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Case Report

Nocturnal Hypotension in a Patient with Morning Hypertensive Urgency: A Case Report

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Abstract

In both healthy normotensive individuals and in most hypertensive patients, daily blood pressure (BP) is characterized by a circadian rhythm, the night-time BP values being 10%–20% lower than daytime ones largely because of a reduction in sympathetic cardiovascular (CV) tone and a parallel increase in vagal tone during the sleep period [1,2]. We report a case of a 91-year-old female who presented with neurogenic symptoms, vertigo and dizziness more in the early morning. The patient known diabetic, diabetic nephropathy and hypertensive. The patient admitted in ordinary medical ward for more investigations. The patient referred to neuro medicine consultant and CT brain done, the CT brain only released age related brain atrophy then the blood pressure rising and the patient transferred to ICU for close monitoring and blood pressure control. In ICU the blood pressure controlled by iv antihypertensive medication with increasing the doses of her oral antihypertensive medication then we found that the blood pressure markedly dropped only during the sleep time in night then rising in the morning. The oral antihypertensive medications changed to long acting calcium channel blocker after that the neurogenic symptoms, vertigo and dizziness started to be improved with blood pressure controlled. The patient discharged from the hospital in good condition. We are reporting this case because of its rarity and improving the symptoms on long acting calcium channel blocker.

Introduction

Definitions

On the basis of the results of observational studies and their meta-analyses 4 circadian BP phenotypes are usually identified, that is (i) dipping: a decrease ≥10% and <20% in mean BP at night compared to mean daytime values; (ii) non-dipping: <10% BP reduction of night-time vs. daytime BP; (iii) reverse dipping: a rise rather than a fall of night-time BP; and (iv) extreme dipping (ED): a night-time BP fall ≥ 20% [3]. The ED pattern was defined as follows: (i) a night time SBP and DBP reduction of both SBP and DBP ≥ 20% compared to daytime values; (ii) a night time SBP reduction ≥ 20% compared to daytime values; (iii) a night time DBP reduction ≥ than 20% compared to daytime values. According to the 2018 European Society Hypertension/European Society Cardiology guidelines, a mean night-time SBP ≥120 mm Hg and/or DBP ≥ 70 mm Hg is the value dividing nocturnal BP normality from elevation [4]. This, and the data of other specific studies, led to the definition of nocturnal hypotension as a mean SBP < 90 mm Hg and/or DBP < 50 mm Hg [5,6]. In a secondary analysis, mean night-time SBP values < 100 mm Hg and/or DBP values < 60 mm Hg were used [7]. These circadian BP phenotypes have been shown to be associated with different risks of subclinical hypertension mediated organ damage (HMOD), CV (cardiovascular) outcomes and all-cause mortality [8,9] in ED, an excessive drop in nocturnal BP may potentially trigger cardiac and/or cerebral hypo perfusion and thus vital organ ischemia leading to an increased CV risk [10,11]. A previous meta-analysis demonstrated that the nocturnal BP values measured by home BP monitoring (HBPM) were similar to those measured by ambulatory BP monitoring (ABPM) and showed a comparable relationship with target organ damage compared to those measured by ABPM, which has been the gold standard for measurement of nocturnal BP levels. A reduction of nocturnal ambulatory BP (ABP) has been linked to cardiovascular protection, and thus, nocturnal home BP (HBP) reduction could be an important strategy for the management of hypertension [12-15].

Case Report

A 91-year-old female presented to our hospital complaining from dizziness and vertigo long time ago with recurrent reviewing the ER with this complain in last months the patient known diabetic type II on oral medication and hypertensive on beta blocker with ACEI. The patient was conscious and oriented. Admitted in the ordinary medical ward as a case of vertigo for more investigations. She was reviewed by ENT consultant 10 days ago and she was free from ENT causes. After the hospital admission CT brain done and released just age related brain atrophy and the patient reviewed by neurology consultant and she reported free from the neurological side. After less than 48 hours from the hospital admission the blood pressure rising up to 180/110 and the patient transferred to ICU as a case of hypertensive urgency for close monitoring and blood pressure control. In ICU, we noticed that the blood pressure markedly dropped during night-time reached up to 80/40 and in the morning the blood pressure rising again with increasing the dizziness and vertigo in the early morning. In the ICU, the blood pressure was controlled initially by intravenous medication (nitro-glycerine) then it is stopped and the blood pressure controlled on oral medications (amlodipine, aldomet, valsartan and bisoprolol) but the blood pressure still fluctuating (rising during day time and dropped during night-time). After that the oral antihypertensive medications changed to long acting calcium channel blocker (nifidpin 90 mg once daily oral) after that the blood pressure controlled all over the time with marked improvement in the neurogenic symptoms, dizziness and vertigo. The patient transferred from ICU to the ordinary medical ward then discharged home in good condition.

Discussion

Nocturnal hypotension is a rare condition associated with cardiovascular target organ damage and cardiovascular mortality in hypertensive patients. Abnormal circadian blood pressure patterns associated with elevated sleep blood pressure include no dipping and reverse dipping, both of which are associated with increased target-organ damage and adverse cardiovascular outcomes. Although nocturnal hypotension is considered the key factor responsible for the increased cardiovascular risk associated with the extreme dipping (ED) pattern, no information is available on its prevalence in this setting. In a previous study A total of 7,074 individuals 24-hour ambulatory BP recordings (ABPM) from untreated individuals with a history of hypertension and treated hypertensive individuals referred to a single out-patient hypertension centre (Clinical Research Unit Meda, institute Auxologico Italiano and University of Milano-Bicocca) by their general practitioners during a 5-year period (November 2013 to December 2018) were analysed. Individuals aged <18 years, with suboptimal ABPM recordings (<70 % successful readings) or doing shift work were excluded from data analysis. Of those patients, 61% exhibited a nocturnal decline ≥20% in both SBP and DBP. In the remaining 39% patients, a decline ≥20% was limited to only DBP, whereas no subject showed an excessive decline of SBP only. In the total sample, prevalence rates of nocturnal hypotension (i.e., mean night-time SBP <90 mm Hg and/or DBP < 50 mm Hg) were 9.1% [16].

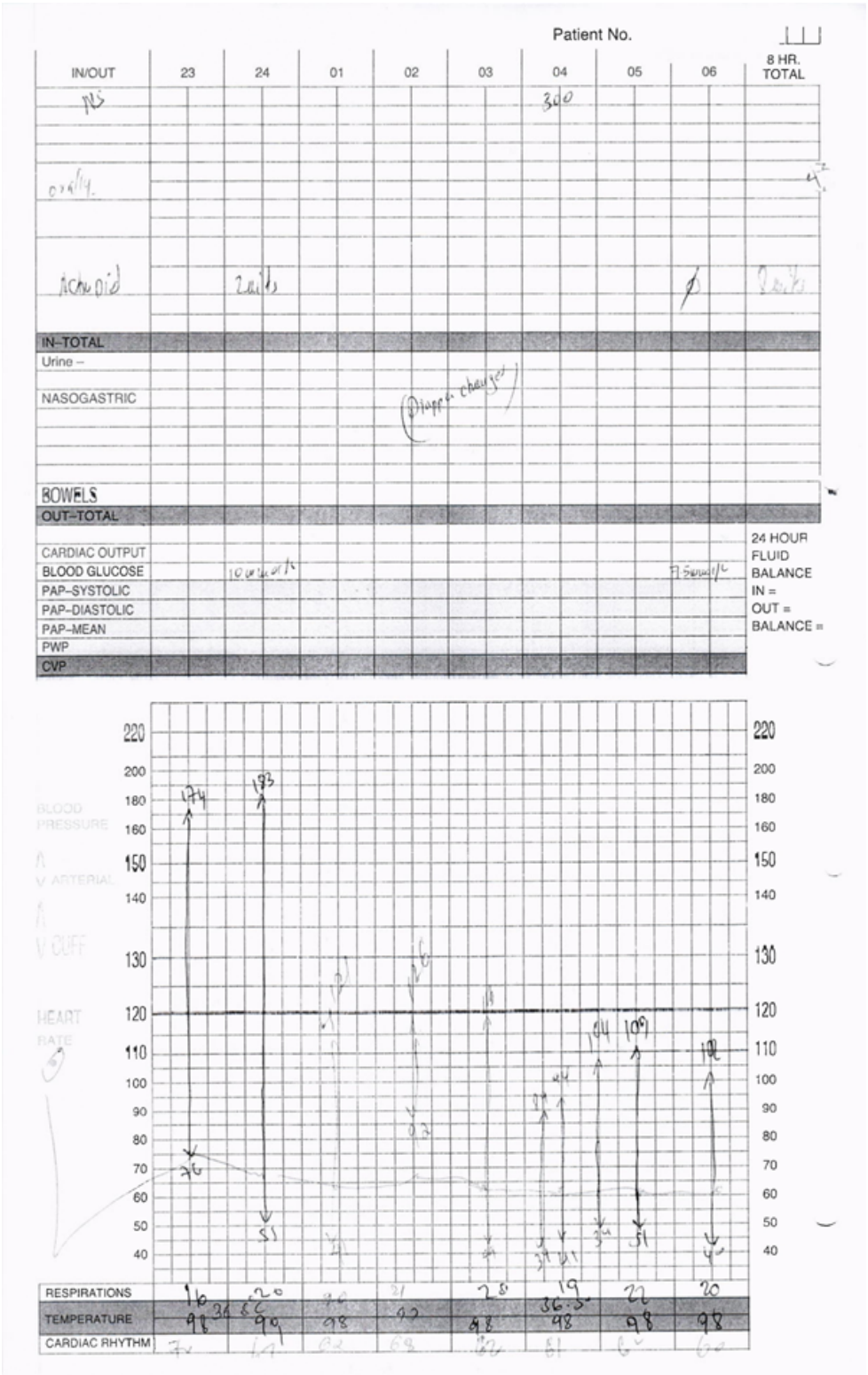

Figure 1: ICU vitals showing morning hypertension and nocturnal hypotension.

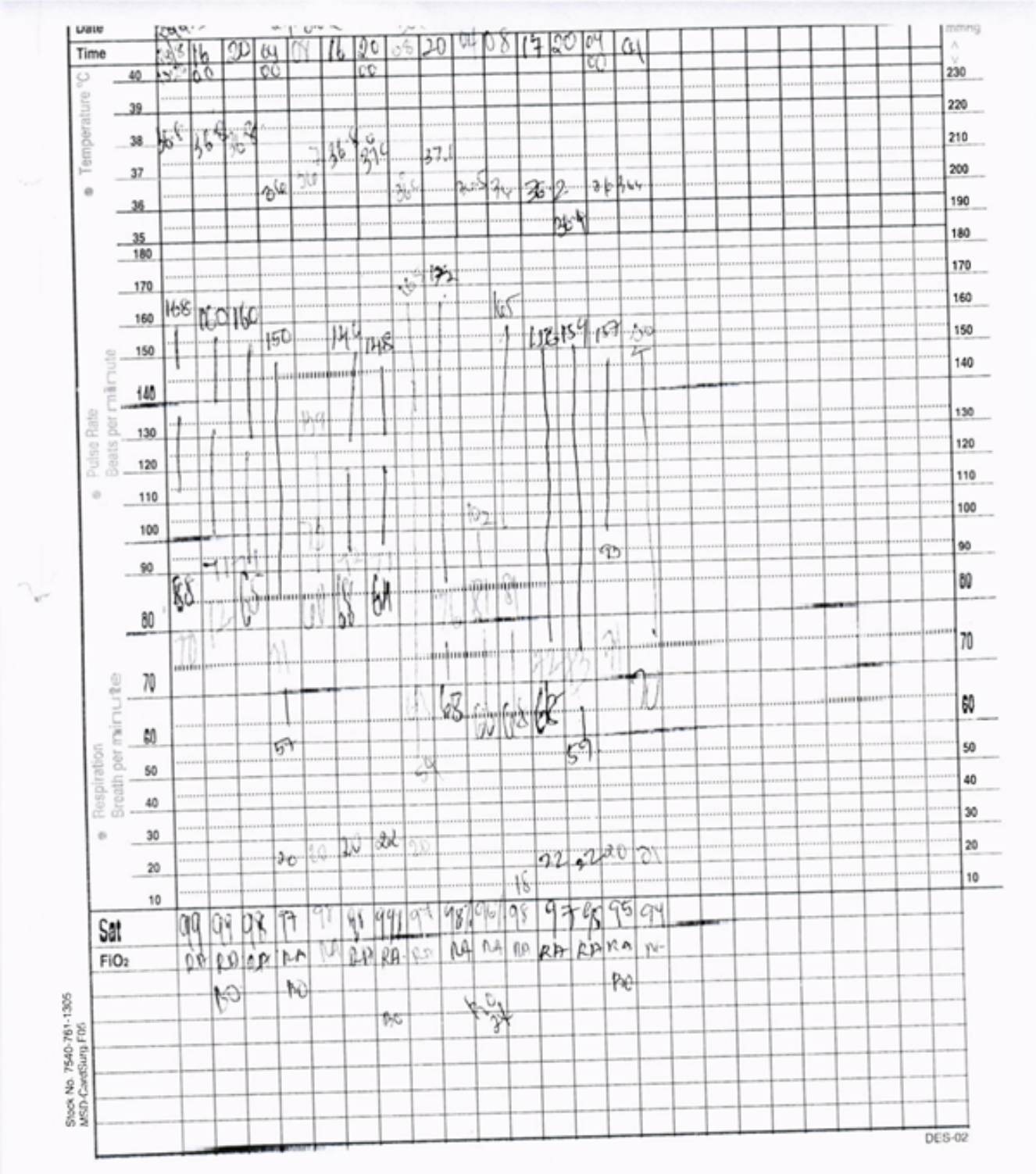

Figure 2: Medical ward vitals after short acting antihypertensive medications changed into long-acting medications.

Although there are certain technical problems in determining nocturnal BP by ambulatory BP monitoring, the information provided on nocturnal BP has possible clinical significance. Antihypertensive effects of drugs with different pharmacologic properties positively correlate with basal ambulatory BP. Therefore, there is a critical BP level, at which the antihypertensive effect disappears. The critical BP level for each drug is in normal BP range but not in the hypotensive range. Therefore, an antihypertensive regimen would be safe even in extreme-dipper hypertension without excessive nocturnal hypotension, and might even be beneficial because of the decreasing amplitude and speed of the nocturnal BP decline. We conclude that an antihypertensive drug regimen should control BP throughout a 24-h period regardless of circadian BP variation. The psychological factors may contribute to the description and classification of patients who fail to exhibit adaptive nocturnal blood pressure dipping. It is interesting to note that the relationship between emotional dysregulation, i.e., difficulty identifying and describing feelings, and excessive BP reduction during sleep (extreme dipping) underlines that some psychological aspects could affect the absence of dipping and a maladaptive increase of the dipping phenomenon [17,18,19]. Nocturnal hypertension can be treated with several approaches that include both lifestyle changes, such as sodium restriction and potassium supplementation, and pharmacological treatments, primarily with bedtime dosing of antihypertensive agents [20]. Since nocturnal BP has been strongly associated with cardiovascular mortality and is easily assessed by HBPM, nocturnal HBPM has the potential to be a generalized method of monitoring. In our case we found that the use of long acting calcium channel blocker (nifdipin), significantly reduced morning systolic BP (SBP) in patients with higher baseline morning SBP and restored the abnormal BP dipping status (including extreme dipping) toward a normal dipping pattern. These results indicated that nifidipin could control the ABP level of patients with increased sympathetic activity and that it does not lower the nocturnal BP levels of patients whose nocturnal BP has already been controlled.

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