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急性心肌梗死诱发室性心律失常研究进展

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摘 要: 急性心肌梗死多是以冠状动脉急性、持续性缺血缺氧所引起的心肌坏死,是世界范围内病死和发病的常见原因。心律失常和传导障碍是常见的,它们与发病率和病死率的增加有直接的关系,目前,在临床上,大多数研究都是以减少急性心肌梗死所诱发的心律失常为准则,来减少发病率和病死率的。该文通过对近年来减缓心律失常的发生的研究报道进行综述,以期为临床诊断和治疗提供支持。

关键词: 急性心肌梗死;室性心律失常;进展
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Research Progress of Ventricular Arrhythmia induced by Acute Myocardial Infarction

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Abstract: Acute myocardial infarction (AMI) is a kind of myocardial necrosis caused by acute and persistent ischemia and hypoxia of coronary artery, which is a common cause of death and morbidity in the world. Arrhythmias and conduction disorders are common and are directly associated with increased morbidity and mortality. Currently, most clinical studies have focused on reducing arrhythmias induced by acute myocardial ischemia. To reduce morbidity and mortality. This article summarizes the experimental and clinical studies in recent years to illustrate that the prevention of arrhythmia after acute myocardial ischemia is a good way to treat acute myocardial ischemia.

Keywords: acute myocardial ischemia; arrhythmia; progress

急性心肌梗死(Acute myocardial infarction, AMI)是冠状动脉急性、持续性缺血缺氧所引起的心肌坏死。一段时间心肌梗死后,可导致心肌细胞电生理紊乱、血流动力学紊乱、代谢紊乱、心肌细胞坏死和凋亡,并伴有多种离子通道和β受体(β-AR)[1-2]异常及连接蛋白[3-5]减少等基因表达的异常改变。心律失常和传导障碍是常见的并发症,也是发病率和病死率增加的原因。室性心律失常(Ventricular arrhythmia, VA)包括心室纤颤(Ventricular Fibrillation, VF)以及持续性单形性和多形性室性心动过速的速率超过100次/min,持续至少30 s[6]。除窦性心动过速外,心房颤动(Atrial fibrillation, AF)是临床上最常见的心律失常[7-8]。目前,对于急性心肌梗死诱发心律失常的机制并没有完全阐释清楚,在治疗上,仅是通过不同方法来减缓心律失常的发生,本文通过对近年来减缓心律失常的发生的研究报道进行综述,以期为临床诊断和治疗提供支持。

1 AMI诱发VA的预测诊断

AMI多由冠状动脉粥样硬化所引起,AMI诱导心律失常以及急性和慢性心力衰竭。这种心脏病发作是世界范围内病死和发病的常见原因[9]。AMI发生后,将引起心脏心电图(如:ST段的抬高,病理性的Q波的发生,T波的倒置)、血流动力学、动脉血气指标、心肌酶学的改变[10-11]。因此,可以通过这一系列的变化及梗死的部位来预测AMI诱发VA的发生,来减少其所引起的病死率。

1.1 肌钙蛋白(Troponin, Tn)和C反应蛋白(C reactive protein, CRP)

Tn是肌肉组织收缩的调节蛋白,在肌肉收缩和舒张过程中起着重要的调节作用,血清中Tn升高反映了心肌细胞受损,其特异性与敏感性均高于以往常用的心肌酶谱,是诊断心肌损伤的重要生化指标。CRP作为一种敏感的急性时相蛋白,是机体非特异性反应的敏感标志物之一,可反应细胞因子的变化过程,是组织急性期损伤的反应性蛋白之一。Hodzic E等[12]发现:Tn与CRP水平和记录的VA呈正相关,Tn和CRP可作为心肌梗死前、下壁心肌梗死VA的指标,对预防、治疗VA和潜在恶性心律失常并可能导致病死具有重要诊断意义。Pei Z等[13]发现:血清胱抑素C(Cystatin-C, CysC)、超敏C-反应蛋白(Hypersensitive C-reactive protein, hs-CRP)、去甲肾上腺素(Norepinephrine, NE)和氨基端前脑钠肽(amino-terminal pro-brain sodium peptides, NT-

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proBNP）的水平呈儿茶素（Catestatin，CST）浓度依赖性升高，恶性心律失常的发生随CST水平升高而明显增加，CST可作为预测急性心肌梗死患者恶性心律失常的一种新的生物学指标。

1.2　心电图

心电图作为AMI诱发心律失常的一项重要指标，伴随心律失常的整个过程，因此，AMI中心电图的观察始终是必不可少的一部分。Arisha MM等[14]发现：AMI诱发心源性猝死（sudden cardiac death，SCD）或危及生命的VA运用心率震颤（heart rate turbulence，HRT）和T波交替（T-wave alternans，TWA）联合有较高的预测能力，可作为早期植入式心脏除颤器（Implantable cardiac defibrillators，ICD）植入的高危患者的鉴别诊断。Ch á vez-Gonz á lez E等[15]测量广泛性前壁AMI心室梗死部位QRS波和校正QT间期的宽度和离散度存在显著差异性，QRS波持续时间和离散程度的增加提示AMI早期发生VA的可能性大于校正QT间期的持续时间和离散度。学者[16-17]发现：AMI前（Early repolarization，ER）的存在能增加室性快速性心律失常（ventricular tachyarrhythmias，VTA）的风险。因此，ER可能是加重AMI致心律失常的底物，也可能是AMI诱发VA或猝死风险的一个标志。Al-Khatib SM等[18]发现：非ST段抬高急性冠状动脉综合征（ST-segment elevation acute coronary syndromes，STE-ACS）和持续性VA患者病死率明显高于STE-ACS心律失常的患者。因此，应尽可能通过随机临床试验来找出最能改善这些患者预后的治疗方法。因此，对于AMI后我们应着重运用HRT和TWA联合QRS波宽度和离散度与ER来预测VA的发生。

1.3　其他指标

AMI诱发心律失常的原因是复杂的，因此对于AMI诱发VA的预测也可以有不同的方式。学者[19-20]发现：梗死部位是预测AMI诱发心律失常并发症的重要指标。下壁或后壁AMI更有可能发生房室结传导异常，而前壁或侧壁AMI更有可能发生VF。有前壁或侧壁AMI或有完全心脏阻滞的患者在出院前病死的可能性更大。Gang UJ等[21]发现：AMI诱发高度房室传导阻滞（High atrioventricular block，HAVB）的长期发生率研究很少。多集中在HAVB急性阶段，且HAVB预示着一个不祥的短期和长期预后，可以作为预测急性心力衰竭（Acute heart failure，AHF）和VA的一个指标。学者[22-23]发现：低钾血症可影响心肌的静息跨膜电位和动作电位的多个阶段，并对心肌膜的自律性、导电性和兴奋性产生重要影响，从而增加心律失常发生的危险。血清钾的水平可作为预测和一定程度上改善心肌梗死溶栓（Thrombolysis in Myocardial Infarction，TIMI）危险评分。Hofmann F等[24]发现：心肌细胞肥大中超极化激活阳离子（hyperpolarization-activated cation，HCN）通道活性的增强能延长心室肌动作电位的复极，从而增加致心律失常的电位。学者[25-26]通过盐酸伊伐布雷定（Ivabradine，HCl）阻断HCN通道减少致死性VA的发生，因此，HCN通道的阻断对于预防心力衰竭猝死的患者是一种有效的方法。

2　AMI诱发VA的治疗

目前临床上治疗AMI诱导VA多采用抑制蛋白、阻滞通道、抑制交感神经的传导等技术来治疗心律失常。

2.1　经皮冠状动脉介入治疗（percutaneous coronary intervention，PCI）

PCI指经心导管技术疏通狭窄甚至闭塞的冠状动脉管腔，从而改善心肌的血流灌注。但是，在冠脉造影或介入过程中，造成的管腔狭窄等，这可能引起心律失常等并发症，从而增加病死率，因此，对于术后的心律失常的防治是关键的一步。Robbers LF等[27]经直接PCI治疗成功的AMI行晚期钆增强心血管磁共振成像（late gadolinium enhanced cardiovascular magnetic resonance imaging，LGE-CMR）量化心肌增强总量，AMI亚急性期的半暗区（最大信号强度的25%~50%）比例越大，发生VT的风险越大。半暗带大小的量化可能成为有用的未来风险分层的工具，并最终防止VA。Takahashi M等[28]经心肺骤停（cardiopulmonary arrest，CPA）复苏后行PCI治疗的AMI患者临床效果良好，但复发VA的危险性较高。因此，对于这类人群，临床医生应该认真考虑强化药物治疗和ICD植入术。

2.2　神经阻断

AMI可能通过神经传导来诱导心律失常的发生。因此，对于AMI诱导心律失常是否可以通过切断神经来治疗。Chen J等[29]发现：切断冠状静脉窦（coronary sinus，CS）和心脏大静脉（great cardiac vein，GCV）周围神经能显著降低VA发生和持续时间，延长有效不应期（effective refractory period，ERP），降低离散度，显著降低CS血中NE的水平。但是，心脏交感神经局部消融后，心率、平均动脉压及缺血面积均无明显变化。Huang B等[30-31]发现：过度活动的肾交感神经（Renal sympathetic nerve，RSN）在缺血性VA的发病机制中起重要作用，肾交感神经切断（Renal sympathetic denervation，RSD）能显著稳定正常心脏心室的电生理特性，减少AMI诱发VA，因此，RSD引起的心脏交感神经张力减弱可能会成为治疗VA的新方法。Wang S等[32]发现：脊髓刺激（Spinal cord stimulation，SCS）引起心室ERP明显延长，心率变异性（heart rate variability，HRV）显著增加，LSG功能和活性明显减弱，SCS可能通过抑制LSG活性来预防AMI诱导的VA。He B等[33]发现：给正常心脏低强度心房神经节丛（Ganglionated plexi，GP）刺激可引起心室ERP和显着延长动作电位时程（Action potential duration，APD），促进APD交替，但不改变心室APD恢复曲线的斜率和SMAX的空间离散度，低强度GP刺激可显著地降低AMI诱发VA，对VA起到保护作用。Linz D等[34]发现：肾去神经（renal denervation，RDN）可减少VA/VF的发生，减轻左心室缺血状态下舒张末压（left ventricular end-diastolic pressure，LVEDP）的升高，但并不影响梗塞面积、心室收缩力、血压和再灌注心律失常的变化。

2.3　离子通道

离子通道在AMI诱发VA过程中扮演着重要的

角色，离子通道几乎参与了所有VA的发生，因此，通过离子通道来减少VA的发生，也是学者所重点研究的。Yin Y等[35]发现：当AMI发生时，大量的氧自由基不能被清除，转化为羟基自由基。超氧化物歧化酶（superoxide dismutase，SOD）和Na^+-K^+-ATP酶活性降低，血清丙二醛（malondialdehyde，MDA）含量升高，SOD、MDA和Na^+-K^+-ATP酶活性测定结合动态心电图对AMI的预后和治疗有重要意义。Zhao Y等[36]松参养心胶囊（Shensong Yangxin capsules，SSYX）能有效地预防缺血性心律失常，延长APD，抑制瞬时外向钾电流（Transient outward K^+ current，ITO）和内向整流钾电流（Inward rectifier K^+ current，IK1），从而降低心肌自律性和兴奋性，减轻心肌Ca^{2+}超载，减轻和消除心肌超微结构损伤。因此，SSYX有可能成为治疗缺血性心律失常的有效药物。Gui L等[37]抑制小电导Ca^{2+}激活K^+（small-conductance Ca^{2+}-activated K^+，SK）通道能缩短单相动作电位时程（monophasic action potential duration，MAPD），减少自发性VA及心律失常，这可能是防治AMI后VA的重要途径，这一推测被Hundahl LA等[38]通过N-（吡啶-2-基）-4-（吡啶-2-基）噻唑-2-胺（N-（pyridin-2-yl）-4-（pyridin-2-yl）thiazol-2-amine，ICA）阻断SK通道减轻缺血性室性心律失常的实验所证实。Zhang J等[39]揭示miR-1231是由缺血状态引起的致心律失常因子，通过一种调节心肌细胞钙信号反应钙通道蛋白cacna2d2的表达而加剧心律失常。因此，干预内源性miR-1231水平可能是抑制缺血心律失常的一种新的潜在途径。Vrana M等[40]发现：抗心律失常药物（antiarrhythmic drugs，AADs）对于预防心律失常的SCD存在风险，但AADs仍在抑制术后心律失常和预防ICD术后SCD方面发挥着不可替代的作用。在交感神经激活（sympathetic neural activation，SNA）中，AADs的促心律失常作用的风险可以通过与β-受体阻滞剂联合使用而降低。抑制恶性VA的最好方法是切断心脏交感神经。

2.4 室性心动过速/室颤（Ventricular tachycardia，VT；Ventricular tachycardia，VF）

VT是指发生在希氏束分叉以下的束支、心肌传导纤维、心室肌的快速性心律失常。VF是一种严重的异位心律，心室丧失有效的整体收缩能力，被各部心肌快而不协调的颤动所代替，是临终前的一种致命性心律失常。因此，有效的控制VT/VF也是对心律失常一种治疗方法。Endo T等[41]发现：血清二十碳五烯酸（Eicosapentaenoic acid，EPA）水平降低是AMI期VT/VF发生的危险因素，J波也参与了VT/VF的发病机制。低水平的血清EPA与J波的出现之间可能是通过KATP通道的激活存在机制联系，这可能是缺血诱导VT/VF的一种新的发病机制，因此，补充EPA可预防AMI诱发VT/VF。Hashimoto T等[42]发现：血清中高EPA/花生四烯酸（arachidonic acid，AA）比值对于AMI急性缺血期具有抗心律失常作用。Liao K等[43]发现：电刺激颈动脉压力感受器（Electrical carotid baroreceptor stimulation，CBS）能使AMI前心室ERP明显延长，恢复动作电位的斜率，减少AMI时室性早搏和VT/VF发作次数，但CBS对缺血面积无明显影响。Chou CC等[44]发现：丹曲林（Dantrolene）抑制室性早搏和（或）延长复极，抑制自发性VF，降低VF诱导性。Liu YB等[45]证明：血脂异常是MI急性期VT/VF的独立预测因子，其作用可能是由基线血脂和甘油三酯在MI急性期心律失常发生中起到了显著的作用。结论也部分解释了降脂药物在降低心脏性猝死风险方面的有益作用。

2.5 受体与蛋白

AMI在一定时间的缺血条件下形成心律失常，通过药物改善心律来抑制心律失常的发生。因此，通过介质来调节从而达到治疗心律失常的目的。Li X等[46]发现：Let-7e通过大鼠心脏β1肾上腺素能受体（β1-adrenoceptor，β1-AR）产生有效的抗心律失常作用，为进一步研究MI大鼠心肌组织中mRNA1-AR和miRNAlet-7e的过度表达可能抑制AMI诱发心律失常的机制提供了新的思路。这也表明靶向miRNAlet-7e可能是调节β1-AR的一种有前途的治疗策略。Chang HY等[47]发现：连接蛋白（Connexin-43，Cx43）在正常心肌组织中呈强阳性表达且分布规则而在AMI中的分布明显紊乱，其表达降低。因此，Cx43的重构是MI后恶性心律失常发生的重要分子解剖学基础，替米沙坦能降低MI后恶性心律失常的发生率，其可能是通过抑制IL-17来增加Cx43的表达从而减少恶性VA。Zhang J等[48]发现：甘松（Nardostachys chinensis，NC）抗心律失常的作用机制可能与减少Cx43降解，改善心肌梗死区Cx43再分布有关，NC有可能成为未来预防AMI院前致死性VA的一种有前途的药物。Javidanpour S等[49]发现：迷迭香酸（Rosmarinic acid，RA）预处理能明显预防心肌肥厚、脂质过氧化、心肌收缩力和舒张性减弱、SIAs和心肌组织NCX1的过度表达，对张力性心肌缺血有一定的抑制作用。

2.6 其他因素

AMI后诱发心律失常的机制是复杂的，目前，对于引起心律失常的原因，并不能完全阐释清楚。因此，任何一个因素将可能导致心律失常的发生。Fan P等[50]发现：骨髓干细胞（Bone marrow stem cells，BMSCs）和肌浆网钙ATP酶（Sarcoplasmic reticulum Ca^{2+}-ATPase，SERCA2a）基因修饰的BMSCs移植可显着改善AMI心功能及BMSCs+rAd.SERCA2a能有效改善大鼠梗死心肌组织的传导，减少MI诱发心律失常的发生。Huikuri HV等[51]发现：MI早期心率震荡（heart rate turbulence，HRT）缺乏似乎是改善严重心律失常的特异性标志。Mirzaiepour F等[52]发现：鸦片成瘾对MI后心律失常的发生有较强的预测作用，鸦片成瘾者窦性心动过速、窦性心动过缓和心房颤动的发生率明显高于非鸦片成瘾者。表明，鸦片成瘾是MI诱发心律失常的一种很强的潜在的风险。Athar MK等[53]发现：即使在控制红细胞的积压和完全排除各种传统危险风险因素外，输送充盈红细胞（Packed red blood cell，PRBC）依旧能增加AMI诱发心律失常和传导异常的风险。Yeh CF等[54]发现：体外膜氧合（Extracorporeal Membrane Oxygenation，ECMO）是治疗AMI顽固性心律失常的一种可行

的抢救方法和血运重建的桥梁。

3 AMI诱发VA的机制

AMI诱发VA的机制是复杂的，神经通路、离子通道等都可以不同程度的影响VA的发生。Yu X等[55]消融远端Marshall韧带（ligament of Marshall，LOMLSPV）可抑制AMI时的VAS，其机制可能与切断左侧星状神经节（left stellate ganglion，LSG）到脑室的交感通路有关。学者[56-57]发现：张力性心律失常（Stretch- induced arrhythmias，SIAs）的发生与细胞内Ca^{2+}稳态调节的基因反向转运蛋白（NCX1）的过度表达和活动有关，在舒张性心肌组织中，由于Na^{+}、Ca^{2+}和K^{+}等一系列离子的开放和随后的内向电流，静息膜电位升高，导致Ca^{2+}稳态障碍和舒张期渗漏。在这一过程中，NCX1作为一种钙稳态调节因子被激活，随后被延迟再被除极，这是致死性心律失常发生的主要因素。Tao YK等[58]发现：人和猪的血小板活化因子（platelet activating factor，PAF）水平分别在AMI和模拟缺血时升高，当发生致命性心律失常时，PAF水平甚至更高。在非缺血条件下，PAF使APD90缩短，而在早期模拟缺血条件下，APD90缩短更为明显。说明PAF对心肌的作用可能是通过多个通道介导的。

急性心肌缺血后诱发VA是AMI的危险因子和疾病加剧增高的病死原因之一，目前，对于AMI诱发VA的预防多通过心电图（ST段的抬高，病理性的Q波的发生，T波的倒置等）、离子的跨膜、蛋白质的变化、心肌酶学的变化及LVEF等来进行，治疗方面多通过血运重建、改善梗死心肌电的导电性、植入式心律转复除颤器、改善细胞膜的离子通道等方式降低诱发VA或治疗VA来减少心肌梗死患者的发生率和病死率。但是，对于AMI后诱发VA的机制目前还不明确，需要更多的研究。◆

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