received: 27.11.2015 **Accepted:** 31.03.2016 **Available as AoP:** 05.05.2016

been investigated. We aimed to investigate the relationship between the SUA levels and MPI in hypertension.

METHODS

A total of 96 consecutive hypertensive patients who were admitted to the cardiology department between March 2014 and June 2014 were included. Hypertension was defined as an adult systolic blood pressure (SBP) of 140 mm Hg or greater, or a diastolic blood pressure (DBP) of 90 mm Hg or greater, or taking antihypertensive medication. Two patients had newly diagnosed hypertension while all other patients had been taking regular hypertensive medication for at least one year. Patients were divided into two groups according to levels of SUA; 49 normouricaemic and 47 hyperuricaemic patients. Hyperuricaemia was defined as SUA level ≥ 7 mg/dL (in men) or ≥ 6.0 mg/dL (in women). None of the patients had any history of hyperlipidaemia, diabetes mellitus, cardiovascular disease, valvular heart disease, hyperthyroidism, chronic obstructive pulmonary disease, obesity, or stroke and none were taking any medications for any of these clinical conditions. Moreover, subjects who were taking uric acid-lowering agents were excluded. The body mass index (BMI) was defined as the body mass in kilograms divided by the square of the body height in metres (BMI = weight/height^2). An adult who had a BMI of 30 or higher was considered as obese. Following 12 h of fasting, venous blood samples of the patients were collected for SUA and other routine biochemical parameters. Biochemistry parameters were determined by a standard analytical technique. SUA concentrations were measured using the uricase enzymatic colorimetric test method (COBAS Integra 400 plus analyser). Informed consent was obtained from each subject for participation in this study. The local ethics committee approved the study.

Echocardiographic examination

Two trained cardiologists performed the echocardiography and recorded images for each patient using the Vivid 7 model of the echocardiography device (GE Vingmed Ultrasound, Horten, Norway). From the standard transthoracic windows, LV end-systolic diameter, LV end-diastolic diameter, and LV ejection fraction (LVEF) were measured. LV tissue Doppler imaging was performed from the apical four-chamber view using a frame rate of greater than 80/s. The Doppler sample volume was placed at the tips of the mitral leaflets to get the LV inflow waveforms from the apical four-chamber view. All sample volumes were positioned with ultrasonic beam alignment to flow. Transmitral E-wave velocity (E) and A-wave velocity were obtained from the recorded data and were averaged to generate the mean value. Tissue Doppler imaging was obtained with the sample volume placed at the medial and lateral corner of the mitral annulus from the apical four-chamber view. On the tissue Doppler images, mitral annular isovolumetric relaxation time (IVRT), isovolumetric contraction time (IVCT), ejection time (ET), early diastolic

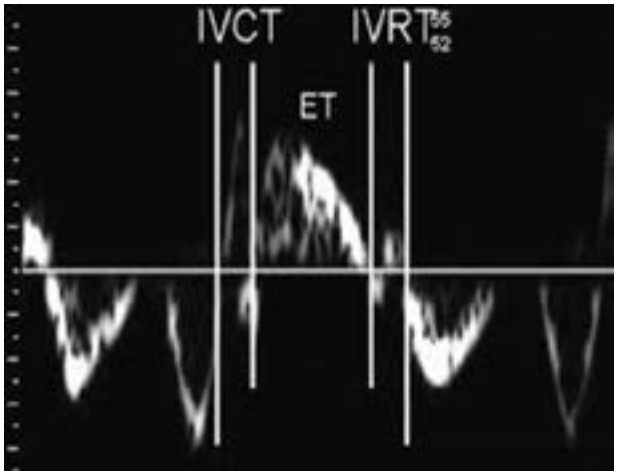

Figure 1. Measurement of the myocardial performance index (MPI). Time intervals of the MPI derived with pulsed wave tissue Doppler echocardiography. MPI was calculated as the sum of isovolumetric contraction time (IVCT) and isovolumetric relaxation time (IVRT) divided by ejection time (ET)

mitral annular velocity (Em), late diastolic mitral annular velocity (Am), and peak systolic mitral annular velocity (S) were measured from the same cardiac cycles, and the data were averaged to give the mean value. All measurements were obtained by two cardiologists in all patients who were blinded to the clinical data.

MPI measurement

Isovolumetric contraction time, defined as the interval measured from the end of the late diastolic mitral annular velocity pattern to the onset of the systolic mitral annular velocity pattern; ET, defined as the interval measured from the onset to the end of the systolic mitral annular velocity pattern; and IVRT, defined as the interval measured from the end of the systolic mitral annular velocity pattern to the onset of the diastolic mitral annular velocity pattern on the same cardiac cycle MPI was defined as the ratio of IVCT + IVRT to ET (Fig. 1).

Reproducibility

The interobserver and intraobserver variability were done for two cardiologists who analysed as offline previously the recorded images. Fifteen patients were randomly selected to evaluate the interobserver variability of the MPI measurements by two independent observers. To determine the intraobserver variability, the same measurements were repeated two weeks apart. Mean percentage error was calculated as the absolute difference divided by the average of the two observations.

Statistical analysis

The statistical package for social sciences software (SPSS ver. 12.0; SPSS Inc., Chicago, Illinois, USA) was used for comparisons of demographic and clinical variables. Variables were analysed for the presence of normal distribution

Table 1. Comparison of clinical and laboratory findings

	Hyperuricaemic group (n = 47)	Normouricaemic group (n = 49)	P
Male	30 (6%)	25 (51%)	0.182
Age [years]	56.4 ± 11.5	54.8 ± 9.9	0.422
Office systolic BP [mm Hg]	155.0 ± 15.0	150.0 ± 10.0	0.464
Office diastolic BP [mm Hg]	85 ± 5.0	80 ± 5.0	0.445
Cigarette smoking [%]	17	15	0.405
Body mass index [kg/m^2]	27.2 ± 3.5	26.6 ± 3.2	0.566
Duration of hypertension	6.1 ± 2.7	5.8 ± 2.0	0.553
Drugs:			
ACEI/ARB	24 (51%)	22 (44%)	0.413
CCB	18 (38%)	17 (34%)	0.385
Beta-blocker	12 (25%)	11 (22%)	0.392
Diuretics	20 (42%)	19 (38%)	0.356
Others	9 (19%)	7 (14%)	0.291
Glucose [mg/dL]	100.3 ± 13	97.6 ± 13.9	0.192
White blood cell [10^3/μL]	5.8 ± 1.9	6.2 ± 1.2	0.513
Haemoglobin [g/dL]	1 2.4 ± 1.5	12.1 ± 1.2	0.642
High-density lipoprotein [mg/dL]	31.5 ± 16.2	38.7 ± 10.9	0.042
Triglyceride [mg/dL]	167.7 ± 61	152.2 ± 93.3	0.046
Creatinine [mg/dL]	0.96 ± 0.19	0.99 ± 0.4	0.715
Uric acid [mg/dL]	6.8 ± 1.3	5.1 ± 1.2	**0.012**

ACEI/ARB — angiotensin converting enzyme inhibitors/angiotensin receptor blockers; BP — blood pressure; CCB — calcium channel blockers

using the Kolmogorov-Smirnov test. All values are given as mean ± standard deviation. Unpaired Student's t-test was used for group comparisons. Categorical data were compared using the χ^2 test. The Pearson correlation was used to evaluate the association between MPI and SUA. Statistical significance was defined as $p < 0.05$.

RESULTS

Forty-seven patients (30 men and 17 women with a mean age of 56.4 ± 11.5) were admitted with a diagnosis of high SUA levels. The rest of the patients (25 men and 24 women with a mean age of 54.8 ± 9.9) had normal SUA levels.

There were no significant differences in terms of age, BMI, smoking status, and duration of hypertension between the hyperuricaemic group and the normouricaemic group. In addition, the groups were similar with respect to serum levels of fasting glucose, haemoglobin, and creatinine and there were no significant differences among medical treatments. Compared to the normouricaemic group, the high-density lipoprotein-cholesterol (HDL-C) level was significantly lower (31.5 ± 16.2 vs. 38.7 ± 10.9, $p = 0.042$) and the triglyceride level was significantly higher (167.7 ± 61 vs. 152.2 ± 93.3, $p = 0.046$) in the hyperuricaemic group (Table 1).

Comparisons of echocardiographic variables are shown in Table 2. LVEF, atrial and ventricular end-diastolic diameters, interventricular septum, posterior wall thickness, E wave, A wave, S, Em, and Am were similar in both groups. In comparison with the normouricaemic group, E/Em and IVRT were significantly higher (9.5 ± 1.1 vs. 9.0 ± 1.2, $p = 0.020$ and 94.20 ± 11.9 vs. 84.34 ± 16.4, $p = 0.010$) and ET was significantly lower in the hyperuricaemic group (271.20 ± 22 vs. 289.55 ± 31.8, $p = 0.015$). The MPI value was significantly higher in the hyperuricaemic group (0.498 ± 0.06 vs. 0.410 ± 0.05, $p < 0.001$) (Table 2). There was also a significant positive correlation between MPI and SUA levels in all study subjects ($p < 0.001$, $r = 0.51$) (Fig. 2).

Reproducibility

The intraobserver and interobserver mean percentage errors for the MPI measurements in study patients were 3.9 ± 1.8% and 5.3 ± 2.9%, respectively.

DISCUSSION

Serum uric acid is the final product of purine metabolism in human beings. Despite the fact that SUA was first identified approximately two centuries ago, certain pathophysiological

Table 2. Comparison of left ventricular tissue Doppler imaging measurements and transthoracic echocardiographic measurements in two groups

	Hyperuricaemic group (n = 47)	Normouricaemic group (n = 49)	P
Left atrium [cm]	3.7 ± 0.42	3.6 ± 0.39	0.115
LV interventricular septum [cm]	1.15 ± 0.11	1.12 ± 0.10	0.204
LV posterior wall [cm]	0.92 ± 0.12	0.95 ± 0.10	0.668
LV end diastolic diameter [cm]	4.7 ± 0.5	4.6 ± 0.4	0.445
LV end systolic diameter [cm]	3.2 ± 0.6	3.3± 0.5	0.384
LV ejection fraction [%]	61.1 ± 5.3	60.6 ± 3.4	0.434
E [cm/s]	81,10 ± 9.3	77.8 ± 10	0.110
A [cm/s]	95.4 ± 7	93.9 ± 6.7	0.267
S [cm/s]	8.6 ± 0.4	8.7 ± 0.3	0.407
Em [cm/s]	8.4 ± 0.4	8.6 ± 0.4	0.054
Am [cm/s]	10.0 ± 1.5	10.2 ± 1.4	0.389
E/Em	9.5 ± 1.1	9.0 ± 1.2	**0.020**
Isovolumetric contraction time [ms]	60.28 ± 14.7	56.41 ± 14.3	0.156
Isovolumetric relaxation time [ms]	94.20 ± 11.9	84.34 ± 16.4	**0.010**
Ejection time [ms]	271.20 ± 22	289.55 ± 31.8	**0.015**
Myocardial performance index	0.498 ± 0.06	0.410 ± 0.05	**< 0.001**

A — transmitral diastolic A-wave velocity; Am — late diastolic mitral annular velocity; E — transmitral diastolic E-wave velocity; Em — early diastolic mitral annular velocity; LV — left ventricular

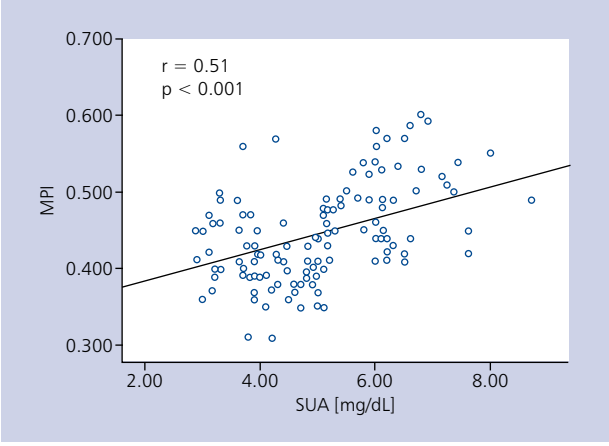

Figure 2. Correlation graph between myocardial performance index (MPI) and serum uric acid (SUA) levels

aspects of hyperuricaemia are still not clearly understood [8]. For years, hyperuricaemia has been thought to be the same as gout, but SUA has now been identified as a marker for a number of metabolic and haemodynamic abnormalities [8].

Serum uric acid is a powerful scavenger of free radicals and provides ~60% of free-radical scavenging capacity in plasma [9]. It is possible that this increase in circulating levels of SUA represents an adaptive response to protect against the detrimental effects of excessive free radicals and oxidative stress [9]. Previously studies demonstrated that circulating SUA was a major antioxidant and might help protect against free-radical oxidative damage [9, 10]. It is possible that an increase in SUA concentration is a protective mechanism to attenuate the adverse effects of an increase in oxidative stress [11]. Moreover, the antioxidant effect of SUA suggests that it might have therapeutic effects [10].

The positive association between SUA and cardiovascular diseases has been confirmed by numerous epidemiological studies [12, 13]. Elevated SUA levels have been linked to hypertension, hyperinsulinaemia, reduced physical activity, increased BMI, and increased alcohol consumption [14, 15]. Hyperuricaemia was associated with high triglyceride levels and low HDL-C levels [16]. Elevated SUA levels in the mentioned disease are thought to be associated with increased oxidative stress. Consistently, we also found higher triglyceride levels and lower HDL-C levels in in the hyperuricaemic group.

The myocardial performance index has been described by Tei et al. [4]. In adults, MPI values of the LV index < 0.40 and for the right ventricle < 0.30 are considered normal. Higher index values correspond to more pathological states with overall cardiac dysfunction. MPI has a high prognostic value in patients with dilated cardiomyopathy, amyloidosis, and coronary heart disease as well as in the general population [17]. MPI has previously been shown to be a sensitive indicator for

symptomatic heart failure in a cross-sectional study and MPI predicts future development of heart failure independently of other echocardiographic measurements [18].

The comorbidity of hypertension and hyperuricaemia is very common. In studies, hyperuricaemia was reported in 25–40% of untreated hypertensive subjects and in 75% of malignant hypertensive subjects [19, 20]. Hyperuricaemia is also associated with an increased risk for incident hypertension, independent of traditional hypertension risk factors [21].

Hyperuricaemia was associated with worse haemodynamic parameters in hypertension patients, such as increased right and left atrial pressure, and decreased cardiac index [22]. A cohort study in Japanese men showed an association between high uric acid levels and LV hypertrophy [23]. Hyperuricaemia was also shown to be a risk factor for incident heart failure [24]. Krishnan et al. [25] showed that increased SUA levels were associated with the abnormalities of myocardial systolic and diastolic function in echocardiography. In our study, IVRT was higher, while ET was lower in the hyperuricaemic group. As a result, we found that that the MPI value was significantly higher in the hyperuricaemic group than in the normouricaemic group in hypertension patients, and there was a positive correlation between the MPI and SUA level. The SUA level increases in response to oxidative stress in patients with hypertension, and the adverse effects of oxidative stress on cardiac function in hyperuricaemic group may be determined with MPI, an indicator of impaired LV function. Moreover, E/Em reflecting LV filling pressure was also higher in the hyperuricaemic group. These results may suggest high SUA caused impairment the systolic and diastolic functions resulting in increased LV filling as well as right and left atrial pressure, and decreased cardiac index as mentioned in previous studies [25].

Although the SUA has been well known for a long time, it still remains unknown whether the SUA is an independent risk factor, a mediator, or merely a marker of worse prognosis in hypertensive patients. The relationship between SUA levels and MPI in hypertension has not been investigated previously. High SUA level seem to be a relevant parameter of impaired LV global function because of its effect on MPI. Thus, SUA may suggest a valuable laboratory finding in assessing the risk of developing subclinical impaired LV global function. It may allow for early detection, treatment, and perhaps prevention of clinically impaired LV function.

Limitations of the study

This study has several limitations. Firstly, it was performed with the limited sample size and a cross–sectional design in a single centre. Secondly, although there was no difference in the frequency of antihypertensive classes between two groups, antihypertensive agents that have an influence on SUA levels and the duration of these antihypertensive agents and MPI were not evaluated. Both specific antihypertensive drugs and treatment duration may have influenced SUA levels and MPI. Presumably, newly diagnosed or untreated hypertensive patients can give more precise information to reveal the relation between MPI and SUA in hypertensive patients. Thirdly, dietary habits or alcohol intake of subjects, which may influence SUA levels, were not questioned. Fourthly, in echocardiographic evaluation, analyses of LV mass and relative wall thickness were not evaluated. They could provide additional information about the relationship between uric acid level and type of LV remodelling (concentric or eccentric hypertrophy). Another limitation of the study is an absence of follow-up in terms of clinical events.

CONCLUSIONS

In this study, we found that impaired MPI was associated with high SUA levels in hypertensive patients. Nevertheless, further studies are needed in order to define the role of SUA and MPI in clinical practice. Furthermore, it has to be determined if the pharmacological and/or non-pharmacological interventions reduce SUA and MPI.

Conflict of interest: *none declared*

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Cite this article as: Basar C, Besli F, Ozhan H et al. Assessment of myocardial performance index in hypertensive patients with or without hyperuricaemia. Kardiol Pol, 2016; 74: 1339–1345. doi: 10.5603/KP.a2016.0061.

W dniu 26 października 2016 roku nominację profesorską otrzymał:

Prof. dr hab. n. med. Wacław Kochman
(Gdański Uniwersytet Medyczny)

Panu Profesorowi
serdeczne gratulacje i okolicznościowe życzenia składają:
Redaktor Naczelny oraz Rada Redakcyjna i Naukowa „Kardiologii Polskiej"

Ocena wskaźnika wydolności mięśnia sercowego u chorych na nadciśnienie tętnicze z hiperurykemią i prawidłowym stężeniem kwasu moczowego

Cengiz Basar[1], Feyzullah Besli[2], Hakan Ozhan[3], Yasin Turker[3], Osman Kayapinar[3], Mesut Kecebas[4]

[1]Department of Cardiology, Duzce Ataturk State Hospital, Duzce, Turcja
[2]Department of Cardiology, Harran University School of Medicine Hospital,, Sanliurfa, Turcja
[3]Department of Cardiology, Duzce University School of Medicine Hospital, Duzce, Turcja
[4]Department of Cardiology, Mudanya Ataturk State Hospital, Bursa, Turcja

Streszczenie

Wstęp: U chorych na nadciśnienie tętnicze wartości wskaźnika wydolności mięśnia sercowego (MPI) są nieprawidłowe. Kwas moczowy jest biologicznie czynną substancją, która może powodować rozwój stresu oksydacyjnego, dysfunkcji śródbłonka, zapalenia i skurczu naczyń. Hiperurykemia może wywierać szkodliwy wpływ, przyczyniając się do pogorszenia MPI u chorych na nadciśnienie tętnicze.

Cel: Badanie zaprojektowano w celu oceny MPI u chorych na nadciśnienie tętnicze z hiperurykemią i prawidłowym stężeniem kwasu moczowego.

Metody: Kolejnych 96 chorych na nadciśnienie podzielono na dwie grupy w zależności od stężenia kwasu moczowego w surowicy (SUA): 49 osób z normourykemią (definiowaną jako SUA < 7,0 mg/dl u mężczyzn i SUA < 6,0 mg/dl u kobiet) oraz 47 pacjentów z hiperurykemią. Wartości SUA i inne parametry biochemiczne określono za pomocą standardowych metod analitycznych. U wszystkich chorych wykonano echokardiografię dwuwymiarową i doplerowską.

Wyniki: Grupy nie różniły się pod względem wieku chorych, wskaźnika masy ciała i palenia tytoniu. Średnie wartości MPI (0,498 ± 0,06 *vs*. 0,410 ± 0,05; p < 0,001) były istotnie wyższe w grupie pacjentów z hiperurykemią niż u osób z prawidłowym stężeniem kwasu moczowego i korelowały dodatnio ze średnimi wartościami SUA (r = 0,51; p < 0,001).

Wnioski: W badaniu wykazano, że wysokie wartości SUA były istotnie związane z gorszym MPI u chorych na nadciśnienie. SUA może być cennym parametrem laboratoryjnym w ocenie ryzyka rozwoju subklinicznych zaburzeń globalnej wydolności lewej komory.

Słowa kluczowe: nadciśnienie, stężenie kwasu moczowego w surowicy, wskaźnik wydolności mięśnia sercowego

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Adres do korespondencji:
Dr Feyzullah Besli, Department of Cardiology, Harran University School of Medicine Hospital, Sanliurfa, Turkey, e-mail: feyzullahbesli@gmail.com
Praca wpłynęła: 27.11.2015 r. **Zaakceptowana do druku:** 31.03.2016 r. **Data publikacji AoP:** 05.05.2016 r.