

· 心肌梗死专题研究 ·

ST 段抬高型心肌梗死患者超窗口期再灌注治疗的思考

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丁绍祥

【摘要】 ST段抬高型心肌梗死（STEMI）是冠心病的严重类型，经皮冠状动脉介入治疗（PCI）是其最佳治疗手段，可以恢复梗死区血液灌注，进而避免更多心肌坏死，但部分患者因各种原因而错过最佳治疗时机。因此，规范STEMI患者超窗口期治疗策略对改善患者预后具有重要意义。近年来，针对有明确胸痛症状或血流动力学不稳定的STEMI患者超窗口期行再灌注治疗无争议，但对血流动力学相对稳定的STEMI患者超窗口期行再灌注治疗存在较大争议。本文主要综述了STEMI的病理特征及STEMI患者超窗口期再灌注治疗情况，以期临床医生选择治疗方案提供一定参考。

【关键词】 心肌梗死；ST段抬高型心肌梗死；再灌注治疗；经皮冠状动脉介入治疗

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Reflection on the Reperfusion Therapy in Patients with ST Segment Elevation Myocardial Infarction Beyond the Optimal Time-Window

DING Shaoxiang

Geriatric Department, the Second People's Hospital of Huishan District, Wuxi 214000, China

【Abstract】 ST segment elevation myocardial infarction (STEMI) is a serious type of coronary heart disease, and percutaneous coronary intervention (PCI) is its best treatment method, which can restore blood perfusion in the infarcted area and avoid more myocardial necrosis. However, some patients miss the best treatment opportunity due to various reasons. Therefore, standardizing the treatment strategy for STEMI patients beyond the optimal time-window is of great significance in improving their prognosis. In recent years, there has been no controversy regarding reperfusion therapy for STEMI patients with clear chest pain symptoms or hemodynamic instability beyond the optimal time-window, but there is significant controversy regarding reperfusion therapy for STEMI patients with relatively stable hemodynamics beyond the optimal time-window. This article mainly reviews the pathological characteristics of STEMI and reperfusion therapy in STEMI patients beyond the optimal time-window, in order to provide some reference for clinical doctors to choose treatment plans.

【Key words】 Myocardial infarction; ST segment elevation myocardial infarction; Reperfusion therapy; Percutaneous coronary intervention

ST段抬高型心肌梗死（ST segment elevation myocardial infarction, STEMI）是冠心病的严重类型，多在冠状动脉不稳定斑块破裂、糜烂、侵蚀及内皮损伤基础上继发血栓形成，致使相应血管急性、持续、完全闭塞，血供急剧减少或中断，进而使心肌细胞缺血、损伤及坏死。尽管近年来冠心病的死亡率有所下降，但其仍是导致居民死亡的主要病因之一^[1]。《中国心血管健康与疾病报告2020》显示，我国冠心病患者已达1 300多万，且STEMI发病率呈快速增长趋势^[2]。目前，经皮冠状动脉介入治疗（percutaneous coronary intervention, PCI）是STEMI患者的最佳治疗手段，其可以恢复梗死区血液灌注，进而避免更多心肌坏死^[3]，但部分患者因各种原因而错过最佳治疗时机^[4]。近年来，针对有明确胸痛症状或血流动力学不稳定的STEMI患者超窗口期行再灌注治

疗无争议，但对血流动力学相对稳定的STEMI患者超窗口期行再灌注治疗存在较大争议^[5]。本文主要综述了STEMI的病理特征及STEMI患者超窗口期再灌注治疗情况，以期临床医生选择治疗方案提供一定参考。

1 冠状动脉血流的病理生理特点

众所周知，冠状动脉血流量与血管直径相关，每平方毫米心肌约有2 500根毛细血管，且几乎每个心肌细胞伴随1根毛细血管，进而有利于氧气摄取及物质交换。冠状动脉之间有丰富的吻合支或侧支，安静时5.0%~33.3%的微血管呈开放状态，一旦需要增加血流，闭合的毛细血管则会迅速开放。冠状动脉突然闭塞，侧支循环无法很快建立，常导致心肌梗死；但若冠状动脉缓慢闭塞，则侧支循环可逐渐代偿扩张并建立新的侧支。

冠状动脉受迷走神经和交感神经的支配，其中迷走神经对冠状动脉血流量的影响较小，对微血管具有保护作用；交感神经兴奋的总效应是使冠状动脉血流量增多^[6]。肾上腺素和去甲肾上腺素均可以通过促进心肌代谢活动和增加心肌耗氧量而

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作者单位：214000江苏省无锡市惠山区第二人民医院老年科

使冠状动脉血流量增加;抗利尿激素可使冠状动脉收缩,血流量减少;前列环素具有扩张冠状动脉的作用,而血栓素A₁的作用正好与之相反。腺苷是心肌代谢释放的最重要且最强烈的舒血管物质^[7];心肌缺血时,冠状动脉内皮细胞合成并释放前列环素增多,进而扩张冠状动脉、调节血流量^[8]。

2 STEMI的病理特征

2.1 炎症反应 STEMI多由梗死区血管闭塞所致^[9]。心肌梗死后炎症反应被启动,该反应是具有血管系统的活体组织对损伤因子产生的防御反应,其中血管反应是炎症反应的中心环节^[10]。炎症反应是人体的自动防御反应,但其对人体自身组织的攻击是有害的^[11]。缺血或缺氧等原因引起组织坏死,而坏死组织又是潜在的致炎因子;变质是由致炎因子直接作用或由炎症反应过程中发生的局部血液循环障碍和免疫机制介导的,其严重程度取决于致炎因子的性质、强度和机体的反应性。研究表明,心肌坏死可触发炎症反应,促使组织和细胞变性、坏死后释放水解酶,进而使受损组织和细胞溶解、液化,并进一步引起周围组织、细胞变质^[12]。

研究表明,早期心肌血流再灌注与较少的心肌坏死/功能障碍及较轻的炎症反应相关^[13]。血管闭塞后20~30 min即有少数心肌坏死;血管闭塞1~12 h绝大部分缺血心肌发生凝固性坏死,心肌间质充血、水肿并伴有大量炎性细胞浸润;血管闭塞4 d后坏死心肌分界明显,心肌溶解并伴有肉芽组织形成^[14]。1~2周后坏死心肌组织开始被吸收,并逐渐纤维化;6~8周后坏死心肌组织形成瘢痕而愈合。

2.2 心脏血流动力学障碍及心肌电生理改变 心肌梗死会导致血流动力学障碍,其发生机制为:梗死区坏死心肌收缩力减弱,心室收缩力不协调,左心室压力曲线最大上升速率减慢,顺应性降低,舒张末期压力升高,舒张和收缩末期容量增大,射血分数降低,心排血量下降,心率增快或伴有血压下降,进而出现心力衰竭或心源性休克;梗死区心室壁薄弱且不能承受心室内压,早期可能发生心脏破裂,后期可发生心室膨胀瘤^[15-16]。研究表明,心肌梗死区细胞电生理改变与心肌缺血、缺氧相关,细胞膜功能不全使膜内外极性程度降低,心肌细胞兴奋性增加,传导性下降;同时,缺血坏死组织使交感神经兴奋,血液中儿茶酚胺浓度增高,进而诱发心律失常^[17]。

3 STEMI患者超窗口期再灌注治疗

3.1 超窗口期心肌顿抑与心肌坏死 STEMI患者罪犯血管供血区心肌血供急剧减少,且越邻近主干供血中心区,心肌缺血程度越严重,若无法及时进行再灌注治疗,则心肌坏死风险极高^[18]。但由于个体差异,罪犯血管急性闭塞的进程并不相同,部分患者心肌梗死前已存在严重血管狭窄,进而可能存在一些侧支代偿,故其坏死心肌相对较少,顿抑心肌相对较多。一项纳入7 775例急性冠脉综合征患者的研究发现,合并心血管危险因素数量少的STEMI患者较合并心血管危险因素数量多的患者更易发生心肺骤停和/或心源性休克,且住院死亡率也更高^[19]。在少量血流供应情况下,顿抑心肌仅能维持细胞最基本的新陈代谢;随着心肌缺血时间的延长,心肌细胞代偿储备消耗增多,不可逆的坏死心肌越多,患者预后则

越差^[20]。因此,针对这部分患者即使错过窗口期,也应考虑行再灌注治疗。

3.2 超窗口期宜行再灌注治疗 目前,STEMI患者行PCI的窗口期仍存在争议^[21]。一般认为,STEMI发病12 h内为窗口期,应首选PCI^[22]。对于合并房室传导阻滞的STEMI患者,即使处于超窗口期也应推荐PCI为优先治疗策略,因为恢复梗死相关动脉再灌注有助于促进房室传导阻滞的恢复^[23]。因此,对于部分STEMI患者,超窗口期再灌注治疗并非总是太晚^[24]。

部分STEMI患者罪犯血管供血区存在缺血预适应且建立了侧支循环,大量顿抑心肌导致绝对缺血区相对减少。研究表明,STEMI患者发病12~48 h内行冠状动脉血运重建与短期、长期预后良好相关^[25]。一项纳入5 427例STEMI患者的研究发现,6.2%的患者就诊时处于超窗口期,其中以老年人、女性、有高血压和糖尿病病史者居多,且窗口期(发病12 h内)行PCI的患者与超窗口期(发病12~24 h内)行PCI的患者的院内死亡风险和1年死亡风险相似^[26]。

研究表明,部分STEMI患者会出现罪犯血管自溶再通,其相应心肌病理改变较为复杂,除受患者基础疾病和自身条件影响外,急性心肌梗死后自溶时间、再通血管血流量、有无心力衰竭及心律失常等均直接影响梗死区濒临坏死心肌的预后^[27]。研究发现,针对发病后12~72 h内发生罪犯血管自溶再通的STEMI患者,延迟行PCI的成功率较高,且不增加住院期间和长期主要不良心血管事件发生率和死亡率^[28]。KRAWCZYK等^[29]研究表明,PCI前罪犯血管自溶再通可以改善STEMI患者的临床预后。因此,针对出现罪犯血管自溶再通的STEMI患者,应积极考虑行超窗口期再灌注治疗,至少理论上会有更多的心肌获益,且可以降低心血管事件发生风险^[30]。

3.3 超窗口期不宜行再灌注治疗 部分STEMI患者发病时未建立侧支循环,故血流突然中断会导致相应供血区单位时间内坏死心肌数量增多,炎症反应较重;此时,若机体不能及时限制罪犯血管的持续损伤效应,则其心血管事件发生风险增高^[31]。与新鲜血栓相比,超窗口期血栓形成程度较高,不易被纤维蛋白原溶解,其破裂后栓塞风险较高,且机体自净效果差;此外,超窗口期血栓稳定性较差,易破碎,炎症反应及心血管事件发生风险较高;再者,STEMI患者急性期易导致躯体及心理精神症状,大量心肌坏死可诱发心力衰竭,故超窗口期行PCI易导致炎症反应持续增强,心肌再灌注损伤加重,易导致血栓形成、远端栓塞、无复流现象,甚至诱发梗死区心脏破裂^[32]。CHO等^[33]研究表明,发病12 h内与发病12~48 h内行PCI的STEMI患者死亡率存在较大差异。且临床研究表明,与延迟/晚期行PCI的STEMI患者相比,接受药物治疗的STEMI患者长期生存率更高^[34-35]。因此,针对未建立侧支循环的STEMI患者,超窗口期若无心肌再缺血证据,不宜行PCI。

4 小结

STEMI患者超窗口期梗死区域心肌组织结构及电生理均不稳定,坏死组织被破坏和吸收,梗死区心肌发生纤维化及重塑,因此,过早干预可能导致心功能恶化,进而增加严重心功能不全、恶性心律失常甚至心脏破裂的发生风险^[36]。因

此,针对血流动力学相对稳定的STEMI患者超窗口期行再灌注治疗存在较大争议。而针对超窗口期且无症状的STEMI患者,应尽早行冠状动脉造影,如发现缺血区存在血流灌注,无论是自溶再通、侧支代偿、痉挛或其他原因所致,均应及时进行干预,以挽救更多的濒死心肌;针对急性完全闭塞伴侧支循环丰富的STEMI患者,窗口期不应 <12 h,其目的是挽救梗死区周围更多的顿抑心肌;而针对罪犯血管早期供血较好、闭塞区周围无有效侧支循环、无明显临床症状的超窗口期STEMI患者,不建议早期盲目行PCI,应在患者度过急性期后再考虑行PCI,除非有新的缺血证据。此外,由于个体痛阈存在差异,需要考虑如何鉴别痛阈较高的STEMI患者是否存在持续心肌坏死,且该类患者的不良预后常与其基线特征和延迟治疗有关^[37-38]。总之,需要结合超窗口期STEMI患者的临床特征评估其疾病风险,且基础疾病越多,患者PCI获益的可能性越大,但同时风险也越高^[39]。

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