Obstructive Sleep Apnea and Nocturnal Hypertension

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Abstract

Obstructive sleep apnea (OSA) is an important but often underrecognized cause of nocturnal hypertension, with well-defined neurocardiovascular mechanisms. Intermittent hypoxia and recurrent episodes of apnea during sleep lead to chronic activation of the sympathetic nervous system, resulting in increased nocturnal blood pressure and loss of the physiological circadian blood pressure dipping pattern. Consequently, a *non-dipping* or even *reverse-dipping* profile is frequently observed, which is strongly associated with an increased risk of cerebrovascular events. At the molecular level, intermittent hypoxia induces oxidative stress, activation of inflammatory pathways (including NF-κB signaling), and endothelial dysfunction accompanied by reduced nitric oxide bioavailability. Additionally, impaired baroreceptor function and increased sympathetic activity contribute to the persistence of hypertension even between apneic episodes. Clinically, OSA is strongly associated with resistant hypertension and isolated nocturnal hypertension, with 24-hour ambulatory blood pressure monitoring (ABPM) playing a key diagnostic role. Continuous positive airway pressure (CPAP) therapy has demonstrated a modest but clinically significant effect on reducing nocturnal blood pressure, with the potential to decrease sympathetic hyperactivity and improve endothelial function. In conclusion, OSA represents an important pathophysiological substrate of nocturnal hypertension, emphasizing the need for an interdisciplinary approach to diagnosis and treatment aimed at reducing overall cardiovascular and cerebrovascular risk.

Keywords: obstructive sleep apnea; hypertension; stroke; myocardial infarction; ambulatory blood pressure monitoring (ABPM)

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Opstruktivna apneja u snu i noćna hipertenzija

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Sažetak

Opstruktivna apneja u snu (OSA) važan je, ali često nedovoljno prepoznat uzrok noćne hipertenzije, s jasno definiranim neurokardiovaskularnim mehanizmima. Intermitentna hipoksija i ponavljane epizode apneje tijekom spavanja dovode do kronične aktivacije simpatičkog živčanog sustava, što rezultira porastom noćnog arterijskog tlaka i gubitkom fiziološkog cirkadijanog obrasca noćnog sniženja krvnog tlaka (*dipping*). Posljedično se često bilježi profil izostanka noćnog sniženja krvnog tlaka (*non-dipping*) ili čak njegova paradoksalnog porasta (*reverse dipping*), što je snažno povezano s povećanim cerebrovaskularnim rizikom. Na molekularnoj razini, intermitentna hipoksija inducira oksidativni stres, aktivaciju upalnih putova (uključujući signalizaciju NF-κB) te endotelnu disfunkciju uz smanjenu biološku raspoloživost dušikova oksida. Dodatno dolazi do poremećaja baroreceptorske funkcije i pojačane simpatičke aktivnosti, što pridonosi održavanju hipertenzije i izvan epizoda apneje. Klinički je OSA snažno povezana s rezistentnom hipertenzijom i izoliranom noćnom hipertenzijom, pri čemu kontinuirano 24-satno mjerenje arterijskog tlaka (KMAT) ima ključnu dijagnostičku ulogu. Terapija kontinuiranim pozitivnim tlakom u dišnim putovima (CPAP) pokazuje umjeren, ali klinički značajan učinak na sniženje noćnog krvnog tlaka, uz potencijalno smanjenje simpatičke hiperaktivnosti i poboljšanje endotelne funkcije. Zaključno, OSA predstavlja važan patofiziološki supstrat noćne hipertenzije, što naglašava potrebu za interdisciplinarnim pristupom dijagnostici i liječenju s ciljem smanjenja ukupnog kardiovaskularnog i cerebrovaskularnog rizika.

Ključne riječi: opstruktivna apneja u snu; hipertenzija; moždani udar; srčani udar; kontinuirano mjerenje arterijskog tlaka (KMAT)

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