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Original Article

Degree of pineal calcification (DOC) is associated with polysomnographic sleep measures in primary insomnia patients

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Abstract

Objective: Melatonin plays a key role in the proper functioning of the circadian timing system (CTS), and exogenous melatonin has been shown to be beneficial in cases of CTS and sleep disturbances. Nevertheless, the concept of "melatonin deficit" has yet to be defined. The aim of our study was, therefore, to determine the relationship between the degree of pineal calcification (DOC) and a range of sleep parameters measured objectively using polysomnography (PSG).

Methods: A total of 31 outpatients (17 women, 14 men, mean age 45.9 years; SD 14.4) with primary insomnia were included in our study. Following an adaptation night, a PSG recording night was performed in the sleep laboratory. Urine samples were collected at predefined intervals over a 32-h period that included both PSG nights. The measurement of 6-sulphatoxymelatonin (aMT6s) levels was determined using ELISA. DOC and volume of calcified pineal tissue (CPT) and uncalcified pineal tissue (UPT) were estimated by means of cranial computed tomography.

Results: UPT was positively associated with 24-h aMT6s excretion ($r = 0.569$; $P = 0.002$), but CPT was not. After controlling for age, aMT6s parameters, CPT, and UPT did not correlate with any of the PSG parameters evaluated. In contrast, DOC was negatively associated with REM sleep percentage ($r = -0.567$, $P = 0.001$), total sleep time ($r = -0.463$, $P = 0.010$), and sleep efficiency ($r = -0.422$, $P = 0.020$).

Conclusion: DOC appears to be a superior indicator of melatonin deficit compared to the absolute amount of melatonin in the circulation. High DOC values indicate changes predominantly in the PSG parameters governed by the circadian timing system. DOC may thus serve as a marker of CTS instability.

Keywords: Sleep; Pineal gland; Melatonin; Degree of pineal calcification (DOC); Polysomnography; Rapid eye movements (REM); Age

1. Introduction

Studies on the role played by endogenous melatonin in humans have yielded some important contradictions [1]. On one hand, with the only relevant excretion site for circulating melatonin being the pineal gland [2], pinealectomy and pharmacological suppression of melatonin excretion have been shown

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