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高血压心肌纤维化的病理机制及治疗进展

薛　琳，邓　玮*

重庆医科大学附属第二医院全科医学科，重庆

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摘　要

高血压是全球心血管疾病预后不良的首要危险因素，长期血压控制不佳最终可演变为高血压心脏病。以成纤维细胞过度增殖，并伴有胶原异常沉积分布为特征的心肌纤维化是高血压心脏病的重要病理基础，可导致心源性猝死等严重心血管并发症。现对高血压心肌纤维化病理生理机制及治疗进展进行梳理，以期为延缓甚至逆转高血压心脏病的不良结局提供可能。

关键词

高血压，心肌纤维化，发病机制，治疗

Pathological Mechanisms and Therapeutic Advances in Hypertensive Myocardial Fibrosis

Lin Xue, Wei Deng*

Department of General Practice, The Second Affiliated Hospital of Chongqing Medical University, Chongqing

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Abstract

Hypertension stands as the primary risk factor for poor prognosis in global cardiovascular diseases, with long-term uncontrolled blood pressure ultimately progressing to hypertensive heart disease. Myocardial fibrosis, characterized by excessive proliferation of fibroblasts accompanied by abnormal collagen deposition and distribution, constitutes a critical pathological basis for hypertensive heart disease, predisposing to severe cardiovascular complications including sudden cardiac death. This review synthesizes current knowledge on the pathophysiological mechanisms underlying

*通讯作者。

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hypertensive myocardial fibrosis and recent therapeutic advancements, aiming to provide insights for potential strategies to delay or even reverse the adverse outcomes of hypertensive heart disease.

Keywords

Hypertension, Myocardial Fibrosis, Pathogenesis, Therapeutics

1. 引言

高血压是一种以体循环动脉血压升高为主要临床表现的常见的慢性疾病。据 2024 年《中国高血压防治指南》[1]显示，我国成人高血压患病率为 27.5%左右，呈逐年上升趋势，且总体预防和治疗的效果仍然较差。高血压是心血管疾病至关重要且独立的风险要素[2]。长期血压控制不良，致使心肌硬度增加，心肌活动受限，心室顺应性下降，最终导致心肌收缩和扩张功能异常。心肌纤维化(myocardial fibrosis, MF)是高血压心脏病的核心病理基础[3]。心肌纤维化以心肌细胞肥大、心肌成纤维细胞过度增生和大量胶原沉积为病理特征，显著增加了心力衰竭、心肌缺血、心律失常和猝死等心血管事件的风险[4]。近年来，高血压心肌纤维化的机制认识及干预手段取得显著进展。本文系统总结该领域的研究现状，以期为其临床治疗提供参考。

2. 高血压心肌纤维化的病理机制

2.1. 肾素－血管紧张素－醛固酮系统

肾素－血管紧张素－醛固酮系统(renin-angiotensin-aldosterone system, RAAS)过度激活是高血压心肌纤维化关键驱动因素，是压力超负荷影响下心肌细胞的主要反应[5]。RAAS 激活可对心脏成纤维细胞(CFs)的增殖过程起到促进作用，进而推动心肌间质胶原的产生。Ang II (angiotensin II, Ang II)和 ALD (aldosterone, ALD)是导致 MF 的主要效应分子，并通过各种信号通路参与 MF 的形成[6]。研究表明受到压力刺激的心肌细胞、免疫细胞和成纤维细胞介导血管紧张素 II 的促纤维化作用，血管紧张素 II 与 1 型血管紧张素受体(AT1)结合，引发心脏内促纤维化效应，包括促使 CFs 增殖并向肌成纤维细胞分化，激活胶原生物合成相关通路，同时对胶原降解通路起到抑制效果[7]。ALD 的作用与 Ang II 相似，能够推动 CFs 进行增殖活动，提升心肌间质的分泌水平并增加心肌胶原纤维的含量[8]。醛固酮通过刺激心脏、血管的炎症反应、促进细胞外基质形成及细胞肥大、凋亡，导致心脏结构重塑和功能障碍[9]。除了循环中的 RAAS 系统，Ang II 和 ALD 还可通过自分泌和旁分泌的途径，在心脏组织内部产生局部纤维化效应[10]。

2.2. 关键信号通路

2.2.1. TGF-β/Smad 信号通路

转化生长因子 β (ransforming growth factor-β, TGF-β)是一种纤维化生长因子，在细胞分化与功能调控等过程中发挥重要作用，在哺乳动物中主要包括 TGF-β1、TGF-β2 和 TGF-β3 三种亚型，其中 TGF-β1 在心肌纤维化中发挥关键作用[3]。TGF-β1 通过诱导心肌成纤维细胞的分化增殖、调控基质金属蛋白酶系统促进胶原合成和沉积、抑制胶原降解酶活性以及刺激细胞外基质(ECM)的合成促进心肌纤维化[10]。TGF-

β/Smad 通路是心肌纤维化进程中最为经典的信号通路。TGF-β 首先与细胞膜表面受体结合，招募并磷酸化下游的 Smad2 和 Smad3，并与 Smad4 结合形成转录复合体，激活 R-Smad 蛋白，将信号传递至细胞质内，当 R-Smad 和 Co-Smad 融合时，其被转移至细胞核内并与靶基因融合，调控下游纤维化相关基因的激活与表达，促进心肌纤维化形成[11]。Smad7 是 TGF-β/Smad 通路的重要负调节因子，通过与 TGF-βI 型受体结合，阻止 R-Smads 的磷酸化，从而抑制 TGF-β 信号的转导，或招募 E3 泛素连接酶，促进 TGF-βI 型受体的泛素化和降解，进一步减弱 TGF-β 信号[3]。除了通过经典的 Smad 依赖的信号通路，TGF-β 还可激活 MAPK、PI3K/AKT、RhoA-ROCK 等非经典信号通路调控心肌纤维化[12]-[14]。

2.2.2. Wnt/β-Catenin 信号通路

Wnt 信号通路根据信号传导机制的不同分为经典通路和非经典通路两大类。经典 Wnt/β-catenin 信号通路在进化过程中高度保守，对胚胎、器官和神经发育、机体损伤修复及维持组织内环境的稳态等关键生理活动有重要意义[15]。Wnt 信号与细胞膜上的卷曲跨膜受体 Frizzled 蛋白(FZD)和共同受体低密度脂蛋白受体相关蛋白 5/6 (LRP5/6)结合，启动诱导信号，抑制 β-catenin 的降解，使之稳定化聚集并转移到细胞核中与 T 细胞因子/淋巴细胞增强因子(TCF/LEF)相互作用，调节基因表达[16]。Wnt/β-catenin 通路的特异性激活可促进心脏成纤维细胞转化为肌成纤维细胞，并刺激细胞因子合成参与了心肌纤维化[17]。在人体组织中，Wnt 信号通路通常保持沉默，但当机体患有高血压性心脏病时，其中的 Wnt/β-catenin 信号通路可能被异常激活。研究证明成纤维细胞可特异性激活 Wnt/β-catenin 介导心脏压力超负荷小鼠的纤维化和心肌细胞肥大[18]。因此，调节 Wnt/β-catenin 信号通路为高血压心肌纤维化的治疗策略提供了新的视角和理论依据。

2.3. 炎症与免疫调控

免疫炎症反应是 MF 的重要诱导剂[19]。在多种心血管疾病中，免疫炎症反应和 MF 常共存于同一病变部位。在高血压心肌损伤和重塑心肌中，免疫系统逐渐被激活，并释放如 TNF-α、IL-1β 和 IL-6 等多种炎症因子[20]。细胞因子参与调节细胞生长和修复受损组织。炎性细胞因子、生长因子、趋化因子等通过直接作用于成纤维细胞，刺激成纤维巨噬细胞和淋巴细胞的募集和活化，以及触发血管细胞和心肌细胞中的成纤维程序，持续调节受损细胞的纤维化[3]。

巨噬细胞介导的炎症在高血压心脏重构中起着重要作用，其中 M1 型巨噬细胞通过产生炎症因子和基质金属蛋白酶等物质，导致机体的炎症损伤，在心肌损伤早期发挥作用[21]；M2 型巨噬细胞促进多胺和脯氨酸的生物合成、胶原蛋白形成和组织修复，在心肌损伤晚期发挥作用，参与心脏纤维化修复[22]。Kassem 等人发现 M1 巨噬细胞的极化过程可被活化的 M2 型巨噬细胞抑制[23]，这一机制可减轻机体的炎症反应，促进组织修复。在高血压患者中，巨噬细胞通过影响 Ang II/ALD 的水平，激活 NLPR-3 炎症小体、分泌细胞因子，增加巨噬细胞的数量、诱导氧化应激反应、激活 TGF-β/Smad3 信号，来促进心肌成纤维细胞转化为肌成纤维细胞，调节细胞外基质的沉积介导纤维化[24] [25]。此外，巨噬细胞还可以通过改变基质金属蛋白酶与基质金属蛋白酶抑制剂的平衡等导致胶原沉积[26]。

不同的 T 细胞亚群通过分泌细胞因子和生长因子，影响细胞外基质重塑和纤维化进程[27]。Th1 细胞是促炎细胞因子，可以促进 MF 的发生和发展，而 Th2 细胞具有抗炎作用，可以抑制 MF。Nevers 等表明，在主动脉横缩的小鼠中，活化的 Th1 细胞和 CFs 之间的直接粘附可以促进 TGF-β 的合成，导致 CFs 转化为肌成纤维细胞[28]。Th1 细胞表达的增加增强了胶原交联，并最终导致心肌僵硬度的增加，而 Th2 细胞表达的增加减少了心肌胶原的量并抑制了 MF。

2.4. 氧化应激

氧化应激是指体内氧自由基的大量产生和氧化物清除能力的下降，导致体内氧化和抗氧化系统失衡，

从而引起细胞氧化损伤的过程[19] [29]。过度压力负荷可以使心肌组织耗氧量增加，心肌组织处于相对缺氧状态，细胞内异常氧化导致心肌细胞功能异常、坏死和凋亡，最终致使心肌纤维化[30]。活性氧(reactive oxygen species, ROS)的产生是氧化应激的关键因素。研究表明 ROS 可能介导 Ang II、ALD、TGF-β 的促纤维化作用[31]。TGF-β 刺激线粒体产生 ROS 并诱导烟酰胺腺嘌呤二核苷酸磷酸(NADPH)氧化酶激活放大并维持 TGF-β 诱导的氧化应激[32]。ROS 刺激 CFs 的 ECM 基因转录，并参与调节 ECM 的翻译后修饰，增加心肌胶原蛋白的产生[3]，还可通过激活基质金属蛋白酶，减少 CFs 中纤维胶原的合成[33]。细胞因子驱动的 NADPH 氧化酶的诱导过程在 ROS 生成中发挥关键作用。Zhao 等人的研究表明心力衰竭时神经内分泌系统被激活，心室机械压力增加，通过激活 CFs 中 Nox4/Akt/mTOR/NF-κB 信号通路增加 TGF-β 的水平，从而诱导 CFs 的增殖和分化并促进 MF [34]。Liu 等也证明 Nox4/ROS/CTGF 信号通路在压力超负荷诱导的 MF 的病理过程中起关键作用[35]。

2.5. 细胞外基质失衡

心肌细胞外基质(extracellular matrix, ECM)主要由 CFs 合成的 I 型胶原和 III 型胶原组成，胶原纤维合成与降解失衡是导致高血压 MF 的关键环节，胶原含量可用于评价 MF 的程度[36]。基质金属蛋白酶(MMPs)和特定 MMPs 的组织抑制剂 TIMPs 之间的相互作用是 ECM 维持稳态的关键。MMPs 是主要由 CFs 分泌的一个蛋白水解酶家族，不仅可以直接降解 ECM 并激活其他类型的 MMPs 以产生级联效应，还可以调节胶原蛋白的合成，以使正常胶原蛋白被缺乏结缔结构的纤维基质取代[37]。研究证明，MMP-2 和 MMP-9 的过度表达可以促进 CFs 的增殖、分化和迁移，以及破坏胶原网络，并导致 ECM 沉积和 MF 形成[38]。TIMPs 以 1:1 的比例与 MMPs 形成复合物，阻断 MMPs 与底物的结合并抑制其活性，是 ECM 更新、组织重塑和细胞行为的重要调节因子，正常情况下，MMPs 和 TIMPs 的表达和活性保持着相对平衡，确保细胞外基质的正常代谢。当受到炎症、氧化、损伤等致病病理因素刺激时，心脏组织中 MMP 和 TIMP 的比例失衡可促进 MF 的发展[37]。Kostov 等的研究指出高血压患者血清中 MMP-1 水平升高可能与心脏 ECM 中胶原降解增加相关，而 TIMP-1 水平升高可能促进 ECM 的沉积、心脏和动脉血管的病理性重构以及纤维化的发生[39]。

2.6. 非编码 RNA 调控

2.6.1. miRNA

微小 RNA (microRNA, miRNA)是一类长度为 18~25 个核苷酸的内源性非编码 RNA，通过促进靶基因 mRNAs 的降解或抑制靶基因的翻译来抑制靶分子的表达[40]。miRNA 经多条信号通路参与了 MF 的调控。抑制 miR-21 的表达可以调控程序性细胞死亡 4 (PDCD4)和转录激活蛋白 1 (AP-1)和 TGF-β1 信号通路改善高血压诱导的心脏重塑[41]。在慢性心脏压力超负荷的小鼠模型中，miR-29 的抑制或遗传缺陷可以防止心脏肥大和纤维化[42]。miR-133 和 miR-101 影响 CFs 的生理功能并发挥抗纤维化作用[43] [44]。Deng 等发现小鼠过表达 miR-451a 可抑制心肌细胞和成纤维细胞中 TGF-β1 的产生，使 TGF-β1/SMAD2/3 信号失活，抑制肌成纤维细胞分化和促炎细胞因子表达以减轻心脏纤维化和炎症[45]。

2.6.2. lncRNA

长链非编码 RNA (long noncoding RNA, lncRNA)是一类长度大于 200 个核苷酸的非编码 RNA，主要通过与 DNA、RNA 或蛋白质的相互作用来调节 MF 相关基因的表达并影响 CF 增殖和 ECM 分泌[46]。Li 等研究结果提示 lncRNA MALAT1 通过抑制 MyoD 转录而促进高血压大鼠的 MF [47]。在心脏 CFs 中的心脏特异性 lncRNA Crnde 抑制 Smad3 与 α 肌动蛋白(α-SMA)基因启动子的结合，减少 CFs 向肌成纤维细胞的转化，发挥抗心肌纤维化的作用[48]。

2.6.3. circRNA

环状 RNA (circular RNA, circRNA)是一类形成闭环结构的非编码 RNA 分子，其环状结构稳定，没有自由末端，不易被核酸酶降解。circRNA 通过调节基因表达并编码蛋白质，在心血管系统分子机制的调节中具有重要作用[49]。circRNA 可以作为竞争性内源性 RNA 与 miRs 结合，从而影响靶基因的表达，从而影响 MF 的病理过程[50]。Li 等指出 circRNA_000203 在 Ang II 处理的 CFs 中显著增加，并通过抑制 miR-26b-5p 和 miR-140-3p 导致 Gata4 水平升高而加剧心脏肥大[51]。circITGa9 是一种源自整合素-α9 的 circRNA，在心脏肥大患者中表现出显着的上调，体内实验显示，circITGa9 水平升高会驱动小鼠心脏重塑和纤维化，靶向 circITGa9 可减轻心脏重塑和纤维化[12]。

2.7. 肠道菌群失调

近年来，肠道菌群失调与高血压心肌纤维化的关联研究取得显著进展。肠道菌群失调通过代谢产物(如短链脂肪酸、氧化三甲胺、色氨酸衍生物等)直接调控心肌间质纤维化进程[52]。如乙酸和丙酸可通过抑制心肌成纤维细胞活化，减少胶原沉积，从而改善纤维化[53]。高血压状态下，肠道微循环障碍导致肠黏膜屏障破坏，细菌内毒素(如脂多糖)和促炎因子(IL6、TNF-α)入血，引发全身性低度炎症，进而激活 TGF-β/Smad 和 NF-κB 等心肌纤维化关键信号通路[52] [54] [55]。动物实验进一步证实，自发性高血压模型中肠道菌群多样性降低与心肌纤维化程度呈正相关，拟杆菌门/厚壁菌门比例变化可能通过调节血管紧张素II信号影响纤维化进程[56] [57]。肠道菌群也可通过调控 T 细胞分化和巨噬细胞极化参与纤维化过程。研究发现，压力超负荷诱导的心力衰竭模型中，菌群失调促进 Th17 细胞活化，加剧心肌纤维化，而抗生素清除肠道菌群可减少心脏 T 细胞浸润和纤维化程度[58]。肠道与心脏具有双向作用，高血压所致心脏后负荷增加和肠道低灌注可引发肠道缺血、水肿及屏障破坏，形成“心脏损伤→肠道菌群紊乱→心肌纤维化恶化”的恶性循环[55] [57] [59]。而恢复菌群平衡(如补充益生菌或膳食纤维)可改善肠道屏障功能，降低血压并减轻左心室纤维化[60]。

3. 高血压心肌纤维化治疗策略

高血压心肌纤维化与心室重构、心力衰竭及心律失常等不良结局密切相关，治疗上需兼顾血压控制与抗纤维化，通过药物治疗、基因与细胞治疗、生活方式干预等手段，抑制纤维化进程、改善心脏功能及预后。

3.1. 药物治疗

3.1.1. 传统降压药物

长期压力负荷通过多个途径致使 MF 的发生，降低血压可减缓 MF 进程。传统的降压药物主要包括血管紧张素转换酶抑制剂(ACEI)、血管紧张素II受体拮抗剂(ARB)、β 受体阻滞剂、利尿剂、钙通道阻滞剂(CCBs)。ACEI 与 ARB 主要作用于 RAAS 系统，从而发挥降血压及抗纤维化作用。β 受体阻滞剂可选择性与肾上腺素能受体结合，起到减慢心率、减弱心肌收缩力和心肌耗氧量的作用，间接抑制纤维化进程。利尿剂是治疗高血压引起的心室重构时唯一能够控制液体潴留的药物[61]。细胞内 Ca^{2+}参与一些生长因子的信号转导，进而促进 CFs 的形成及增殖[62]。因此，CCBs 具有降低血压和抗 MF 的直接作用。尽管上述药物可在一定程度上减缓心肌纤维化进程，但完全逆转纤维化较为困难，且治疗疗效不稳定、药物治疗依从性差、肝功能损害和电解质失衡等限制其应用。

3.1.2. 靶向纤维化通路的新型药物

目前，临床上针对心肌纤维化的治疗药物主要涉及 RAAS 系统及 TGF-β 信号通路靶点。血管紧张素

受体脑啡肽酶抑制剂(angiotensin receptor/neprilysin inhibitor, ARNI)沙库巴曲/缬沙坦(LCZ696)同时增加利钠肽循环水平并阻断血管紧张素 II 型受体，可改善心力衰竭患者的心脏功能并减少心肌肥厚[63]。LCZ696 诱导的心功能改善与心肌胶原体积分数和 I、III 型胶原含量降低相关，表明对心脏纤维化有影响[64]。在压力超负荷诱导的心脏纤维化的动物模型中，LCZ696 也可防止心脏肥大并改善心脏功能[65]。其机制为降低 smad3 的活性和增加抑制性 smad7 的表达，从而使 TGF-β 途径失活调节心肌纤维化，也可能通过其对 Wnt/β-catenin 途径的抑制活性来介导[66]。LCZ696 已被批准用于射血分数降低的心力衰竭患者，但在高血压心肌纤维化中的应用需进一步研究。

考虑到 TGF-β 在心脏纤维化中的中心作用，阻断 TGF-β 信号传导活性被认为是对抗纤维化的有前途的方法。吡非尼酮通过降低 TGF-β 表达，已获批用于特发性肺纤维化的治疗，研究证实，它对心脏具有保护功效[67]。以 TGF-β 为靶点的药物治疗 MF 具有广阔的发展前景，但其有效性和安全性制约着临床应用。既往研究发现长期应用严重的心脏毒性和肝毒性[68] [69]，因此，探索安全有效的 TGF-β 抑制剂是未来研究的重点。

恩格列净、达格列净等钠-葡萄糖协同转运蛋白 2 抑制剂(SGLT2i)药物除了降糖作用外，还可通过减轻氧化应激、改善心肌能量代谢，间接抑制纤维化，恩格列净对 Ang II 依赖性高血压的心脏保护作用可能是由心肌交感神经活动和炎症的减少引起的，并且与血压和血糖水平的调节无关[70]。达格列净通过调节 TGF-β 信号通路改善 Ang II 诱导的心肌重构[71]，可能对高血压心肌纤维化有潜在治疗效果。

3.1.3. 中医药治疗

高血压 MF 发病机制复杂，中医药因其多机制、多靶点的调节作用成为治疗高血压心肌纤维化极具潜力的热点研究领域[72]。提取自益母草的益母草碱能拮抗 Ang II 诱导的心肌肥厚和纤维化，改善高血压心脏重塑，动物实验显示其能显著降低胶原沉积和 TGF-β1 表达[73]。经证实复方制剂真武汤可通过调控 TLR4/NF-κB 和 Nrf2/HO-1 通路抑制炎症和氧化应激，减轻高血压相关心肌纤维化[74]。松龄血脉康胶囊通过降低血压和抑制心肌重构间接改善心肌纤维化[75]。中医药干预高血压心肌纤维化的核心机制涉及调节 RAAS 系统的过度激活、抗纤维化信号通路调控、CFs 增殖和分化减少心肌胶原沉积、抑制免疫炎症反应、减少氧化应激损伤等[76]。其优势在于多靶点干预、副作用较少，适用于长期管理，然而部分复方制剂的机制需进一步明确，且缺乏高质量的大规模临床验证。

3.2. 生活方式干预

生活方式干预是高血压心肌纤维化防治的重要基础。高钠、高脂肪、高糖饮食可影响肠道微生物群组成和功能的改变，导致微生物群衍生代谢物的变化以及肠道屏障和免疫功能的损害，并可能通过直接影响成纤维细胞对纤维胶原的代谢或慢性炎症激活间接作用导致心肌纤维化[52] [77]。低钠饮食通过促进保护性肠道微生物群落的生长，从而减少全身炎症，这可能有助于减少心肌纤维化[78]，高纤维饮食改善了肠道微生物群落的丰度，已被证明可以降低高血压模型中的血压和心肌纤维化。此外，规律运动、戒烟限酒、减轻体重、管理压力等生活方式的改变，也能够有效控制血压，延缓心肌纤维化的进程，降低心血管疾病的风险，提高患者的生活质量。

3.3. 基因与细胞治疗

基因与细胞疗法研究在近年来取得显著进展。通过重组腺相关病毒(rAAV)递送 SIRT7 基因可调节铜代谢相关通路，抑制血管紧张素 II 诱导的心肌纤维化和重构[79]。靶向心肌转录重塑的 JP2NT 基因治疗也在心脏压力模型中显示出显著疗效[80]。但基因治疗常用的载体腺相关病毒(AAV)，可能引发剂量依赖性免疫反应，包括肝毒性、获得性溶血性尿毒症综合征和心肌炎[81]。miRNA 通过基因转录前后的多种

途径参与纤维化的发生和发展，如 miR-29b 通过外泌体递送可抑制成纤维细胞向肌成纤维细胞转化，减少胶原沉积[82]，但部分基因(如 miR-654-3p)的过度沉默可能加重心肌损伤[83]，需精准调控基因表达水平以避免脱靶效应。

嵌合抗原受体 T 细胞(CAR-T)疗法通过体外重编程患者 T 细胞并回输体内，靶向成纤维细胞活化蛋白(FAP)可显著减少小鼠心肌纤维化并促进功能恢复[84]。但改造后的 CAR-T 细胞因长期存在，可能增加未来组织损伤风险。有研究利用 CD5 靶向脂质纳米颗粒递送经修饰的 mRNA 至心衰小鼠体内，可生成靶向 FAP 的瞬时抗纤维化 CAR-T 细胞，通过清除肌成纤维细胞减少心脏纤维化并改善心功能。由于 mRNA 未整合至 T 细胞 DNA，显著降低了细胞改造的毒副作用[85]。

此外，利用 CRISPR/Cas9 技术靶向编辑纤维化相关基因，直接移植心肌细胞移植到心脏损伤部位或内源性诱导祖细胞或心肌细胞增殖来改善心脏功能的干细胞疗法等，也为心肌纤维化治疗提供新的希望[86]。目前基因与细胞疗法的大多数研究处于临床前阶段，需优化载体设计和细胞疗法策略以降低免疫原性和脱靶风险，并解决包括载体基因组整合导致的突变风险，以及持续表达治疗基因可能产生的未知副作用等长期安全性及成本效益问题。

4. 总结及展望

长期高血压可导致心肌胶原组织比例失衡，心肌僵硬度增加，进而降低心室顺应性及心室收缩与舒张功能，最终引起心肌重构。目前，关于高血压心肌纤维化的发病机制复杂，涉及多个环节和多种因素的相互作用，治疗策略日益多样化，药物治疗不断优化，中医药治疗、细胞治疗和基因治疗展现出良好的应用前景。然而，仍有许多问题亟待解决，如深入阐明发病机制中的关键分子靶点，开发更有效的治疗技术，提高治疗的安全性和有效性等。未来需加强基础与临床研究的协同创新，推动精准医学在心血管领域的应用，随着多学科交叉融合，有望在高血压心肌纤维化的防治方面取得更大突破，为患者带来更好的治疗效果和预后。

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