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急性心肌梗死(AMI)与抑郁症相关性研究进展

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摘　要

近年来关于AMI与心理卫生障碍之间的关系得到越来越多的关注。在不同心理卫生障碍中抑郁症对患者的影响最大，不仅对患者的心理和躯体产生不利影响，还可增加患者的自杀率，直接威胁患者的生命健康。本综述就AMI与抑郁症之间近十年的研究进展进行综述。本综述主要从AMI合并抑郁症的流行病学、危险因素、相互作用机制、影响、评估方式、治疗这几个方面阐述，希望能在AMI患者的后续康复和综合管理中提供一点帮助。

关键词

急性心肌梗死，抑郁症，双心医学

Research Progress on the Correlation between Acute Myocardial Infarction (AMI) and Depression

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Abstract

The relationship between AMI and mental health disorders has received increasing attention in recent years. Among different mental health disorders, depression has the greatest impact on pa-

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tients. It not only has adverse effects on patients' mental and physical health, but also can increase the suicide rate of patients and directly threaten the life and health of patients. This review reviews the research progress of AMI and depression in recent ten years. This review mainly expounds the epidemiology, risk factors, interaction mechanism, influence, evaluation method and treatment of AMI combined with depression, hoping to provide some help in the follow-up rehabilitation and comprehensive management of AMI patients.

Keywords

Acute Myocardial Infarction, Depression, Psycho-Cardiology

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1. AMI 合并抑郁症的流行病学

1.1. AMI 患者抑郁症的患病率

国内外关于 AMI 患者抑郁症患病率的研究不是特别多，且差别较大。既往研究抑郁的患病率最低为13%。近年来，根据国外[1] [2]的研究发现 AMI 后抑郁的患病率在 20%~40%，与之前相比患病率明显升高。尽管国内外关于抑郁症在 AMI 患者的患病率不尽相同，但总体可以看出 AMI 患者抑郁症患病率均较高，且呈上升趋势。国外 Petr Kala 等人[3]研究表明接受经皮冠状动脉介入治疗(PCI)的 ST 段抬高型心肌梗死(STEMI)患者抑郁和焦虑症状的总体患病率相对较低。而国内的研究有不同的结论，杨光铭等人[4]的研究发现抑郁症在 PCI 治疗的患者中同样很常见，中年心梗 PCI 术后患者抑郁的发病率在 37.57%以上。关于接受 PCI 治疗后是否能降低抑郁的患病率仍待考究，需要更多的临床统计进行验证。

1.2. AMI 合并抑郁症的危险因素

总结 AMI 合并抑郁症的危险因素，从而有效地规避危险因素，降低患抑郁症的风险是很有必要的。一方面可以促进患者的身体健康，提高生活质量；另一方面降低住院率，减短住院时间，减少患者经济花费的同时可以节约医疗资源。

多篇文章[5] [6] [7] [8]表明女性性别、年龄、低收入、高等教育水平、单身、体重指数(BMI)减低、体力活动减少、长时间工作、吸烟、心功能受损、D 型人格(社会接触不良或受歧视)、配偶或父母患有慢性重大疾病是 AMI 合并抑郁的危险因素。而 Teymoor Yary 等[9]人的研究认为年龄、收入、冠心病家族史、受教育程度、性别、就业和吸烟与严重抑郁症无关。

2. 抑郁对 AMI 预后的影响

少数文章[10] [11]研究表明抑郁与心血管疾病(CVD)发病率无关。大多数文章[12] [13] [14] [15] [16]研究认为抑郁症会增加 CVD 的发生风险，与死亡风险增加有关。合并抑郁症的患者 CVD 的发生风险或死亡风险都是无抑郁症患者的两倍。患有非典型重度抑郁症或双重抑郁症的成年人可能是抑郁症人群中新发 CVD 风险特别高的亚组。Bilge Burçak Annagür 等[17]人发现抑郁症可能会增加早期心肌梗死的风险。在儿童和人类免疫缺陷病毒(HIV)感染的成年人中的研究结果与之一致。同时，抑郁症会影响心梗患者的生活质量，延长患者的住院时间。年轻心梗患者的抑郁终生史率更高。与无抑郁症的 AMI 幸存者相比，

患有抑郁症的 AMI 幸存者更超重，体力活动更少，有较高的膳食脂肪摄入量，吸烟的可能性更高(可由社会人口统计学因素解释)。此外，抑郁会导致患者产生习得性无助感[18] [19] [20] [21]。从患者血液成分的变化中发现了抑郁症对 AMI 患者的影响。据研究表明，抑郁症对促进粥样硬化方面起重要作用[22]。AMI 合并抑郁的患者，抑郁评分越高，血脂异常越明显，与甘油三酯(TG)、总胆固醇(TC)、低密度脂蛋白(LDL-C)呈现正相关，与高密度脂蛋白(HDL-C)呈现负相关[23] [24]。患有 AMI 的患者中存在抑郁症状会导致体内肿瘤坏死因子-α (TNF-α)、内皮素-1 (ET-1)、血清高敏 C 反应蛋白(hs-CRP)、血清髓过氧化物酶(MPO)、脂质素 A4 (LXA4)、脑钠肽前体(NT-proBNP)水平以及 M/L 比升高，皮质醇水平、血管内皮生长因子-A (VEGF-A)水平降低[25]-[30]。提示抑郁症对高血压、高血脂、心衰、体内炎症水平有促进作用，对免疫系统或有抑制作用。然而，Kim G Smolderen 等人[31]的研究认为抑郁症与 hs-CRP、NT-proBNP、白细胞或血小板计数这些生物标志物无关。

3. AMI 与抑郁症相互作用的机制

根据以往的研究结果，抑郁症影响冠心病的机制主要从炎症反应、免疫反应与内皮功能、脑 5-羟色胺(5-HT)的功能、血小板的激活以及高凝状态、交感神经过度激活、促凋亡途径及基因学等方面解释。

3.1. 炎症反应

炎症反应可能是心梗后抑郁发生的机制[32]。在动物研究中发现，抑郁大鼠循环血中及海马区炎症因子显著增高，出现中枢神经炎症[33]。且发现前炎症细胞因子阻滞剂-PTX (已酮可可碱)可抑制这种神经炎症反应，抑郁症状也得到改善[34]。Alina Wilkowska 等人[35]发现 IL-17a、IL-1β 的增加可能起重要的作用。S100A9 属于 DAMPs 家族，通过刺激白细胞募集和细胞因子分泌在介导炎症反应中发挥重要作用[36]。孙一泽等人[37]发现 S100A9 是心肌和大脑海马区共享的唯一差异表达蛋白，在 AMI 模型中显著增高，接受双心治疗后，海马及心肌梗死周围边缘区表达明显下调，其介导的巨噬细胞/小胶质细胞炎症可能是心理－心脏病的重要致病过程。陶禄远等人[38]发现心脏中巨噬细胞迁移抑制因子(MIF)表达下降，可能是抑郁与心梗后不良预后联系起来的合理机制。

3.2. 免疫反应与内皮功能

Rossella Di Stefano 等人[39]发现重度抑郁发作的急性冠脉综合征(ACS)患者的循环 CD34CD133KDR 细胞数量减少。考虑可能与内皮祖细胞(EPCs)在 ACS 患者中的修复作用有关。Andreas Baranyi 等人[40]研究结果表明，抑郁症引发的一氧化氮(NO)减少可能是心血管风险增加的机制，不对称二甲基精氨酸(ADMA)和对称二甲基精氨酸(SDMA)可能是生物标志物。

3.3. 脑 5-HT 的功能

脑 5-HT 的减低可能是心梗后抑郁的机制[41]。有研究发现增强 5-HT 递质系统功能，可改善大鼠抑郁行为[42]。卢晓芳等人[43]发现外周血 5-HT 水平与海马体 5-HT 水平呈反比关系，肠道中异常的 5-HT 代谢在心肌梗死后抑郁的表现中起着至关重要的作用。葛英斌等人[44]发现银杏内酯可以通过 STAT3 途径调节中枢神经系统中的 5-HT 和白介素-1β (IL-1β)来逆转心肌梗死引起的抑郁样行为。

3.4. 血小板的激活以及高凝状态

血小板的激活以及高凝状态可能是心梗后抑郁的机制。血小板裂解物中 5-羟色胺 2A 受体(5-HT2AR)的浓度和血清、血小板中的 5-HT 转运蛋白，内质网(ER)分子伴侣 sigma-1 受体，脑源性神经营养因子(Brain-derived neurotrophic factor, BDNF)可能在心梗伴抑郁的病理生理学中发挥作用[45] [46] [47]。

3.5. 交感神经过度激活

Aysha Almas 等人[48]发现高儿茶酚胺氧位甲基转移酶(COMT Val158Met)活性基因型增加了抑郁症患者患 CVD 的风险。交感神经过度激活是抑郁与心梗后不良预后联系起来的合理机制[49]。

3.6. 促凋亡途径及基因学 MMP/TIMP

基质金属蛋白酶(MMPs)参与多种情绪和心血管疾病的发病机制，MMP 失衡在血管壁通过血小板释放或诱导炎症转化为病理性血栓形成过程中发挥着重要作用。同时多项研究已证明 MMP 与细胞凋亡之间的关联，其在通过细胞凋亡促进心脏保护方面发挥着至关重要的作用。心肌梗死合并抑郁可能会破坏中枢和外周系统的基质金属蛋白酶(MMP)平衡从而参与心梗后抑郁的机制[50]。戴振国等人[51]发现 GNB3，CNR1，MTHFR 和 NCAM1 基因，可能影响心肌梗死与抑郁症之间的相互作用。Julia Brandt 等人[52]研究发现 FKBP5 基因型在冠心病患者中具有相关性。FKBP5 C 等位基因的数量较多与抑郁症状增加有关，其可能赋予 CHD 和抑郁症共同的遗传风险。Simon Jönsson 等人[53]研究认为血液单核细胞中基质金属蛋白酶-9 (MMP-9)、金属蛋白酶组织抑制因子(TIMPs)的过表达和抑郁症状的升高是心肌梗死后两种不相关的现象。

4. AMI 合并抑郁的评估

Elizabeth C Pino 等人[54]研究认为心血管事件的潜在未被充分诊断的心理健康问题实际上是整个慢性疾病。有研究表明负面情绪会增加冠心病患者预后不良的风险，对冠心病患者进行抑郁的筛查具有预后益处[55]。多篇文章表明心梗患者的抑郁症治疗可降低死亡或住院风险，持续监测和适当治疗可以改善患者的预后和生活质量[56] [57] [58]。因此，抑郁症的及时诊断具有重要意义。抑郁症的诊断主要依靠评分量表，BDI 评分、BDI-II、PHQ-9、GAD-7、蒙哥马利奥斯伯格抑郁评定量表(MADRS)、重度抑郁症量表(MDI)、HADS 评分均可识别患有抑郁症的 CAD 患者[59] [60]。研究发现 BDI 评分可能部分反映了急性躯体疾病或其治疗的症状，而不是抑郁症。后修订的 BDI-II 旨在减少躯体症状对总分的影响，在 MI 后患者中，可能优于 BDI [61] [62]。陈毅孝等人[63]表明重度抑郁症量表(MDI)可高度可靠的用于评估中国心脏病患者的抑郁症。Franziska Geiser 等人[64]研究发现凝血和纤维蛋白溶解标志物与医院焦虑抑郁量表(HADS)中焦虑和抑郁评分没有显着关联。后续研究可进一步发现 HADS 评分是否真正适用于患抑郁症的 CAD 患者，以及凝血、纤维蛋白溶解标志物与抑郁之间的相关性。

5. AMI 合并抑郁的治疗

目前关于AMI合并抑郁症的治疗，根据相关文献的研究主要分为非药物治疗、药物治疗及联合治疗。健康的生活状态、运动和心脏康复被证实对 AMI 合并抑郁的患者是有益的。一些药物主要根据抑郁症影响冠心病的可能机制进行治疗，主要通过抑制 5-HT 再摄取、抑制炎症反应、调节免疫等方面进行干预。某些中药作用机制暂不明确，但效果尚可。有些治疗对于 AMI 的受益程度及治疗的耐受程度并没有明确报道，相关临床研究仍然是今后可研究的方向。

5.1. 非药物治疗

1) 健康饮食，包括纤维摄入量和水果、蔬菜的摄入量，可减轻抑郁症状，提高乐观情绪。其中，多篇动物实验研究表明，补充益生菌以及 n-3 多不饱和脂肪酸(n-3 PUFA)可减轻心梗后抑郁症状。其机制可能与减轻边缘系统的凋亡，以及激活中枢系统某些酶的活性有关，需要进一步研究[65] [66] [67]。

2) 定期体力活动与心梗后抑郁症状呈负相关，尤其是女性[68] [69]。

3) 有效沟通，提供充分的疾病信息、情感支持、社会支持，增强对患者的感知控制，可有效改善患者的焦虑抑郁情绪，改善预后[70] [71] [72] [73]。

4) 近五年的研究[74]发现认知行为疗法(CBT)是初级保健中抑郁症患者的一线心理治疗，其对急性心肌梗死后的抑郁症有效。然而有研究发现基于互联网的认知行为疗法在减少自我报告的抑郁或焦虑症状方面并不优越，考虑治疗依从性低，这可能影响了治疗参与度和结局。但对于 AMI 后的阻塞性睡眠呼吸暂停(OSA)患者，CBT 可能效果较差[75] [76]。

5) 心脏康复，多篇文章表明心脏康复可使心梗合并抑郁的患者受益。心脏康复可能有助于改善身体活动(PA)和功能能力，降低抑郁程度及死亡风险。然而有研究表明睡眠障碍会降低心脏康复带来的益处[77] [78] [79]。

6) Mohammad Behnammoghadam 等人[80]发现眼动脱敏再处理疗法(EMDR)是一种可用于治疗和减少心肌梗死患者抑郁的有用、高效且非侵入性的方法。

7) Jaakko Erkkilä 等人发现[81]综合即兴音乐疗法(IIMT)对短期和中期抑郁症状以及焦虑和功能的有益作用。Eleanor J Cole 等人[82]与唐乃龙等人[83]研究发现斯坦福加速智能神经调节疗法(SAINT)具有快速、高效、安全地降低自杀观念和缓解抑郁症状的潜在优势，同时能改善受损脑网络的功能连接度。用于治疗难治性抑郁症，是一种加速、高剂量、iTBS 方案，采用 fcMRI 引导靶向，耐受性良好且安全。但综合即兴音乐疗法(IIMT)与斯坦福加速智能神经调节疗法(SAINT)两项研究未在冠心病合并抑郁症患者中进行试验，对共病患者是否有效、是否安全值得进一步研究。

5.2. 药物治疗

5.2.1. 西药治疗的研究

大多研究表明抗抑郁药尤其是选择性 5-HT 再摄取抑制剂(SSRI)的使用与主要不良心血管事件(MACE)率的显着降低有关[84]，部分研究认为抗抑郁药与 CVD 事件风险降低无关，Marij Zuidersma 等人的研究同时表明接受抑郁症治疗增加了生存率[85]。还有文章表明抗抑郁药的使用会增加心梗的风险[86] [87]。关于冠心病合并抑郁症的患者使用抗抑郁药治疗的利弊仍然值得考究。

关于艾司西酞普兰的研究表明其具有心脏保护作用，可降低心脏事件的风险[88]。动物实验研究表明，西酞普兰治疗导致小鼠心肌梗死后死亡率增加，其机制可能是 MMP-13 上调导致心室破裂，用 PD166793 抑制 MMP 可以部分逆转西酞普兰的作用[89]。帕罗西汀可有效改善 AMI 伴抑郁(AMID)患者的心脏功能[90]。梁金军等人[91]的动物实验表明氟西汀可降低心肌梗死后大鼠室性心律失常的发生率，可能部分与氟西汀对 Kv4.2 的上调有关。最好避免使用氟西汀和氟伏沙明，因为这两种 SSRI 类药物都可能降低氯吡格雷的疗效[92]。同样 Katsiaryna Bykov 等人[93]的研究也表明使用抑制 cyp2c19 的 SSRI 治疗可能与氯吡格雷的有效性轻微下降有关。AMI 后开始β阻滞剂治疗及他汀类药物治疗与抑郁症状的增加无关[94] [95]。

曲美他嗪可以对大鼠 5-HT、5-HT(2A)R 和血清素转运蛋白(SERT)水平的调节发挥作用，在精神心脏病方面可能有治疗作用，需要进一步研究[96] [97]。

5.2.2. 中药治疗的研究

人参皂苷对 5-HT 系统的调节中起着重要作用，其对急性心血管事件和抑郁症共病的治疗潜力似乎很有希望[98] [99]。酸枣仁汤联合常规药物治疗可改善 AMI 合并抑郁患者心理状态、提高生活质量、改善心功能、减少心律失常和临床心血管事件的发生[100]。银杏内酯 B (GB)通过 STAT3 途径降低促炎细胞因子的水平，在有效治疗心肌梗死后抑郁症方面具有很大益处[44]。益心宁神片可以通过抵抗炎症和增加单胺神经递质的可用性来缓解抑郁症，可用作治疗心肌梗死和抑郁合并症的潜在药物[101]。有研究表明

开心散、舒肝解郁胶囊对心肌梗死伴抑郁症具有疗效[102] [103]。

5.2.3. 新治疗策略

最近研究发现，人脐带间充质干细胞(HUC-MSCs)具有由心肌梗死诱导的心脏保护和潜在的抗抑郁作用，可能与调节 Jmjd3 和小胶质细胞极化改善的炎症有关[104]。孙楠等人[105]发现外周 ghrelin 给药可抑制抑郁样行为和神经炎症，靶向(神经)炎症可能代表心力衰竭和抑郁患者的新治疗策略[106]。刘轩等人[107]发现 Alda-1 通过增加大鼠海马体中的 VEGF 表达来改善心肌梗死后大鼠的抑郁样行为。

5.3. 联合治疗

对于 AMI 并发抑郁症的患者，在积极有效药物治疗的同时配合心理干预，采用针对性的心理支持疗法，唤起患者的积极情绪，使之配合治疗，尤其对于女性患者，有助于消除患者的焦虑抑郁症状，使患者平安度过危险期，提高生活质量和临床治愈率，在明显改善预后的同时，真正体现治疗个性化[108]。而另一篇文章认为双心医疗的应用可促进 AMI 合并焦虑抑郁患者心功能、生活质量以及日常生活活动能力的改善[109]。

6. 结论

随着生活节奏的加快、生活水平的提高，一方面大众心理健康问题日益突出，另一方面人们对心理健康问题越来越重视，双心医学的发展正是符合当下生物－心理－社会的现代化医疗模式，结合我国传统中医学中的整体观念，身心健康密不可分，心理和社会因素对身体健康的影响，发挥着不可忽视的作用。对于 AMI 合并抑郁症的患者，积极有效的药物治疗联合心理干预能明显改善预后。

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