

### RELATIONSHIP BETWEEN MASKED ARTERIAL HYPERTENSION AND ERECTILE DYSFUNCTION

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#### ABSTRACT

##### Background

Erectile dysfunction (ED) has a marked negative effect on quality of life. The association between sustained hypertension (HT) and ED has been clearly shown. However, there is no study evaluating masked HT and ED. We aimed to assess the prevalence of masked HT and the related factors in patients with ED.

##### Methods

A total of 64 consecutive males with ED (mean age:  $50.4 \pm 9.8$  years) were enrolled in the study. The Sexual Health Inventory for Men (SHIM) questionnaire was used to evaluate the erectile status of the patients. Office and 24-h ambulatory blood pressure (BP) of all patients were measured.

##### Results

We detected masked HT in 24 of 64 patients with ED (37.5%). The SHIM score was slightly lower in masked HT group compared to true normotensives, but the difference was not statistically significant ( $10.8 \pm 5.2$  vs.  $11.4 \pm 4.6$ ;  $p=0.65$ ). There was no significant correlation between all-day systolic and diastolic BP with SHIM scores ( $R=0.076$ ,  $p=0.55$ ;  $R=0.079$ ;  $p=0.53$ ). When the patients with masked

HT were classified according to the nocturnal BP reduction, the SHIM scores of patients with the nondipping pattern were lower than the dippers ( $9.8 \pm 5.3$  vs.  $12.0 \pm 5.1$ ;  $p=0.001$ ).

### Conclusions

The prevalence of masked HT is high in patients with ED. Patients with masked HT and nondipping nocturnal BP pattern have more profound ED. The coexistence of masked HT and ED is thought to be a marker of increased cardiovascular risk.

**Key Words:** *arterial hypertension; circadian blood pressure; dipping; erectile dysfunction; masked hypertension*

## INTRODUCTION

Erectile dysfunction (ED) is an important disorder in men that affects sexual life and the total quality of life. ED is defined as the loss of the ability to provide and/or maintain the erectile status.<sup>1</sup> ED is a multifactorial disease in which the vascular, psychogenic, and neurohormonal factors play a role in its pathophysiology.<sup>2</sup> There are various reports with respect to the prevalence of the ED due to the differences in study populations. It has been reported that the ED prevalence in the general population varies between 20% and 50%.<sup>1,3</sup> The prevalence of ED was shown to increase with age.<sup>4</sup> The close relationship between ED and some diseases, such as coronary artery disease, hypertension (HT), diabetes mellitus, dyslipidemia, and metabolic syndrome, was demonstrated in many studies.<sup>5,6</sup> This relationship was based on increased oxidative stress, impaired endothelial function, and decreased nitric oxide (NO) release.<sup>7,8</sup>

Even though the office blood pressure (BP) values were normal or high normal (<140/90 mmHg), having the high ambulatory or home BP (daytime average  $\geq 135/85$  mmHg, 24 h average  $\geq 130/80$  mmHg, night-time average  $\geq 120/80$  mmHg) was defined as masked HT according to the European Society of Cardiology 2018 arterial hypertension guidelines.<sup>9</sup> The prevalence of the masked HT was indicated between 10% and 17% in the population based on the studies.<sup>9,10</sup> Smoking, alcohol use, anxiety, obesity, diabetes

mellitus, chronic renal failure, and family history of HT were associated with increased prevalence of masked HT.<sup>10</sup> It has been reported that the long-term risks of diabetes mellitus and sustained HT increased in patients with masked HT.<sup>10,11</sup> In the cross-sectional studies, it has been reported that the risk of target organ damage was higher in the masked HT patients when compared with true normotensive patients with.<sup>12</sup> According to a meta-analysis of prospective studies, the incidence of cardiovascular events in masked HT patients is twofold more compared with true normotensive patients, whereas it was similar compared to hypertensive patients.<sup>13</sup>

The ED prevalence in hypertensive patients was reported between 15% and 67% according to the study populations.<sup>14</sup> It was demonstrated that the ED prevalence was twofold more in men with systolic BP  $\geq 140$  mmHg compared to those with BP <140 mmHg.<sup>15</sup> In another study, the increment in pulse pressure, which was known as the arterial stiffness parameter, was shown to be associated with deterioration of erectile function and increase in ED prevalence.<sup>16</sup> However, there is no study regarding the relationship between masked HT and ED. In this study, we aimed to assess the prevalence of masked HT and the related factors in patients with ED.

## MATERIALS AND METHODS

In our study, we evaluated the findings of consecutive 185 male patients (age range: 30–70 years)

who were diagnosed with ED. A total of 64 patients who met the inclusion/exclusion criteria (mean age  $50.4 \pm 9.8$  years) were enrolled in the study. Written informed consent forms were obtained from all the patients. The study was approved by the local ethics committee (Medical Park Hospital Institutional Review Board [IRB], IRB number: 2019/13). All procedures followed were in accordance with the ethical standards of the IRB on human experimentation and with the Declaration of Helsinki.

The inclusion criteria were as follows: 30–70-year-old male patients who were diagnosed with ED in the urology clinic. The exclusion criteria were as follows: having signs of ischemia according to the electrocardiography or exercise treadmill test, a history of myocardial infarction, percutaneous coronary intervention, unstable angina pectoris, heart failure with reduced ejection fraction (EF) (EF < 40%), severe valvular diseases, diabetes mellitus, glomerular filtration rate (GFR) < 60 mL/min, liver dysfunction, gout, malignancy, dyslipidemia, any systemic disorders, alcohol and drug addiction, obstructive sleep apnea syndrome, any organic or vascular disorders that can lead to ED, and benign prostate hypertrophy. In addition, use of any medication affecting BP, drug use due to psychiatric disorders and undergoing urologic surgery were also among the exclusion criteria.

The demographic and clinical characteristics of all patients, such as age, height, weight, resting BP, and drug use, were recorded. A 24-h ambulatory BP monitoring was performed in all patients. The erectile status of the patients was evaluated by using the Sexual Health Inventory for Men (SHIM) questionnaire. This SHIM questionnaire is also known as the International Index of Erectile Function (IIEF)-5. The SHIM questionnaire contains five items and it is the short form of the IIEF questionnaire that contains 15 items. Each item is scored either from 0 to 5 or from 1 to 5 and yielding a global sexual function score between 1 and 25. SHIM score < 21 was defined as a probable ED.<sup>14</sup>

Blood analysis was performed upon at least 12-h starving period. Therefore, the fasting blood samples were taken from patients by ante-cubital vein. Laboratory results were obtained by using the Sysmex KX-21N autoanalyzer (Sysmex Corporation, Lincolnshire, IL, USA). When the patients first arrived, measurements with 2-min intervals were performed in the office from both left and right arms by using brachial artery after resting 10 min. The results were recorded. For this purpose, the mercury sphygmomanometer (ERKA D-83646 Bad Tolz, Kallmeyer Medizintechnik GmbH Co. KG, Germany) was used. The measurements were repeated in case we detected more than 5 mmHg difference between two measurements. The 24-h ambulatory BPs of all patients were recorded by using a noninvasive automatic device (Tracker NIBP2, Del Mar Reynolds Ltd, Hertford, England, UK), and the cuff of the device was placed in a less-used side. The participants were suggested that they should continue their normal daily activities and should not change their sleeping habits and move their arms during the measurement. BP measurements were planned as ones in every 30 min during the day and ones in every 60 min at nights. Measurements were performed at least 24 h. The average values were calculated as three periods. The first period was measured between the 1 am and 6 am during the night, the second period was measured between 9 am and 9 pm, and the third period was composed of all-day measurements. The patients with office BP < 140/90 mmHg and average BP  $\geq 135/85$  mmHg at 24-h ambulatory BP monitoring records were evaluated as the masked HT.<sup>9</sup> The day, night, and all-day systolic, diastolic, and mean BPs were evaluated. When there was more than 10% drop in BPs between day and night, it was defined as “dipper,” and when there was drop of 10% or less than 10% in BPs, it was defined as “nondipper.”<sup>17</sup>

## STATISTICAL ANALYSIS

All data were analyzed using SPSS software version 15.0 (SPSS, IBM, Chicago, IL, USA). The statistical analyses of the study were performed by the Kolmogorov–Smirnov test and the Mann–Whitney *U* test. Comparisons between the groups were made by chi-square, Student's *t*, and one-way Analysis of Variance (ANOVA) tests, where appropriate. Fischer's test was used in cases where chi-square test was not appropriate. Categorical data were studied by Pearson's correlation analysis, whereas discrete data were evaluated by Spearman's correlation analysis. The level of statistical significance was accepted as  $p < 0.05$  for all tests.

## RESULTS

Among 64 patients (mean age:  $50.4 \pm 9.8$  years), masked HT was detected in 24 patients (37.5%). ED patients were examined in two subgroups called as masked HT and true normotensives. SHIM score was slightly lower in masked HT group than in true normotensives; however, the difference was not statistically significant ( $10.8 \pm 5.2$  vs.  $11.4 \pm 4.6$ ;  $p=0.65$ ). Demographic characteristics and laboratory findings of patients with and without masked HT are shown in Table 1.

Although the office systolic and diastolic BPs were in the normal range in both groups,

**TABLE 1** Demographic Characteristics and Laboratory Findings of Patients with Normal BP or Masked HT

Parameter	Normotensives, (n=40)	Masked HT, (n=24)	p
Age, years	49±9	52±11	0.22
Body mass index (kg/m <sup>2</sup> )	25.4±2.2	24.7±3.4	0.32
Hip circumference, cm	106±10	106±12	0.97
Waist circumference, cm	102±13	99±14	0.45
Smoking, %	67	77	0.60
Glucose, mg/dL	118±48	106±33	0.80
Creatinine, mg/dL	0.81±0.93	0.92±0.12	0.07
Triglyceride, mg/dL	170±96	249±233	0.10
Total cholesterol, mg/dL	211±34	219±49	0.6
HDL cholesterol, mg/dL	44±10	41±8	0.95
LDL cholesterol, mg/dL	130±34	132±35	0.84
Uric acid, mg/dL	5.4±1.0	4.1±1.2	0.75
Hemoglobin (g/l)	15.5±1.9	15.3±1.2	0.66
White blood cell count, 10 <sup>3</sup> /mL	8.2±2.1	8.6±2.3	0.56
Serum C-reactive protein, mg/L	5.6±15	4.2±5	0.69
Mean platelet volume	9.1±1.9	8.9±1.6	0.68
Alanine aminotransferase, U/L	19±5.4	22±7.9	0.26
SHIM score	11.4±4.6	10.8±5.2	0.65

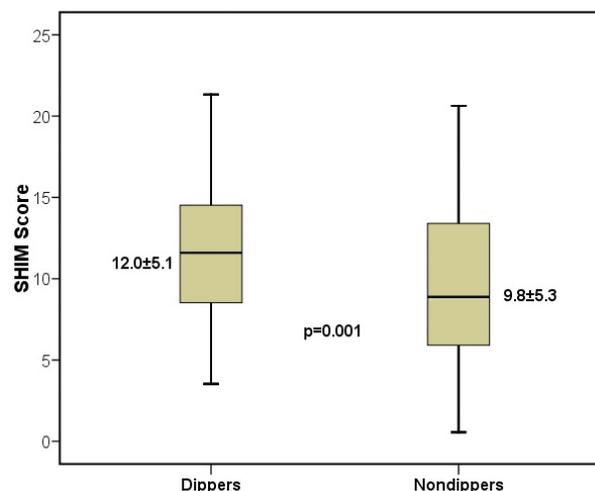
BP = blood pressure; HDL = high density lipoprotein; HT = hypertension; LDL = low density lipoprotein; SHIM = Sexual Health Inventory for Men questionnaire.

Numerical variables were presented as the mean±SD and categorical variables were presented as percentages. The *p*-values lower than 0.05 was considered significant and made boldface.

the office BPs of masked HT patients were significantly higher than that of the true normotensive group ( $47.2 \pm 12.2$  vs.  $41.1 \pm 7.3$ ;  $p=0.03$ ) (Table 2). All-day ambulatory systolic, diastolic, and pulse pressure measurements of masked HT group were higher as expected (Table 2). All patients were further classified as dippers and nondippers according to nocturnal BP patterns. There was no statistically significant difference between dippers and nondippers with ED with respect to their SHIM scores ( $11.1 \pm 4.6$  and  $11.3 \pm 5.2$ ;  $p=0.84$ ). SHIM scores of the masked HT with nondipping pattern were lower than that of the masked HT patients with dipping pattern ( $9.84 \pm 5.33$  vs.  $12.00 \pm 5.13$ ;  $p=0.001$ ) (Figure 1).

There was no statistically significant correlation between the SHIM scores and all-day ambulatory BP measurements in ED patients (24-h systolic BPs [ $R: 0.076$ ,  $p=0.55$ ], 24-h diastolic BPs [ $R: 0.079$ ,  $p=0.53$ ]). Patients were classified into

four groups (SHIM score <7, 7–11, 11–16, 17–21) according to the ED severity. Masked HT prevalence was evaluated in ED groups using the



**FIG. 1** The severity of erectile dysfunction in patients with masked HT with dipping and nondipping patterns.

**TABLE 2** Office and Ambulatory Blood Pressure Monitoring Findings of Patients with Normal BP or Masked HT

Parameter	Normotensives, (n=40)	Masked HT, (n=24)	p
Office BP, easurements (mm/Hg)			
First SBP	116.8±11.5	128.7±18.7	<b>0.002</b>
Second SBP	118.2±11.6	130.21±17.5	<b>0.002</b>
First DBP	75.7±8.5	81.5±9.5	<b>0.016</b>
Second DBP	77.8±8.2	79.6±7.9	0.38
PP (mmHg)	41.1±7.3	47.2±12.2	0.03
Ambulatory BP (mm/Hg)			
Daytime SBP	117.2±8.2	146.7±16.5	<b>0.001</b>
Daytime DBP	76.1±5.6	93.0±10	<b>0.001</b>
Night time SBP	105.8±19	137.8±17	<b>0.001</b>
Night time DBP	68.2±8.1	84.7±11.4	<b>0.001</b>
All day SBP	114.9±7.9	143.6±16.4	<b>0.001</b>
All day DBP	74.0±5.2	90.0±11	<b>0.001</b>
All day PP	40.8±5.6	53.3±10.2	<b>0.001</b>

BP = blood pressure; DBP = diastolic blood pressure; HT = hypertension; PP = pulse pressure; SBP: systolic blood pressure. Numerical variables were presented as the mean±SD and categorical variables were presented as percentages. The p-values lower than 0.05 was considered significant and made boldface.

Kruskal–Wallis test, but there was no statistically significant difference ( $p=0.57$ ).

### DISCUSSION

In our study, masked HT prevalence was 37% in ED patients. However, we could not find any association between ED severity and masked HT prevalence. Also, we did not detect a correlation between BP levels and SHIM scores in ED patients. However, SHIM scores of masked HT patients with nondipping pattern were statistically significant lower than that of the masked HT patients with dipping pattern.

Erection is a complex process including neuropsychosomatic triggering and vascular response.<sup>18</sup> Endothelial cells are the main source of NO.<sup>19</sup> The parasympathetic system is activated by sexual arousal consequently, due to the NO release cyclic guanosine monophosphate (cGMP) concentration is increased. The increment in the cGMP concentration leads to the erection that occurs due to the arterial smooth muscle relaxation in the penis.<sup>19</sup> Some factors can have some toxic effects on endothelial cells. These factors can lead to deterioration of the NO use and its functions resulting in leukocyte and platelet adhesion and aggregation with the release of vasoconstrictive substances.<sup>8</sup> It has been stated that the common pathophysiological mechanism of HT and ED was endothelial dysfunction.<sup>14</sup> There are two novel factors that could interfere with the endothelial function: apelin and relaxin. Apelin, which is an adipokine, plays a role in endothelium-dependent vasodilatation and indirectly decreases arterial HT. Relaxin, which is a protein hormone, promotes the NO and vascular endothelial growth factor production and antagonizes the effect of endothelin and angiotensin II (Ang II).<sup>20</sup> According to Papadopoulos et al.,<sup>20</sup> serum apelin and relaxin levels were found significantly lower in masked HT patients than in normotensive adults. Furthermore, it was demonstrated that apelin and its receptor expression

decreased in vasculogenic ED in mice which could be a pathophysiological explanation for the ED in masked HT.<sup>21</sup>

Moreover, vasoconstrictors, such as the Ang II, predominantly play a role in the arterial HT mechanism. Elevated levels of the Ang II increase vascular smooth muscle (VSM) contraction, so disrupts the erectile mechanism. Patients diagnosed with masked HT have not only the elevated levels of the AngII, but also the AT<sub>2</sub> receptor activity is augmented. In addition, it has been known that HT can increase penile vascular wall thickness with the collagen deposition and decrease lumen diameter. This might affect the penile blood flow and could result the ischemia of the VSM cells and Schwann cells.<sup>22</sup>

In some studies, “nondipping” pattern was clearly shown to have an association with the augmentation of the cardiovascular risks in patients with HT.<sup>23,24</sup> Similarly, it has been claimed that the “nondipping” pattern increased the risk of the damage in the target organ in normotensive individuals.<sup>25–27</sup> In experimental studies, the drop in the night BP was shown to be related to the autonomic nervous system activity.<sup>28</sup> In our study, there was a relationship between the nondipping pattern and ED severity in masked HT patients. It is possible to support the hypothesis that the increased cardiovascular risk, the target organ damage and the autonomic dysfunction in nondippers can also lead to ED.

Masked HT prevalence in ED patients was detected as 37% in our study and this was significantly higher than the masked HT prevalence of the general population (10–17%).<sup>9</sup> Similarly, even though the office BPs of ED and masked HT group patients were in the normal range, the office BPs of masked HT patients were higher than that of normotensive individuals. Many studies have shown the association between HT and ED was shown.<sup>14,29</sup> However, there is no paper about the relationships between masked HT and ED. It has been indicated that there was

an augmentation in the sustained HT risk in masked HT patients.<sup>11</sup> These findings can show us that there are similar pathophysiological mechanisms between ED and masked HT as it has already been indicated for ED and HT.

The risk of the target organ damage was shown to be increased in the patients with masked HT.<sup>30,31</sup> It has been reported that there was an increment in the left ventricular mass in masked HT.<sup>30</sup> In another interesting study, in masked HT patients, the increment in the carotid intima-media thickness was shown to be similar to the values of sustained HT.<sup>31</sup> On the contrary, the relationship between the traditional cardiovascular risk factors and masked HT was indicated in many studies.<sup>32</sup> This close association between atherosclerosis and masked HT can be the reason for the detection of the increment in masked HT prevalence in ED patients. Even though the design of our study is not suitable for the analysis of the cause and effect relationships, the findings let us think that the factors that lead to masked HT can also be the causes of ED.

There are many studies that indicate that the ambulatory BP and masked HT prevalence increase in individuals who work under stress.<sup>33,34</sup> When the close relationship of ED and psychogenic factors is considered, there can be another reason for the association between masked HT and ED, and this should be searched by further studies.<sup>35</sup>

### LIMITATIONS

There are some limitations of this study. Although the power of our analysis is statistically sufficient, we studied in a small population due to our strict exclusion criteria. In addition, this is an observational study and SHIM questionnaire used in the ED evaluation can lead to misunderstandings as it is a subjective test. These findings cannot be generalized to the entire community because there are other reasons for ED except vascular ones. In this study, whether masked HT

is an independent indicator of ED cannot be evaluated due to the lack of the healthy control group. Further studies and randomized controlled trials are needed to reveal the relationship between masked HT and ED.

### CONCLUSIONS

The prevalence of masked HT in ED patients could be increased. Furthermore, it has been shown that the nondipping pattern could be associated with ED severity in masked HT. The coexistence of masked HT and ED is thought to be a marker of increased cardiovascular risk.

### COMPLIANCE WITH ETHICAL STANDARDS

#### CONFLICT OF INTEREST

No potential conflict of interest was reported by the authors.

#### FUNDING

No funding was received for this work.

#### ETHICAL APPROVAL

The study protocol was approved by the local ethics committees.

#### INFORMED CONSENT

Informed consent was obtained from all individual participants included in the study.

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