·脑卒中研究进展·

症状性大脑中动脉粥样硬化性狭窄干预策略

陈平 刘嘉琳 林嘉星 关敏 乔宏宇 黄立安

【摘要】 颅内动脉粥样硬化性狭窄是亚洲人群缺血性卒中的常见原因,好发于大脑中动脉。症状性大脑中动脉粥样硬化性狭窄(sMCAS)致缺血性卒中的复发风险较高,需积极的干预治疗,但其干预策略的选择存有争议。本文对sMCAS的发病机制、临床表现、影像学评估和干预策略进行综述,旨在为个体化选择干预策略提供参考。

【关键词】 颅内动脉硬化; 大脑中动脉; 综述

Intervention strategies for symptomatic middle cerebral atherosclerotic stenosis

CHEN Ping[1], LIU Jia-lin[2], LIN Jia-xing[2], GUAN Min[2], QIAO Hong-yu[2], HUANG Li-an[2]

[1]*Department of Neurology, The First Hospital of Putian City, Putian 351100, Fujian, China*

[2]*Department of Neurology, The First Affiliated Hospital of Ji'nan University, Guangzhou 510630, Guangdong, China*

Corresponding author: HUANG Li-an (Email: huanglian1306@126.com)

【Abstract】 Intracranial atherosclerotic stenosis (ICAS) is the most common cause of ischemic stroke among Asian population, and its predilection site is middle cerebral artery. Symptomatic middle cerebral atherosclerotic stenosis (sMCAS) has a high risk of stroke recurrence and needs active interventions, but the choice of intervention strategy is controversial. This article reviews the pathogenesis, clinical manifestations, imaging evaluation and intervention strategies of sMCAS, aiming to provide reference for individual selection of intervention strategies.

【Key words】 Intracranial arteriosclerosis; Middle cerebral artery; Review

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颅内动脉粥样硬化性狭窄(ICAS)是亚洲人群缺血性卒中的常见病因,其中约50%发生于大脑中动脉(MCA)[1]。我国非心源性卒中患者颅内大动脉粥样硬化性狭窄和闭塞的患病率高达46.6%,尤以大脑中动脉重度狭窄或者闭塞的发生率最高[2]。《症状性颅内外动脉粥样硬化性大动脉狭窄管理规范——中国卒中学会科学声明》[3]将症状性大脑中动脉粥样硬化性狭窄(sMCAS)定义为:近3或6个月内发生的缺血性卒中和(或)短暂性脑缺血发作,伴大脑中动脉粥样硬化性狭窄(狭窄率50%~99%),梗死灶位于大脑中动脉供血区或者短暂性脑缺血发作症状与大脑中动脉供血区神经功能相匹配。若不积极干预,大部分sMCAS易复发,甚至有相当一部分可发生大脑中动脉闭塞(MCAO)[4]。有文献报道,sMCAS致缺血性卒中的年复发率明显高于无症状性大脑中动脉粥样硬化性狭窄[5]。治疗方法主要包括药物治疗、血管内介入治疗(机械取栓术、血管成形术和动脉溶栓)和颅内外动脉搭桥术,但干预策略的选择尚存争议,需依据sMCAS分期,结合临床表现和影像学评估,准确识别其发病机制,再个体化选择干预策略。

一、发病机制

慢性动脉粥样硬化斑块形成是sMCAS的主要病理生理学基础。大脑中动脉狭窄处不稳定型斑块破裂出血或溃疡表面血栓形成,可以导致远端栓

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作者单位:351100 福建省莆田市第一医院神经内科(陈平);510630 广州,暨南大学附属第一医院神经内科(刘嘉琳,林嘉星,关敏,乔宏宇,黄立安)

通讯作者:黄立安,Email:huanglian1306@126.com

塞、穿支动脉闭塞、主干狭窄或闭塞。sMCAS急性期发病机制为：斑块或血栓脱落引起动脉-动脉栓塞；斑块或血栓延伸阻塞豆纹动脉开口或脱落栓塞豆纹动脉致穿支动脉闭塞；血流动力学障碍致脑低灌注；以及慢性狭窄基础上形成的急性闭塞；抑或是上述机制共同作用所致[6-7]。慢性狭窄基础上急性闭塞的发生机制与不稳定型斑块及其破裂所致炎症反应密切相关，炎症促进局部血小板聚集和血栓形成，进而阻塞血管。sMCAS非急性期的发病机制主要为慢性狭窄所引起的脑低灌注和皮质微栓塞[8-9]。而脑低灌注和动脉-动脉栓塞为sMCAS性缺血性卒中的重要病因机制，且两种机制常并存[10]。明确sMCAS的发病机制是制定最佳干预策略的基础，单纯动脉-动脉栓塞和(或)穿支动脉闭塞，以单纯药物治疗(包括危险因素干预)为主；并发脑低灌注者，需考虑血管内介入治疗或颅内外动脉搭桥术；慢性狭窄基础上急性闭塞则应选择急诊血管再通治疗，包括静脉溶栓和急诊血管内介入治疗[6,11]。

二、临床评估

1. 临床表现　sMCAS急性期主要表现为急性缺血性卒中和短暂性脑缺血发作，临床症状与大脑中动脉闭塞段供血区神经功能相对应，例如，M1段急性闭塞者症状较为严重，除表现为对侧肢体运动和感觉障碍外，常伴有意识障碍，优势半球受累者还可合并失语；皮质支或豆纹动脉闭塞通常无意识障碍；M1段慢性狭窄基础上的急性闭塞，症状通常较心源性栓塞(CE)轻微，这是由于慢性狭窄后侧支循环已建立。除急性期后遗症，sMCAS非急性期患者通常无症状，可表现为认知功能减退(如注意力涣散、记忆力减退等)[8]，多见于左侧大脑半球受累患者，但不易识别。

2. 超声和影像学评估　(1)大脑中动脉主干及豆纹动脉成像：传统方法为TCD和经颅彩色多普勒超声(TCCD)等超声检测，以及CTA、MRA和DSA等血管成像检查。目前，DSA仍是评估颅内动脉粥样硬化性狭窄(包括大脑中动脉粥样硬化性狭窄)的“金标准”，其次为CTA，CTA评估颅内动脉狭窄，尤其是对狭窄程度＞50%者的诊断灵敏度和特异度均＞95%，且敏感性和阳性预测值优于三维时间飞跃(3D-TOF)MRA[7]。高分辨率MRI(HRMRI)作为一种新兴的影像学技术，不仅可用于检测血管狭窄率和动脉粥样硬化管壁特征(如斑块大小、形态和稳定性等)，还可用于检测斑块与穿支动脉开口的位置关系。本研究团队近期采用HRMRI与3D-DSA融合技术，直观地观察到粥样斑块与穿支动脉开口的位置关系[12]，有助于阐明sMCAS发病机制和指导干预策略。Chung等[13]采用TOF-MRA和HRMRI技术观察大脑中动脉粥样硬化性狭窄血管结构异常变化，发现sMCAS具有更高的管腔狭窄率、正向重构率、斑块厚度和斑块强化程度。Natori教授团队采用7T HRMRA评估急性缺血性卒中患者豆纹动脉再通情况，发现血管再通患者随访期间豆纹动脉长度明显大于无血管再通患者[14]。(2)组织窗评估：SPECT是评估组织窗(缺血半暗带)的“金标准”，由于该项检查方法费用昂贵、技术要求严格，使其在临床的普及率较低。目前主要采用CT和MRI两种模式，其中，CT评估模式主要包括CT灌注成像(CTP)和CTA源图像两种方式，CTP通过计算梗死核心体积与异常灌注区体积差值确定缺血半暗带；无法行CTP检查者，可以采用CTA源图像进行初步评估，基于CTA源图像的Alberta脑卒中计划早期CT评分(ASPECTS)＞5与预后良好密切相关[15]。MRI评估模式主要包括DWI-灌注成像(PWI)/动脉自旋标记(ASL)不匹配和DWI-FLAIR不匹配两种方式，DWI高信号代表梗死核心区，PWI残余功能达峰值时间＞6秒代表低灌注区，二者差值即为缺血半暗带，亦可以ASL替代PWI；DWI高信号而FILAIR信号改变不明显为DWI-FLAIR不匹配，代表发病时间＜4.5小时，提示存在缺血半暗带。(3)侧支代偿和远端灌注评估：包括TCD、MRA、CTA和DSA等结构影像学评估方法；以及CTP、PWI和ASL等功能影像学评估技术，通常需二者结合综合评估。大脑中动脉粥样硬化性狭窄主要依靠软脑膜侧支循环进行代偿，有研究显示，无症状性大脑中动脉粥样硬化性狭窄患者软脑膜侧支代偿优于症状性患者[16]，且侧支代偿较差者易并发认知功能障碍[8]。(4)血流动力学评估：大脑中动脉狭窄率＞75%时，其血流动力学危象和大面积脑梗死风险明显增加[17]，DSA和TCD是临床常用的脑血流动力学实时评估方法，TCD还可用于微栓子监测，其他评估方法还包括病灶远端/近端信号强度比值(SIR)、计算流体力学(CFD)、定量MRA(QMRA)和应用压力导丝直接测量等，但其临床价值尚待进一步验证。Fang等[18]的研究显示，大脑中动脉M1段狭窄后信号强度比值降低提示严重的血流动力学损伤，易发生严重的同侧白质病变。Leng等[19]基于CTA

图像构建计算流体力学模型，发现跨大脑中动脉狭窄病变的压力梯度与软脑膜侧支代偿相关。Mori等[20]采用HRMRA相关CFD法观察并分析豆纹动脉梗死患者血流动力学指标，发现豆纹动脉血流动力学改变可能是豆纹动脉梗死的危险因素。

三、干预策略

1.急性期（<24小时）干预 （1）静脉溶栓：于2018年发布的美国心脏协会（AHA）/美国卒中协会（ASA）指南[21]和2019年《中国脑血管病临床管理指南》[22]均推荐：对于明确发病时间窗<4.5小时的急性缺血性卒中患者（包括sMCAS急性闭塞致缺血性卒中），若无溶栓禁忌证，首选静脉溶栓治疗。而对于发病时间不明确或超过静脉溶栓时间窗，且经多模式影像学评估（如DWI-FLAIR不匹配）证实存在较大缺血半暗带者，静脉溶栓治疗仍安全、有效[23]。由于颅内大动脉（包括大脑中动脉M1段）闭塞后静脉溶栓的血管再通率较低，因此静脉溶栓是sMCAS致豆纹动脉或皮质动脉闭塞后血管再通的主要方法。（2）静脉抗血小板治疗：血小板糖蛋白ⅡB/Ⅲa受体阻断剂（GPI）如替罗非班，可以快速抑制狭窄局部血小板聚集，阻断急性新鲜血栓形成。《替罗非班在动脉粥样硬化性脑血管疾病中的临床应用专家共识》[24]指出，对于小动脉闭塞（SAO）型进展性缺血性卒中，单独应用替罗非班可能有效，因此sMCAS致豆纹动脉闭塞单独应用替罗非班是合理的，但有待大样本临床研究的验证；共识还指出，缺血性卒中急性期静脉溶栓序贯替罗非班可能增加动脉粥样硬化性狭窄或闭塞（包括sMCAS）的血管再通率并改善远期预后，但亦待进一步临床研究加以证实。（3）血管内机械取栓术：AHA/ASA指南[21]以及《中国脑血管病临床管理指南》[22]均推荐：对发病时间窗<6小时的急性缺血性卒中，大脑中动脉M1段闭塞首选静脉溶栓桥接机械取栓，M2和M3段闭塞可以考虑血管内机械取栓术。然而，2019年来自日本的SKIP研究[25]和2020年来自中国的DIRECT-MT研究[26]均显示，直接机械取栓后90天的预后良好率与静脉溶栓桥接机械取栓相当，提示对于大脑中动脉慢性狭窄基础上的急性闭塞患者，可直接行血管内机械取栓术。而发病时间6～24小时的sMCAS患者，可否采用血管内机械取栓术则依据DAWN研究和DEFFUSE2研究的标准[22]。（4）血管成形术（球囊扩张术和支架植入术）：大脑中动脉M1段急性闭塞机械取栓后，易发生血管再闭塞或影响远端灌注，若球囊扩张术仍不能有效改善前向血流，支架植入术可作为补救措施[27]。（5）动脉溶栓：《中国脑血管病临床管理指南》[22]指出，对于发病时间<6小时的大脑中动脉闭塞，若不适宜行静脉溶栓和血管内机械取栓术者，可以考虑动脉溶栓治疗。既往研究显示，rt-PA或尿激酶联合替罗非班动脉溶栓可提高急性大脑中动脉闭塞的血管再通率，促进早期神经功能恢复[28-29]；远端动脉如大脑中动脉M2段栓塞，经动脉予以替罗非班治疗亦可实现血管再通[30]。

2.非急性期（>24小时）干预 （1）药物治疗：自SAMMPRIS研究[31]和VISSIT研究[32]结果公布以来，对症状性颅内动脉粥样硬化性狭窄（包括sMCAS）的二级预防，采取标准双联抗血小板治疗（阿司匹林+氯吡格雷），以及严格的危险因素（如高血压、糖尿病、高脂血症等）控制业已成为共识，并根据抗栓效果（如血小板抑制率）和药物耐受情况调整双联抗血小板药物组合，且双抗治疗时间≤3个月[33]。（2）血管成形术（球囊扩张术和支架植入术）：重度慢性sMCAS患者，药物治疗难以在短期内改善其远端低灌注，血管成形术可通过增加脑灌注而改善神经功能[34]，但需关注患者围手术期并发症发生风险。近年研究显示，除神经介入科医师积累经验外，严格筛选病例（如狭窄率>70%合并远端低灌注、强化药物治疗无效或侧支代偿不良、术前mRS评分<3、非大面积脑梗死等），以及合理选择手术时机（发病后2周）均可显著降低血管成形术围手术期并发症发生率[35-36]。由于sMCAS病变通常位于豆纹动脉开口处或其邻近区域，应用影像学融合技术（HRMRI与3D-DSA融合）于术前评估并指导血管成形术亦可显著降低穿支动脉闭塞的风险，特别是斑块位于大脑中动脉后上壁或距离豆纹动脉开口处较近者，需谨慎手术。此外，手术方式（如单纯球囊扩张术）和手术材料（如药物球囊）也有助于减少围手术期并发症[37-38]。（3）颅内外动脉搭桥术：1985年的国际颅内外动脉搭桥试验（EC-IC）提出，颅内外动脉搭桥术并不能降低sMCAS患者缺血性卒中之复发率[39]。然而晚近研究显示，对于重度狭窄所致远端血流动力学损害的sMCAS患者，颞浅动脉-大脑中动脉（STA-MCA）搭桥术可有效改善临床症状并降低缺血性卒中复发率[40-41]，因此，对于药物治疗无效且无法行血管内介入治疗的重度sMCAS患者，可尝试行颞浅动脉-大脑中动脉搭桥术。

四、展望

目前，积极的药物治疗和严格的危险因素控制是sMCAS的主要干预策略，但对于药物治疗无效的sMCAS患者，可考虑血管内介入治疗和颅内外动脉搭桥术。sMCAS发病机制十分复杂，准确识别sMCAS发病机制是制定最佳干预策略的关键，需精准的影像学评估以指导治疗，尤其应关注斑块与豆纹动脉开口的位置关系、侧支代偿和远端灌注情况，未来研究应侧重于sMCAS患者从血管内介入治疗或颅内外动脉搭桥术中受益。

利益冲突　无

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