



## Original Research

# Abnormal Blood Pressure Dipping Pattern in Women with Hypopituitarism Secondary to Sheehan Syndrome: A Case-Control Study

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### Abstract

**Objectives:** The aims of this study were to assess the 24-hour ambulatory BP levels and to determine the prevalence of abnormal circadian BP dipping patterns in women with hypopituitarism secondary to Sheehan syndrome.

**Methods:** This was a cross-sectional study including 35 women with complete anterior hypopituitarism secondary to Sheehan syndrome and 47 age- and body-mass index-matched control women. Subjects receiving treatment for hypertension were not included. All participants underwent clinical examination, laboratory tests, and BP measurement using ambulatory 24-hour monitoring.

**Results:** The mean age was 61.3±10.6 years in patients vs 60.5±8.5 years in controls (p=0.720). Compared to controls, women with Sheehan syndrome had a higher prevalence of dyslipidemia (p=0.032) and metabolic syndrome (p=0.028). The prevalence of hypertension was 68% in patients and 62% in controls (p=0.520). Altered day-night BP variation was more frequent in patients (85%) than in controls (54%) (p=0.004). Additionally, patients had a significantly higher prevalence of nocturnal hypertension (38% versus 3%; p=0.002). Sheehan syndrome was positively associated with a non-dipper and riser BP profile (Odds Ratio=4.7, 95% confidence interval: 1.54–14.33, p=0.004).

**Conclusion:** Women with hypopituitarism secondary to Sheehan syndrome had a higher disruption of the circadian BP rhythm than controls. Although the prevalence of newly diagnosed hypertension was comparable between patients and controls, women with Sheehan syndrome had a higher prevalence of nocturnal hypertension.

**Keywords:** Blood pressure dipping pattern, GH deficiency, hydrocortisone, hypopituitarism, nocturnal hypertension, Sheehan syndrome

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Sheehan syndrome is caused by ischemic necrosis of the anterior pituitary gland following delivery hemorrhage. It represents a rare cause of hypopituitarism. The interval between the hemorrhagic event and the diagnosis of Sheehan syndrome can vary from a few months to many years.

<sup>[1,2]</sup> The non-specific clinical presentations contribute to the underdiagnosis and underestimation of the true prevalence of this condition. Typically, women with Sheehan syndrome present with hypopituitarism affecting the lactotropin, corticotropin, thyrotropin, somatotropin, and gonadotropin axes

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to varying degrees. Complete anterior pituitary dysfunction has been reported in 55.3% to 84% of cases, and growth hormone (GH) deficiency was almost constant.<sup>[3,4]</sup>

Treatment of Sheehan syndrome relies on hormone replacement therapy. However, growth hormone (GH) deficiency is generally insufficiently or not at all substituted. In adults, GH and gonadotropin deficiencies are responsible for an increase in fat mass with a visceral fat deposit.<sup>[5]</sup> Furthermore, conventional treatment of corticotropin deficiency, including short-acting glucocorticoids, does not mimic the physiological circadian cortisol rhythm. This may lead to chronic overexposure to hydrocortisone. All these mechanisms justify the increased risk of metabolic and cardiovascular complications in patients with hypopituitarism.<sup>[5,6]</sup>

A non-dipping blood pressure (BP) profile is defined by a disorder of the circadian BP rhythm with an inadequate decrease in BP levels during the nocturnal period. It is associated with poorer renal and cardiovascular outcomes.<sup>[7]</sup> Reduced nocturnal BP decline was observed in patients with hypopituitarism and was considered a contributor to the increased cardiovascular risk in this population.<sup>[8]</sup>

The aims of the study were to evaluate the 24-hour ambulatory BP levels and to determine the prevalence of abnormal BP dipping patterns in women with hypopituitarism secondary to Sheehan syndrome.

## Methods

### Study Protocol

A cross-sectional case-control study was carried out in the Department of Endocrinology between March 2021 and December 2022. Thirty-five women with Sheehan syndrome and 47 age- and body mass index (BMI)-matched women without Sheehan syndrome were consecutively enrolled. The diagnosis of Sheehan syndrome was established in the presence of the following criteria:<sup>[9]</sup> history of delivery hemorrhage with severe hypotension or postpartum shock, failure of postpartum lactation, failure to resume menses after delivery, partial or complete anterior pituitary insufficiency, and empty sella turcica on pituitary imaging.

The non-inclusion criteria for patients were all other causes of anterior pituitary insufficiency (pituitary tumors, traumatic causes, autoimmune hypophysitis, etc.), other endocrinopathies (primary hypothyroidism, hypoparathyroidism, hyperparathyroidism, primary hyperaldosteronism, pheochromocytoma, etc.), type 1 diabetes, diabetic neuropathy, treated hypertension, coronary heart disease, heart failure, creatinine clearance  $<30$  ml/min/1.73 m<sup>2</sup>, liver failure, respiratory failure, neoplasia, chronic inflammatory and/or infectious diseases, and smoking. Patients

with non-adherence to hydrocortisone and levothyroxine replacement therapy were excluded.

Control women with no pituitary insufficiency were recruited from the hospital paramedical staff and relatives of patients. The same non-inclusion criteria were applied to the controls.

The study was conducted in accordance with the Declaration of Helsinki and was approved by the hospital ethics committee (CEBM.EPS.HR/24/2021 date: 14/04/2021/n: 24). Patients and controls were informed about the study's aims and were included after signing a consent form.

### Data Collection

Participants underwent a structured interview, a physical examination, 24-hour ambulatory BP monitoring (ABPM), and a biochemical analysis.

Demographic characteristics, Sheehan syndrome history, pituitary deficiencies, replacement therapy, and past medical history were determined. All participants underwent physical examination, including weight, height, and waist circumference (WC) measurements. BMI was calculated.

The 24-hour ABPM was performed using a BP holter (Contec ABPM50). BP was measured every 30 minutes during the day and every 60 minutes during the night. Diurnal, nocturnal, and 24-hour systolic BP (SBP), diastolic blood pressure (DBP), and mean arterial pressure (MAP) were recorded. The diagnosis of hypertension was made if the mean 24-hour SBP was higher than 130 mmHg and/or the mean 24-hour DBP was higher than 80 mmHg.<sup>[10]</sup> Nocturnal hypertension was defined by an SBP higher than 120 mmHg and/or a DBP higher than 70 mmHg.

The BP variation between day and night was calculated according to the following formula: BP variation (%)=(day BP - night BP)/day BP $\times$ 100. Participants were classified as non-dippers if BP variation was  $<10\%$  and as risers if BP variation was negative.<sup>[7]</sup>

Venous blood samples were collected after an overnight fast of at least 12 hours for the measurement of fasting blood glucose (Enzymatic/hexokinase method), insulin (Chemiluminescence), total cholesterol (Cholesterol oxidase), triglycerides (UV endpoint/Lipase glycerol kinase), and HDL-cholesterol (UV endpoint/DSBmT). Oral glucose tolerance test was performed in non-diabetic women.

Insulin resistance was evaluated using the homeostasis model assessment for insulin resistance (HOMA-IR) index: HOMA-IR=fasting insulin ( $\mu$ U/ml) $\times$ fasting glucose (mmol/l)/22.5. It was defined by a HOMA index superior to 2.5.

The diagnosis of metabolic syndrome was made according to the IDF definition.<sup>[11]</sup>

## Statistical Analysis

All data were analyzed using the Statistical Program for Social Sciences, SPSS Statistics 21.0 (Armonk, NY: IBM Corp.). Continuous variables were expressed as mean±standard deviation (SD) or as median with interquartile (25%–75% percentiles) range (IQR) if not normally distributed. Categorical variables were expressed as percentages. Comparison of continuous variables was made using the Student's t-test and, in case of nonvalidity, the nonparametric Mann-Whitney test. Comparison of categorical variables was made by Pearson's chi-squared test and, in case of nonvalidity, by Fisher's exact bilateral test. Significance was defined as a p-value <0.05.

## Results

Thirty-five women with complete anterior pituitary deficiency and 47 age- and BMI-matched controls were in-

cluded in this study. Their epidemiological, clinical, and biochemical features are shown in Table 1.

The mean duration of Sheehan syndrome was 31.1±10.9 years, with a mean diagnosis delay of 10.6±9.7 years. All patients were treated with hydrocortisone (mean dose: 20.4±3.1 mg/day) and levothyroxine (mean dose: 1.67±0.62 µg/kg/day) and were naïve to GH replacement therapy. Oestroprogestative therapy was prescribed to 16 patients (46%) until the age of 45 years. Compared to controls, women with Sheehan syndrome had a higher prevalence of dyslipidemia and metabolic syndrome.

24-hour ABPM revealed higher 24-hour DBP, 24-hour MAP, day SBP, day DBP, and day MAP in controls than in patients (Table 2). In addition, patients had lower day-night BP variation than controls (Table 2).

**Table 1.** Demographic, clinical, and biochemical characteristics of the study population

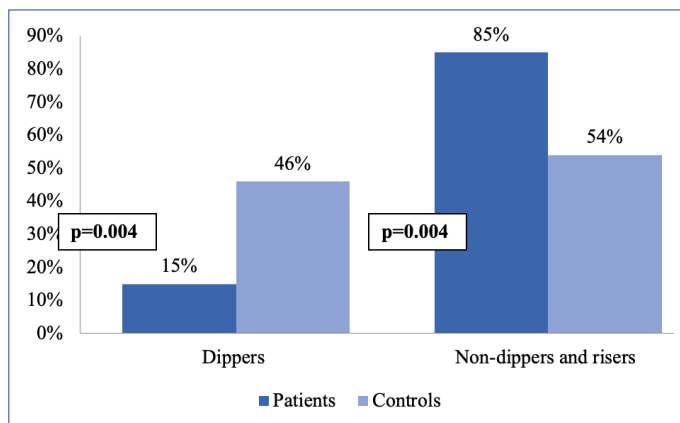
	Patients (n=35)	Controls (n=47)	p
Age, mean±SD (years)	61.3±10.6	60.5±8.5	0.720
Weight, mean±SD (kg)	69.1±15.1	74.4±13.8	0.106
BMI, mean±SD (kg/m <sup>2</sup> )	28.4±5.7	29.8±5.2	0.216
Waist circumference, mean±SD (cm)	99±10.1	95.4±10.5	0.120
Waist circumference ≥ 80 cm (%)	97	85	0.069
Obesity (%)	35	48	0.221
Insulin resistance (%)	31	53	0.050
Diabetes (%)	20	15	0.543
Dyslipidemia (%)	60	36	<b>0.032</b>
Metabolic syndrome (%)	63	38	<b>0.028</b>

n: number; SD: Standard deviation; BMI: Body mass index.

**Table 2.** 24-hour ambulatory blood pressure levels in women with Sheehan syndrome and in controls

	Patients (n=35)	Controls (n=47)	p
24-h SBP, mean±SD (mmHg)	131.6±16.2	138.2±18.7	0.093
24-h DBP, mean±SD (mmHg)	75.3±11.7	81.6±12.8	<b>0.025</b>
24-h MAP, mean±SD (mmHg)	94.0±12.0	100.4±14.1	<b>0.031</b>
Day SBP, mean±SD (mmHg)	131.6±16.6	141.2±18.7	<b>0.019</b>
Day DBP, mean±SD (mmHg)	76.4±12.2	85.5±13.8	<b>0.003</b>
Day MAP, mean±SD (mmHg)	94.8±12.4	104.0±14.8	<b>0.004</b>
Night SBP, mean±SD (mmHg)	129.9±20.2	129.0±20.4	0.856
Night DBP, mean±SD (mmHg)	72.9±15.0	72.5±12.8	0.910
Night MAP, mean±SD (mmHg)	91.9±15.8	91.3±14.7	0.881
SBP variation, median (IQR) (%)	1.8 (-3.8-6.3)	8.4 (4.2-13.2)	<b>&lt;0.001</b>
DBP variation, median (IQR) (%)	6.5 (-3.4-15.6)	16.8 (9.2-21.4)	<b>0.029</b>
MAP variation, median (IQR) (%)	4.6 (-4.8-10.6)	12.6 (7.9-16.8)	<b>0.007</b>

n: number; h: hour; SBP: systolic blood pressure; DBP: diastolic blood pressure; MAP: mean arterial pressure; SD: standard deviation; IQR: interquartile range.



**Figure 1.** Blood pressure dipping patterns in patients with Sheehan syndrome and in controls.

The prevalence of newly diagnosed hypertension was 68% in patients and 62% in controls ( $p=0.520$ ). The prevalence of nocturnal hypertension was 38% in patients and 3% in controls ( $p=0.002$ ).

Figure 1 shows the BP dipping pattern in patients with Sheehan syndrome and controls. The majority of patients with Sheehan syndrome (85%) had an abnormal BP dipping pattern compared to 54% of controls ( $p=0.004$ ). Sheehan syndrome was positively associated with the abnormal BP dipping pattern (Odds Ratio=4.7, 95% confidence interval: 1.54–14.33,  $p=0.004$ ).

## Discussion

The prevalence of newly diagnosed hypertension was comparable between patients with Sheehan syndrome and controls. However, patients had a significantly higher prevalence of nocturnal hypertension and abnormal BP dipping patterns, including non-dipping and rising profiles, compared to controls.

Epidemiological studies have shown that patients with hypopituitarism, especially women, have an increased standardized mortality ratio compared to the general population.<sup>[12,13]</sup> This was largely due to a higher incidence of cardiovascular complications.<sup>[12–14]</sup> Visceral adiposity, insulin resistance, dyslipidemia, and hypertension were reported to be more prevalent in patients with hypopituitarism than in the general population.<sup>[6]</sup> In the present study, dyslipidemia and metabolic syndrome were significantly more frequent in women with Sheehan syndrome than in controls. Although the prevalence of newly diagnosed hypertension was comparable between the two groups, patients had a significantly higher prevalence of nocturnal hypertension than controls. Furthermore, the abnormal BP dipping pattern was significantly more prevalent in women with Sheehan syndrome than in controls. Patients with Sheehan syn-

drome were 4.7 times more likely than controls to exhibit non-dipping or rising profiles. A high prevalence of the BP non-dipping pattern was reported in the study by Krzyzanowska et al.,<sup>[8]</sup> which included 61 patients with hypopituitarism and 20 healthy controls. Non-dipping was observed in 51% of patients with hypopituitarism compared to none of the controls. Moreover, the prevalence of non-dipping was higher in patients with untreated GH deficiency (69%) than in GH-treated patients (46%).<sup>[8]</sup> The higher prevalence of non-dipping in our study may be attributed to the fact that all enrolled patients had GH deficiency and were naïve to GH replacement therapy.

GH deficiency appears to be indirectly involved in the increase in BP through several mechanisms: increased visceral fat mass, inflammation, insulin resistance, atherogenic dyslipidemia, increased vascular resistance, endothelial dysfunction, and impaired nitric oxide synthesis.<sup>[15,16]</sup> However, the exact role of GH deficiency in reducing nocturnal BP decline remains unclear and is subject to ongoing debate. Some authors reported a high prevalence of non-dippers in patients with GH deficiency,<sup>[17]</sup> while other studies showed an increase in the prevalence of non-dippers and nocturnal DBP after GH replacement.<sup>[18,19]</sup>

Hydrocortisone treatment is another factor that can modulate circadian BP variation in patients with hypopituitarism.<sup>[20]</sup> Complications of long-term treatment for adrenal insufficiency are still unclear.<sup>[21]</sup> The regimens currently used for the replacement of adrenal insufficiency, whether primary or secondary, do not reproduce endogenous hormone production. Cortisol secretion follows a circadian rhythm, with a morning peak around 2 hours before awakening and a nadir around 11 pm. In its classic formulation, hydrocortisone does not reproduce the nycthemeral secretion of cortisol. Moreover, there are no reliable biological markers for monitoring patients on hydrocortisone. As a result, inappropriate hydrocortisone substitution is very frequent in these patients and may contribute to the development of metabolic and cardiovascular disorders, in particular hypertension.<sup>[21]</sup> The hypertensive effect of glucocorticoids is thought to result from their permissive action on vasoactive agents, such as angiotensin II and catecholamines, and their mineralocorticoid action secondary to saturation of the 11 $\beta$ -hydroxysteroid dehydrogenase isoenzyme type 2.<sup>[22]</sup>

Behan et al.<sup>[23]</sup> showed that loss of physiological nocturnal BP lowering was more frequent in patients with hypopituitarism receiving high doses of hydrocortisone, and that lower doses may be associated with lower arterial stiffness and more physiological nocturnal BP lowering. Dunne et al.,<sup>[24]</sup> in their study including 13 patients with hypopituitarism on routine replacement therapy and 20 age- and BMI-

matched controls, showed that mean 24-hour BP, day, and night-time BP levels were lower in patients than in controls and did not change significantly after reduction in hydrocortisone dose. This finding may be potentially limited by the small sample size.

In the present study, all patients had gonadotropin deficiency, and only 42% had received estrogen and progesterone therapy. Estrogen deficiency has been shown to be associated with atherosclerosis and hypertension.<sup>[25,26]</sup> Seely et al.,<sup>[27]</sup> in their study including 15 healthy postmenopausal women, revealed that transdermal estradiol with or without progesterone had a hypotensive effect, particularly at night. In line with these findings, Zacharieva et al.<sup>[28]</sup> showed that combined estrogen-progestogen replacement therapy led to lower ambulatory BP in normotensive postmenopausal women, with a more significant decrease observed in nocturnal BP levels. The recovery of the nocturnal dipping pattern was suggested to reduce cardiovascular risk.<sup>[28]</sup>

In the present study, all patients had thyrotropin deficiency and were treated with levothyroxine. The effect of thyrotropin deficiency on the BP dipping pattern is not known. Few studies have shown a higher number of non-dippers in patients with primary hypothyroidism compared to control groups and suggested that elevated thyroid-stimulating hormone (TSH) levels may increase the risk of non-dipping in normotensive patients.<sup>[29]</sup>

Our study aimed to assess the BP dipping pattern in women with Sheehan syndrome. We included a homogeneous population of women with complete anterior pituitary deficiency and age- and BMI-matched controls. We used 24-hour ABPM, which is a more precise tool and more predictive of cardiovascular complications. However, our study was limited by its cross-sectional design and the relatively small sample size. In addition, sodium intake was not determined in patients and controls. This may influence our results since high sodium intake has been reported to be associated with a non-dipping BP pattern.<sup>[30]</sup> Furthermore, sleep disorders and sleep apnea syndrome were not investigated in patients and controls. Obstructive sleep apnea is associated with a non-dipping pattern and nocturnal hypertension.<sup>[31,32]</sup> This was explained by sympathetic excitation due to intermittent hypoxia and sleep fragmentation, upregulation of the renin-angiotensin-aldosterone system, increased oxidative stress, and endothelial dysfunction.<sup>[31,32]</sup>

## Conclusion

Although the prevalence of newly diagnosed hypertension was comparable between patients and controls, women with Sheehan syndrome had a higher prevalence of noc-

turnal hypertension and non-dipping profiles. These findings may explain the increased risk of cardiovascular mortality in this population.

## Disclosures

**Ethics Committee Approval:** The study was approved by La Rabta University Hospital Ethics Committee (No: CEBM.EPS. HR/24/2021, Date: 14/04/2021).

**Informed Consent:** Patients and controls were informed about the study's aims and were included after signing a consent form.

**Conflict of Interest:** The authors declare that there is no conflict of interest.

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