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卒中后认知障碍的相关危险因素及治疗的研究进展

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摘 要

卒中后认知障碍(post-stroke cognitive impairment, PSCI)是指在脑卒中后6个月内出现达到认知障碍诊断标准的一系列综合征，是脑卒中的主要并发症之一，影响超过三分之一的脑卒中患者，不仅影响脑卒中后患者的生活质量，也影响患者对治疗方案的依从性，威胁他们的生活质量并增加残疾和死亡的风险。其影响因素众多，这篇综述就PSCI的发病机制、危险因素和治疗方法进行概述。主要包括：年龄、教育程度、经济收入、血压、血糖等对PSCI的影响及目前常见的治疗方法。为PSCI的早期诊断及预防提供思路。

关键词

脑卒中，卒中后认知障碍，危险因素，治疗

Research Progress on Risk Factors and Treatment Related to Cognitive Impairment after Stroke

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Abstract

Post-stroke cognitive impairment (PSCI) refers to a series of syndromes that meet the diagnostic

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criteria for cognitive impairment within 6 months after stroke, and is one of the main complications of stroke, affecting more than one-third of stroke patients, not only affecting the quality of life of patients after stroke, but also affecting patients' adherence to treatment plans, threatening their quality of life and increasing the risk of disability and death. There are many factors influencing it, and this review provides an overview of the pathogenesis, risk factors, and treatment of PSCI. It mainly includes the impact of age, education, economic income, blood pressure, blood sugar, etc., on PSCI and the current common treatments. To provide ideas for the early diagnosis and prevention of PSCI.

Keywords

Stroke, Cognitive Impairment after Stroke, Risk Factors, Treatment

1. 背景介绍

全球有超过 1220 万人经历过脑血管事件，且在过去 30 年中翻了一番[1]。从 2010 年到 2020 年，其患病率在全球范围内呈上升趋势。脑卒中是第二大死因，有 650 万人死于脑卒中。据统计，在脑卒中患者中，62%~87%患有缺血性脑卒中，其他人患有出血性脑卒中[2]。除死亡率高外，脑卒中还是导致残疾的主要原因[3]。即使是轻微的脑卒中也会影响日常生活、执行功能和认知功能，从而影响生活质量和工作效率[4]。然而，尽管有前瞻性数据，但结果是相互矛盾的，除了与年龄和血管危险因素相关的认知能力下降之外，我们对卒中事件的直接认知影响仍然知之甚少。卒中后，身体损伤往往会或多或少地改善；然而，由于未知的原因，认知障碍逐渐恶化。

脑卒中患者的认知变化是一个动态过程。脑卒中幸存者可能会在脑卒中后立即出现认知功能和神经精神症状急剧下降。3 个月后，神经功能缺损的恢复达到平台期。大部分的脑卒中患者在脑卒中后 3 个月内遇到了认知障碍。考虑到卒中后神经可塑性的时间限制窗口，早期识别高危人群和最佳干预很重要，因此需要在卒中事件发生后 3~6 个月内评估认知功能。

2. 发病机制

支撑 PSCI 的确切病理生理机制尚不清楚。目前认为，灌注不足、氧化应激、炎症和淀粉样蛋白级联反应单独或联合作用，影响神经血管单位，因此参与血管认知障碍的发病机制，具体如下：

在以前的研究中，抗氧化应激被证明可以减少脑卒中后损伤并缓解动物实验中神经变性的进展[5] [6]。这些结果表明，抗氧化应激能力降低可能是 PSCI 的致病机制。

炎症和免疫反应参与 AIS 的发病机制。在疾病发作之前，异常的免疫反应可诱发血管壁内和周围的炎症，从而促进血栓形成，改变血管反应性并促进动脉粥样硬化。在缺血性卒中发作后的数小时内，外周血中性粒细胞计数呈指数增加，淋巴细胞计数呈指数下降。中性粒细胞通过产生活性氧(reactive oxygen species, ROS)和合成细胞因子、趋化因子和细胞间粘附分子[7]对血脑屏障(blood-brain barrier, BBB)产生破坏性作用，并且它们脱颗粒产生髓过氧化物酶(Myeloperoxidase, MPO)和中性粒细胞弹性蛋白酶(neutrophil elastase, NE) [8]，这反过来又可能通过炎症和 BBB 破坏之间的恶性循环加剧对神经血管单位的损害。大量研究表明，神经血管单位的损伤与认知障碍的发作有关[9]。

最近的研究发现，PSCI 与阿尔茨海默病的核心病理变化密切相关，表明两者可能具有相同的诊断生物标志物[10] [11]。β 淀粉样蛋白(Amyloid β-protein, Aβ)是以阿尔茨海默病为特征的老年斑块的主要成分[12]。既往研究表明，血清 Aβ 可能是一个独立的脑血管危险因素，可提示痴呆期间血管损伤。Yu 等人[13]揭示了 Aβ 不仅存在于阿尔茨海默病中，而且与脑卒中进展相关。一些文献探讨了 Aβ1-42 水平对 PSCI 的潜在机制。根据实验研究，认为急性缺血会增加缺血性损伤后不久 Aβ 前体蛋白的表达和 Aβ 的产生，从而增强脑中 Aβ 的沉积和清除。有证据表明，Aβ 低聚物通过前馈神经变性环导致脑卒中后神经元丢失[14]。此外，缺血细胞释放的 Aβ 寡聚物能够触发小胶质细胞活化以诱导促炎状态，导致神经元死亡。Goulay 等人[15]推测，脑卒中后血管周围间隙完整性受损，炎症，缺氧和 BBB 破裂可导致脑实质和脑血管壁内 Aβ 加速沉积或脑淀粉样血管病恶化。Aβ 在实质中的沉积将成为导致突触功能障碍的起始事件，诱导认知能力下降和痴呆。Moulin 等人[16]认为血浆 Aβ1-40 参与血管方面，而 Aβ1-42 可能参与神经退行性过程。脑卒中动物模型和血管痴呆患者的脑组织中存在 Aβ1-42 沉积，可能与诱导一氧化氮、蛋白质和膜损伤有关。

3. 危险因素

3.1. 年龄、受教育程度、收入

年龄是中国 PSCI 最常见且不可改变的危险因素之一。PSCI 的发病率沿社会经济梯度分布，因此，受教育程度低和收入较少的患者更有可能患 PSCI。此外，一些研究表明，教育可以减少由脑损伤引起的认知障碍，这可能是由于突触连接增加导致大脑储备增加[17]。一项调查显示，体力劳动和服务业从业人员患 PSCI 风险是专业技术人员和管理人员的 2~3 倍，这可能是由于不同职业的个人经济状况或不同生活环境的社会经济状况造成的[18]。

3.2. 血压

研究表明，血压变异性升高或收缩压升高可能会增加 PSCI 的风险，但关于对血压的控制和 PSCI 预防的证据尚无定论[19]。较高的收缩压可能对海马体和齿状回产生负面影响[20] [21]，从而对大脑储备产生负面影响。

另有研究发现，非 PSCI 患者的高血压比例高于 PSCI 受试者，高血压可能是 PSCI 的保护因素。对于患者，尤其是老年人，脑灌注可能依赖于血压升高，高血压可能在一定程度上维持良好的脑灌注[22] [23]。另一方面，除了血压控制之外，降压药对认知具有保护作用。在动物研究中，血管紧张素受体阻滞剂可有效预防 Aβ42 毒性、反应性小胶质细胞增生和凋亡细胞死亡，从而减少梗死面积并预防 PSCI [24] [25]。研究指出，长期抗高血压治疗可将痴呆风险降低 55% [26]。

3.3. 血糖

糖尿病是 PSCI 的相关危险因素。小胶质细胞的激活在炎症反应中起重要作用。长期高血糖状态通过内皮素-1 系统导致促炎性小胶质细胞增殖[27]。相比之下，动物模型已经证明，抑制小胶质细胞活化可以改善脑卒中后的炎症反应，并显着预防这些动物的 PSCI [28]。除了炎症反应外，高血糖状态还会导致海马体中的 tau 蛋白过度磷酸化，从而加剧脑卒中后的认知缺陷[29]。

3.4. 肠道微生物

研究发现 PSCI 相关的肠道微生物群破坏肠道屏障，升高脂多糖和促使炎症的发生，从而加剧 BBB 破坏，小胶质细胞活化，海马细胞凋亡和丘脑 Aβ 沉积，导致脑卒中小鼠认知功能障碍[30]。

4. 治疗

4.1. 运动训练

相关研究表明，运动可有效改善认知功能障碍，这可能与运动可以增加脑神经营养因子有关。一项荟萃分析表明，对于 PSCI 患者，运动训练可以对整体认知产生积极影响，有氧运动和力量训练相结合会产生最大的认知益处。运动还可以改善慢性卒中的认知功能。它可适度改善治疗速度，可作为改善 PSCI 的干预策略[31]。因此，适当的长期有氧运动有助于预防脑梗死后轻度认知障碍。

4.2. 认知训练

认知训练对于治疗 PSCI 也是必不可少的，主要分为代偿训练策略和直接修复认知训练[32]。直接修复认知训练包括实践练习、记忆训练(如背诵首字母缩略词或歌曲)和计算机辅助认知训练。

4.3. 药物

目前，国家指南没有推荐的 PSCI 标准治疗药物。在临床上，有常用的药物在神经病理学和神经化学机制上与 AD 重叠来治疗这种疾病。最常见的药物是胆碱酯酶抑制剂(多奈哌齐、加兰他敏或卡巴拉汀)和非竞争性 N-甲基-D-天冬氨酸受体拮抗剂(美金刚) [33]。此外，2021 年中国“卒中后认知障碍管理专家共识 2021”将胆碱酯酶抑制剂多奈哌齐和卡巴拉汀列为 I 级推荐[34]。

4.4. 康复训练

康复训练在治疗本病中可以发挥不可或缺的作用。认知康复训练可以加速受损神经细胞的修复和皮质重建。根据对患者认知障碍的评估，康复治疗师对受损的认知区域进行有针对性的认知训练[35]。为了获得更好的结果，通常需要护理人员和家庭成员的共同参与。提示、补偿和环境适应等训练工具用于改善患者的主观参与和与外部刺激的互动。该领域的专家一致认为，应强调早期 PSCI 筛查和及时对卒中幸存者进行全面干预[36]。

4.5. 重复经颅磁刺激(Repetitive Transcranial Magnetic Stimulation, rTMS)

重复经颅磁刺激(rTMS)已成为 PSCI 的无创无痛治疗方法。经颅磁刺激(TMS)因其在调节皮质区域活动方面的功效而广泛用于治疗大脑神经调控[37] [38] [39] [40]。rTMS 的治疗机制是通过电磁感应在人类大脑皮层中产生亚阈值或超阈值电流，从而调节大脑皮层的兴奋性。一项荟萃研究发现，目前对证据的分析表明，rTMS 安全有效地促进 PSCI 患者的认知恢复，在治疗过程中发生一些短暂的不良反应，但都在患者的耐受范围内，对患者没有明显的负面影响[41]。高频 rTMS 和低频 rTMS 刺激延髓背外侧前额皮质层均能有效加速改善患者认知功能，增强患者日常生活活动能力。PSCI 患者高频 rTMS 和低频 rTMS 的治疗效果无显著差异。

5. 总结

本文参考了权威和经常被引用的文献和期刊，了解了 PSCI 研究的趋势，概括了该疾病的讨论热点。目前，对 PSCI 机制和治疗的研究还有限。同时，本文对近年来 PSCI 的相关危险因素进行总结，结果表示，高龄、低教育程度、低经济收入和糖尿病的脑卒中患者更容易患 PSCI；目前，血压水平对 PSCI 的患病仍有争议；肠道微生物可能造成 PSCI 的发生。综上所述，希望本次综述能够有效突出 PSCI 的研究轨迹，为 PSCI 的早期识别和早期干预提供思路及依据，为社会带来更多益处和更多价值。

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