

OSAHS与冠心病：从病理机制到临床管理的进展

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摘 要

阻塞性睡眠呼吸暂停低通气综合征(OSAHS)是一种发病率日益上升的慢性睡眠呼吸障碍性疾病, 其导致的反复低通气和间歇性缺氧对心血管疾病的发生发展具有显著影响, 尤其与冠心病、高血压、心律失常、心力衰竭等心血管系统疾病关系密切, 已成为冠心病的独立危险因素。本文将对冠心病与OSAHS相关的病理生理机制、诊断、综合治疗等方面的研究进展进行综述。

关键词

阻塞性睡眠呼吸暂停低通气综合征, 冠心病, 病理生理机制, 治疗

OSAHS and Coronary Heart Disease: Progress from Pathological Mechanism to Clinical Management

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Abstract

Obstructive sleep apnea hypopnea syndrome (OSAHS) is a chronic sleep disordered disease with an increasing incidence. Repeated hypoventilation and intermittent hypoxia caused by OSAHS have a significant impact on the occurrence and development of cardiovascular diseases, especially with coronary heart disease, hypertension, arrhythmia, heart failure and other cardiovascular diseases.

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OSAHS has become an independent risk factor of coronary heart disease. This article will review the research progress of pathophysiological mechanism, diagnosis and comprehensive treatment of coronary heart disease and OSAHS.

Keywords

Obstructive Sleep Apnea Hypopnea Syndrome, Coronary Heart Disease, Pathophysiological Mechanism, Treatment

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1. 概述

冠心病(Coronary Heart Disease, CHD)是指由冠状动脉粥样硬化导致血管狭窄、闭塞或冠脉痉挛、微循环功能障碍, 引发心肌缺血缺氧甚至坏死的一类疾病, 主要分为慢性冠状动脉疾病和急性冠状动脉综合征[1]。其主要危险因素可分为可干预因素和不可干预因素。不可干预因素主要包括: 年龄、性别、家族史等, 可干预因素主要包括: 吸烟、肥胖、血脂水平、社会心理因素等[2]。

睡眠呼吸暂停低通气综合征(Sleep Apnea Hypopnea Syndrome, SAHS)是一类以睡眠中反复呼吸暂停和/或低通气, 导致睡眠结构紊乱、白天嗜睡等症状的疾病, 主要分为中枢性睡眠呼吸暂停低通气综合征(Central Sleep Apnea Hypopnea Syndrome, CSAHS)和阻塞性睡眠呼吸暂停低通气综合征(Obstructive Sleep Apnea-Hypopnea Syndrome, OSAHS)两型。其中 CSAHS 临床少见, 发病机制主要与中枢神经系统反馈调节异常及通气中枢不稳定相关, 在心力衰竭、脑卒中患者中较为高发[3], 主要与年龄、潮式呼吸(Cheyne Stokes Respiration, CSR)、药物诱导、高海拔等因素相关[4]。OSAHS 的核心致病机制为上呼吸道阻塞, 进而引发间歇性低氧血症, 发病与年龄、性别、肥胖、腺扁桃体肥大、吸烟等危险因素密切相关[5]。

大量临床研究表明, OSAHS 导致的反复低通气和间歇性缺氧, 可导致自主神经功能紊乱、氧化应激、代谢紊乱, 进而推动冠心病发生与进展, 显著增加心肌梗死、心律失常等不良心血管事件及全因死亡风险[6]。同时, 冠心病患者的心脏结构与功能异常, 也可能加重睡眠呼吸紊乱程度, 从而形成恶性循环[7]。鉴于 OSAHS 在冠心病发生、发展中具有重要作用, 且临床中冠心病合并 OSAHS 患者的漏诊率较高, 因此优化冠心病合并 OSAHS 患者的诊疗策略对改善预后至关重要[8]。本文将围绕 OSAHS 与冠心病相关的病理生理机制、诊断方法、治疗策略等研究进展进行综述。

2. OSAHS 影响冠心病的病理生理机制

2.1. 自主神经功能紊乱与血流动力学异常

OSAHS 患者在夜间睡眠中反复出现低氧血症和高碳酸血症, 导致交感神经持续激活并扰乱自主神经平衡, 这一过程被认为是 OSAHS 诱发并加重心血管疾病的关键环节。交感神经兴奋性增高可导致外周血管收缩、心率加快, 引发夜间血压升高, 增加心脏负荷, 长期持续的交感神经高反应状态还会促进左心室重构与心肌缺血, 加速冠心病的进展[9]。此外, 交感神经过度激活可通过刺激儿茶酚胺过量分泌、激活肾素-血管紧张素-醛固酮系统, 进一步加剧外周血管收缩, 升高左心室后负荷, 最终导致心排量下降及冠脉血流减少; 而夜间反复发生的吸气性气流受阻会使胸腔负压急剧增加, 促进静脉回流增多, 进而升高心脏前负荷[9]。上述血流动力学剧烈波动与心脏前后负荷异常改变, 共同导致心肌耗氧量增加,

心肌缺血加重, 不仅可能诱发 CHD 还会加速病情进展, 增加相关并发症的发生风险[10]。

2.2. 全身炎症反应与氧化应激及代谢紊乱

OSAHS 与全身慢性炎症密切相关, 其主要表现为肿瘤坏死因子 α (TNF- α)、白细胞介素-6 (IL-6) 等促炎因子及 C 反应蛋白 (CRP) 等炎症标志物水平升高, 且这些炎症介质表达强度和夜间低氧血症的严重程度呈正相关[11]。有研究表明, 间歇性缺氧 (Intermittent Hypoxia, IH) 导致的炎症反应, 在颈动脉内膜中层厚度 (IMT) 增加及动脉粥样硬化发生发展中起关键作用[12]。氧化应激是连接 OSAHS 与 CHD 的另一个核心机制: IH 引发的缺氧-再氧化循环可激活缺氧诱导因子 (HIF), 进而触发炎症通路, 加剧血管内皮功能障碍, 同时这一过程还会诱导过量的活性氧 (ROS) 产生, 当 ROS 的生成超出体内抗氧化系统清除能力时, 便会直接破坏内皮细胞、损害血管功能, 推动动脉粥样硬化进行性发展[6][11]。有临床试验显示 OSA 患者体内抗氧化酶活性显著下降, 氧化型低密度脂蛋白 (ox-LDL) 水平升高且内皮一氧化氮 (ENO) 生成受损, 这些变化会进一步加重血管壁损伤与内皮功能障碍, 加速粥样硬化病理演变[13]。值得注意的是: OSAHS 相关的脂代谢紊乱、胰岛素抵抗 (Insulin Resistance, IR) 与氧化应激并非孤立存在, 而是相互协同、放大炎症效应, 通过多重途径加重血管内皮损伤, 最终促进 CHD 进展。甘油三酯-葡萄糖指数 (TyG 指数), 作为 IR 的有效替代评估指标, 已被证实是 OSAHS 患者发生 CHD 的独立危险因素, 其水平可反映冠状动脉粥样硬化的严重程度, 尤其对中重度 OSAHS 患者的 CHD 风险具有重要预测价值[14]。

2.3. 低氧负荷与心肌损伤

夜间间断性低氧负荷是 OSAHS 患者的核心病理特征之一, 其直接或间接导致心肌损伤的机制已得到广泛研究的证实。反复低氧暴露可显著上调心肌细胞活性氧的生成, 诱导脂质过氧化反应异常激活, 造成心肌细胞结构破坏[15]。而间歇性缺氧引发的 ROS 蓄积, 还可进一步靶向作用于细胞核及线粒体等关键细胞器, 通过调控凋亡相关信号通路, 最终导致心肌细胞凋亡、坏死、间质纤维化及功能障碍[16]。从临床表型来看, 间断性低氧可促使肌钙蛋白等心肌损伤生物标志物释放增加, 其中高敏肌钙蛋白 T (hs-TnT) 作为高特异性心肌损伤指标, 其水平变化能反映亚临床心肌损伤状态。一项纳入 1655 名无冠心病及心力衰竭病史受试者的临床研究证实, OSAHS 的严重程度与 hs-TnT 水平呈显著正相关, 这一结果进一步验证了 OSAHS 通过间歇性低氧介导亚临床心肌损伤的潜在机制[17]。

3. OSAHS 对冠心病患者预后的影响

OSAHS 已被证实是冠心病患者病情进展及预后恶化的重要危险因素, 对不同分型的冠心病病情进展均有不同的影响。

对于稳定性冠心病患者, 其冠脉病变及心肌缺血情况相对稳定可控, 而 OSAHS 导致的低氧可刺激冠脉收缩加重冠脉狭窄、增加静息心绞痛等发生几率, 且 OSAHS 与血管重塑进程加速、慢性肾病并发症风险升高及心律失常易感性增加密切相关[18]。中重度的 OSAHS 与稳定性冠心病患者心血管不良事件风险增高显著相关[19]。而在非阻塞性冠心病患者中, OSAHS 更是全因死亡率及主要不良心脑血管事件风险升高的独立危险因素[20]。

美国心脏协会 (AHA) 发布的循证声明已明确 OSAHS 为心血管疾病的独立危险因素, 尤其在急性冠脉综合征 (Acute Coronary Syndrome, ACS) 等冠心病患者中, OSAHS 患病率显著升高, 且对心血管疾病结局存在明确不良影响[21]。大量临床证据表明, 在 ACS 急性期, OSAHS 与心肌肌钙蛋白、N 末端 B 型脑钠肽前体 (NT-proBNP) 等心肌损伤标志物水平呈正相关, 合并严重 OSAHS 的 ACS 患者, 住院期间心律失常、心力衰竭及再梗死等不良事件风险显著升高, 直接影响短期治疗效果与康复进程[22]-[24]。一项回顾性研究对 206 名行支架植入术的患者进行为期 15 个月随访, 其中中度至重度 OSAHS 患者的支架内再

狭窄(In-Stent Restenosis, ISR)发生率显著增加, 表明即使在接受选择性药物洗脱支架置入术后, OSAHS 仍为 ISR 的独立危险因素[25]。

4. OSAHS 合并冠心病患者的筛查与诊断进展

OSAHS 的临床筛查工具日趋多元化。多导睡眠监测(Polysomnography, PSG)作为诊断睡眠呼吸暂停低通气综合征的金标准, 能够精准监测睡眠期间呼吸、脑电、肌电、血氧等多项生理参数对 OSAHS 的严重程度分级具权威性[26]。临床上通常依据呼吸暂停低通气指数(Apnea Hypopnea Index, AHI)来对 OSAHS 的严重程度进行分级, 当 AHI > 5 和 <15/小时睡眠时, 视为轻度; 当 AHI > 15 和 <30/小时睡眠时, 视为中度; 当 AHI 超过 30/小时睡眠时, 视为重度[27]。然而, PSG 的高成本、可及性受限及对睡眠期间对呼吸的干扰导致了 OSAHS 漏诊率高, 在 OSAHS 患者中, 存在一个“心率循环模式”, 呼吸暂停, 心率减慢, 呼吸暂停结束和过度通气开始, 心率增快[28]。阻塞性和中枢性睡眠呼吸暂停患者的自主神经功能障碍和心律失常已有不少研究已经提出了基于心电图的 OSAHS 检测方法, 以自动化多导睡眠图程序并减少其不适感。在 24 小时心电图动态心电图监测期间测量胸阻抗和心率变异性, 可以计算出估计的呼吸暂停低通气指数(Estimated Apnea Hypopnea Index, eAHI), 可用于筛查测试来诊断 OSAHS 患者[29]。近年来家庭睡眠呼吸监测(home sleep apnea test, HSAT)技术进步显著, 尤其优势在于便携性、可重复性及患者依从性提升, HSAT 可用于疑似 OSAS 患者的初步筛查, 准确率达到 90%。但对于中度至重度 OSAHS 患者, 准确率低于 80%, 对于通过 HSAT 诊断为 OSAHS 的患者, 建议安排 PSG 以确定 OSAHS 的严重程度并给予适当的治疗[30]。

5. OSAHS 合并冠心病的综合治疗策略

5.1. 基础治疗: 生活方式干预

CHD 合并 OSAHS 患者的生活方式干预尤为重要, 无论持续气道正压通气(Continuous Positive Airway Pressure, CPAP)治疗依从性及疗效如何, 生活方式干预均为 OSAHS 患者的优先干预策略, 这样才能与 CPAP 等针对性治疗共同实施来获取最大化的疗效[31]。超重与肥胖作为冠心病合并 OSAHS 的主要危险因素, 通过减重可以减少咽部脂肪堆积、改善气道通畅性从而降低呼吸暂停事件发生率, 并且对血压、胰岛素抵抗及血脂异常等 CHD 相关危险因素产生正向调控作用[32]。心理干预与社会支持也是 CHD 合并 OSAHS 患者基础治疗的重要辅助手段, 心理干预可有效改善患者压力与焦虑情绪, 改善睡眠质量, 间接提升慢病管理依从性[33]。良好的社会支持结合长期随访管理, 能协同强化生活方式干预效果、精准控制危险因素, 最终降低再住院率并提升患者生活质量, 从而更高效地实现慢病综合控制目标[34]。

5.2. CPAP 及其对冠心病预后的影响

CPAP 是指在睡眠过程中通过面罩向气道持续输送设定的压力和气流, 支撑上气道保持开放, 避免塌陷, 从而改善气道阻塞、纠正间歇性低氧, 已成为阻塞性睡眠呼吸暂停低通气综合征的核心治疗手段。临床研究证实, OSAHS 患者 CPAP 能有效改善患者夜间高血压、降低动脉僵硬度, 并减轻全身炎症反应, 而这些因素均为 CHD 进展的关键驱动因素[35] [36]。OSAHS 严重程度与冠状动脉病变程度呈正相关, 规律 CPAP 治疗可显著改善 CHD 患者长期预后, 尤其对中重度 OSAHS 合并 CHD 人群, 能有效减少主要不良心血管事件(Major Adverse Cardiovascular Events, MACE)及全因死亡风险[37] [38]。长期规律 CPAP 治疗可显著缩小左心室收缩末期及舒张末期内径, 改善患者心功能储备, 从而为改善 CHD 预后奠定基础[39]。CPAP 在改善睡眠结构及提升患者整体生活质量方面效果确切, 并为 CHD 合并 OSAHS 患者的综合管理提供支持[40]。但对于行冠脉血运重建后的患者而言, CPAP 虽能部分改善血管内皮环境,

但高 CPAP 水平可能上调促炎因子、下调心脏保护性因子, 进而削弱部分心血管获益[41]。因此并非所有患者均能从 CPAP 治疗中等比例获益, 需结合 OSAHS 严重程度、治疗依从性来调整治疗方案。尽管 CPAP 在短期危险因素调控中效果明确, 但在长期减少 MACE 事件方面, 仍缺乏足够样本量、长随访周期的前瞻性随机对照试验提供高级别证据支持[40]。

5.3. 药物协同治疗(心血管药物与呼吸调节药物应用现状)

心血管保护药物在 CHD 合并 OSAHS 患者中的基础作用不可替代。抗血小板药物、他汀类降脂药、 β 受体阻滞剂及血管紧张素转换酶抑制剂常规用于二级预防, 并可减轻 OSAHS 相关缺氧对心肌的损伤风险[31] [42]。虽然 PAP 可以非常有效地减少阻塞性呼吸暂停和低通气的数量, 但其对预防不良心血管后果的影响仍然存在争议, 并且治疗依从性通常很差。因此, 有必要采用新的治疗方案来帮助那些无法坚持气道正压治疗的患者[43]。部分药物如选择性多巴胺再摄取抑制剂、选择性去甲肾上腺素抑制剂、联合抗胆碱能药物以及 Orexin 激动剂, 主要改善 OSAHS 患者呼吸指标和提高睡眠质量方面, 可能有一定的辅助改善效果, 但缺乏大型临床随机对照试验验证, 长期获益与安全性需要进一步研究[40]。

6. 展望

OSAHS 通过间歇性低氧诱发自主神经功能紊乱、血流动力学异常、氧化应激、全身炎症及代谢紊乱、心肌细胞损伤, 对不同亚型的冠心病均存在显著不良影响, 是冠心病发生、进展及预后恶化的重要独立危险因素。OSAHS 与冠心病共病会显著增加心肌损伤、支架内再狭窄、全因死亡等风险。PSG 作为金标准可精准分级 OSAHS (依据 AHI 指标), 但存在可及性低、患者依从性差等问题; HSAT 及心电图衍生的 eAHI 指标为筛查提供新路径, 临床需结合患者实际情况选择适合的诊断工具。治疗方面, 生活方式干预是基础, 结合长期规律的 CPAP 治疗能有效改善 OSAHS 合并冠心病患者的心功能、降低血压及炎症反应, 减少不良心血管事件, 药物协同治疗(心血管保护药物为主)及气道重建手术(针对 PAP 依从性不佳者)可作为补充。在冠心病临床诊疗中, 对可疑 OSAHS 人群实施规范筛查, 是优化治疗策略、改善患者长期预后的关键环节。

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