The influence of circadian pattern and obstructive sleep apnea of blood pressure in nocturnal arrhythmias in hypertensive patients

Authors:
R Marinheiro¹, L Parreira¹, P Amador¹, D Mesquita¹, J Farinha¹, M Fonseca¹, A Esteves¹, A Pinheiro¹, S Sousa¹, M Silva¹, C Lopes¹, A Fernandes¹, A Guerreiro¹, D Chambel¹, R Caria¹, ¹Hospital Center of Setubal - Setubal - Portugal,

Topic(s):
Hypertension – Epidemiology, Prognosis, Outcome

Citation:
Introduction: There is growing evidence suggesting hypertension is related to the occurrence of arrhythmias. When occurring predominantly during the night, two potential entities commonly present in hypertensive patients could be related with increased arrhythmogenesis: (1) the lack of normal nocturnal dipping of blood pressure (BP) (non-dipping pattern of hypertension) or (2) obstructive sleep apnea (OSA). Thus, nocturnal arrhythmias (NAs) can identify hypertensive patients with OSA and/or non-dipping pattern, both related with adverse outcomes.

Aims: To determine if NAs are related with non-dipping hypertension, OSA or both.

Methods: We studied hypertensive patients who performed ambulatory blood pressure monitoring (ABPM) and also polysomnography and 24-hour Holter monitoring. Non-dipping pattern was considered when nocturnal BP reduction was inferior to 10%. Based on Holter monitoring, NAs were present when atrial fibrillation, frequent premature atrial contractions (PACs) (>30 PACs/hour), runs of >4 consecutive PACs, frequent premature ventricular contractions (PVCs) (>30 PVCs/hour) or runs of > 4 consecutive PVCs were present predominantly during sleeping hours. During polysomnography, apnoea/hypopnoea index (AHI) and oxygen saturation (SaO2) were analysed. Moderate to severe OSA was considered when AHI >15.

Results: We studied 104 patients [median age 62 (54-70) years, 65% male): 42 (40%) had moderate to severe OSA (median AHI=11 (6-26), mean SaO2=94% (92-95)] and 64 (61%) were non-dippers. NAs occurred in 18 patients (17%) and they were independently associated with AHI (Odds Ratio (OR) for a one unit increase 1.04, 95% confidence interval (CI) 1.01-1.07, p=0.03) but not with SaO2 (OR 0.96, CI 0.78-1.19, p=0.73) nor non-dipping pattern (OR 1.23, CI 0.38-3.98, p=0.72). No interaction was found between OSA and non-dipping hypertension (p=0.35). In patients with dipping pattern (n=40), AHI was higher in NAs patients comparing with no NAs patients (median AHI 29 versus 10, p=0.04), while in those with non-dipping pattern (n=64), AHI was not statistically different between patients with and without NAs (21 versus 11, p=0.12) (figure).

Conclusion: In this population of hypertensive patients, the presence of NAs was associated with OSA severity (i.e AHI), but not with the non-dipping pattern of hypertension. The importance of obstructive events in arrhythmogenesis seemed to be more pronounced in dipping patients, suggesting the abnormal high blood pressure during the night may also have some impact on NAs in non-dipping patients. Overall, our results suggest that OSA screening should be considered when nocturnal arrhythmias are detected in hypertensive patients, but ABPM should not be forgotten since multiple mechanisms can be involved in nocturnal arrhythmogenesis.
Abstract: P3563

The influence of circadian patterns and obstructive sleep apnea on blood pressure in nocturnal arrhythmias in hypertensive patients


1 Hospital Center of Setubal - Setubal - Portugal,

Topic(s): Hypertension – Epidemiology, Prognosis, Outcome

Introduction: There is growing evidence suggesting hypertension is related to the occurrence of arrhythmias. When occurring predominantly during the night, two potential entities commonly present in hypertensive patients could be related with increased arrhythmogenesis: (1) the lack of normal nocturnal dipping of blood pressure (non-dipping pattern of hypertension) or (2) obstructive sleep apnea (OSA). Thus, nocturnal arrhythmias (NAs) can identify hypertensive patients with OSA and/or non-dipping pattern, both related with adverse outcomes.

Aims: To determine if NAs are related with non-dipping hypertension, OSA or both.

Methods: We studied hypertensive patients who performed ambulatory blood pressure monitoring (ABPM) and also polysomnography and 24-hour Holter monitoring. Non-dipping pattern was considered when nocturnal BP reduction was inferior to 10%. Based on Holter monitoring, NAs were present when atrial fibrillation, frequent premature atrial contractions (PACs) (>30 PACs/hour), runs of >4 consecutive PACs, frequent premature ventricular contractions (PVCs) (>30 PVCs/hour) or runs of >4 consecutive PVCs were present predominantly during sleeping hours. During polysomnography, apnoea/hypopnoea index (AHI) and oxygen saturation (SaO2) were analysed. Moderate to severe OSA was considered when AHI >15.

Results: We studied 104 patients [median age 62 (54–70) years, 65% male]: 42 (40%) had moderate to severe OSA (median AHI=11 (6–26), mean SaO2=94% (92–95)) and 64 (61%) were non-dippers. NAs occurred in 18 patients (17%) and they were independently associated with AHI (Odds Ratio (OR) for a one unit increase 1.04, 95% confidence interval (CI) 1.01–1.07, p=0.03) but not with SaO2 (OR 0.96, CI 0.78–1.19, p=0.73) nor non-dipping pattern (OR 1.23, CI 0.38–3.98, p=0.72). No interaction was found between OSA and non-dipping hypertension (p=0.35). In patients with dipping pattern (n=40), AHI was higher in NAs patients comparing with no NAs patients (median AHI 29 versus 10, p=0.04), while in those with non-dipping pattern (n=64), AHI was not statistically different between patients with and without NAs (21 versus 11, p=0.12) (figure).

Conclusion: In this population of hypertensive patients, the presence of NAs was associated with OSA severity (i.e. AHI), but not with the non-dipping pattern of hypertension. The importance of obstructive events in arrhythmogenesis seemed to be more pronounced in dipping patients, suggesting the abnormal high blood pressure during the night may also have some impact on NAs in non-dipping patients. Overall, our results suggest that OSA screening should be considered when nocturnal arrhythmias are detected in hypertensive patients, but ABPM should not be forgotten since multiple mechanisms can be involved in nocturnal arrhythmogenesis.

Box-and-Whisker plot analysis

AHI: apnoea/hypopnoea index