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Hans汉斯

多囊卵巢综合征相关心血管代谢健康的研究现状

王月媛，郝丽娟*

重庆医科大学附属妇女儿童医院妇科，重庆

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摘　要

多囊卵巢综合征(Polycystic Ovary Syndrome, PCOS)作为育龄期女性常见的生殖内分泌疾病，与患者的生殖功能、代谢失调及精神心理等诸多方面存在紧密关联，对女性的身心健康构成重大威胁。近年来大量研究表明，PCOS患者心血管代谢疾病(如2型糖尿病、高血压、动脉粥样硬化等)的患病风险显著增加，且贯穿女性的全生命周期。目前，关于PCOS患者的心血管代谢风险的系统性研究仍有待完善。本文从PCOS的流行病学特征、心血管代谢异常风险、临床表现及管理策略等维度展开综述，旨在为PCOS相关心血管代谢疾病的早期干预和长期管理提供循证依据，深化对PCOS女性心血管代谢问题的认知。

关键词

多囊卵巢综合征，心血管代谢，胰岛素抵抗，肥胖，血脂异常，肠道微生物群

Cardiometabolic Health in Polycystic Ovary Syndrome (PCOS): A Comprehensive Review of the Current Research

Yueyuan Wang, Lijuan Hao*

Department of Gynecology, Women and Children's Hospital of Chongqing Medical University, Chongqing

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Abstract

Polycystic Ovary Syndrome (PCOS), a common reproductive and endocrine disorder among women of reproductive age, is closely associated with multiple dimensions of patients' health, including

*通讯作者。

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reproductive dysfunction, metabolic imbalance, and psychological status, thus posing a substantial threat to their physical and mental well-being. Mounting evidence in recent years has demonstrated that women with PCOS face a markedly elevated risk of developing cardiometabolic diseases such as type 2 diabetes mellitus, hypertension, and atherosclerosis, and this heightened risk persists throughout their entire lifespan. Nevertheless, systematic research focusing on the cardiometabolic risks in PCOS patients remains insufficient and warrants further refinement. This review synthesizes current findings from the perspectives of PCOS epidemiological features, cardiometabolic risk profiles, clinical manifestations, and management strategies. It aims to provide evidence-based foundations for the early intervention and long-term management of PCOS-related cardiometabolic diseases, as well as to deepen the comprehensive understanding of cardiometabolic abnormalities in women with PCOS.

Keywords

PCOS, Cardiometabolic, Insulin Resistance, Obesity, Dyslipidemia, Gut Microbiota

1. 多囊卵巢综合征：从生殖综合征到全身心疾病的转变

多囊卵巢综合征(PCOS)是育龄期女性中常见但复杂的生殖内分泌紊乱疾病，全球患病率约为8%~13% [1]。在临床诊疗中，月经失调、不孕、痤疮、肥胖等是育龄期和青春期PCOS女性的主要临床表现，也是该人群就诊的核心原因。糖尿病、高血压、血脂代谢紊乱、冠状动脉粥样硬化及子宫内膜癌等，则成为中老年PCOS患者的主要并发症与远期临床表现[1] [2]。PCOS的危害早已超越传统认知中的生殖健康范畴，其对女性的影响贯穿全生命周期且涉及多系统，其中心血管代谢相关的长期风险尤其引人关注。近年来的大型队列研究及Meta分析表明，PCOS女性发生主要不良心血管事件、心肌梗死及缺血性脑卒中的风险较非PCOS女性增加约30%~80%左右[3] [4]。另有研究表明，年轻的PCOS女性中，亚临床动脉粥样硬化相关标志物的异常表达更为显著[5]。Khomami等人[6]的研究表明，PCOS与妊娠期体重过度增加、妊娠期糖尿病、妊娠期高血压及子痫前期的发病风险增加存在显著关联。在普通人群中，妊娠期糖尿病、妊娠期高血压和子痫前期已被列为心血管危险因素[7]，上述研究结果进一步表明，PCOS与心血管疾病的发生存在密切的关联性。值得注意的是，既往研究虽一致表明，PCOS患者心血管疾病危险因素及相关标志物水平升高，但其与心血管事件的因果关系尚未完全明确。

迄今为止，国际上尚未形成统一的PCOS诊断标准，当前的主流诊断标准包括美国国立卫生研究院制定的NIH标准[8]，欧美生殖医学协会联合提出的鹿特丹标准[9]，雄激素过多协会制定的AES标准[10]，以及2011年我国卫生部发布的中国PCOS诊断标准专家共识[11]，其核心差异在于是否将高雄激素血症作为诊断的必备条件。目前，鹿特丹标准在临床中应用最为广泛且认可度最高，该标准所划分的四种表型(A-D)在代谢风险方面存在显著差异。最新研究突破传统表型分类，通过聚类分析识别出代谢主导型及生殖主导型等亚组[12]。其中，生殖亚型主要表现出体重指数(BMI)降低、空腹胰岛素水平下降，黄体生成素、性激素结合球蛋白、卵泡总数及抗苗勒管激素水平升高等特点，这一特征提示神经内分泌系统、促性腺激素通路及卵泡发育调控机制可能存在异常[13]。该发现提示，针对代谢亚组实施针对性生活方式干预，可实现心脏代谢风险的筛查及分层管理，进而有助于降低胰岛素抵抗发生风险。同时，该研究存在研究队列种族同质性的局限性，后续需在不同地理区域及种族人群中开展拓展研究，以进一步验证相

关结论。这为未来 PCOS 患者的精准风险评估及个体化干预提供了革命性框架。

PCOS 的发病机制尚未完全阐明，多数学者认为其由遗传因素和环境因素交互作用所引发。其中，胰岛素抵抗(IR)及其带来的神经内分泌和代谢紊乱是核心环节，病理生理改变的中心环节是卵巢的功能紊乱，同时伴随全身的代谢异常，从而造成 PCOS 的近期及远期并发症的发生[14]。一项孟德尔随机化研究显示，遗传决定的高雄激素血症是 PCOS 的上游风险因素，且通过影响胰岛素敏感性与脂肪分布，间接导致 2 型糖尿病的发病风险升高[15]。有动物研究证实，宫内高雄激素暴露可对胎儿下丘脑－垂体－性腺轴及代谢系统进行编程调控，进而导致子代出现 PCOS 相关表型[16]。

综上，PCOS 并非局限于生殖系统的疾病，除了下丘脑－垂体－卵巢轴的异常，还常伴随全身代谢稳态失衡，且存在远期心血管代谢风险。目前针对 PCOS 的管理策略大多集中于缓解患者当前的临床症状，如通过激素调节改善月经周期、借助促排卵药物助孕、采用抗雄激素药物控制多毛及痤疮等。普遍忽视了 PCOS 潜藏的长期健康风险，包括高血压、亚临床动脉粥样硬化、子宫内膜癌及阻塞性睡眠呼吸障碍等等。这可能导致临床诊疗错失对 PCOS 患者进行早期风险分层及实施预防措施的时机。因此对 PCOS 患者进行早期的风险识别及管理对其远期的健康尤为重要。

2. 多囊卵巢综合征相关心血管代谢危险因素及关联机制

(1) 胰岛素抵抗与高雄激素血症：核心因素

50%~70%的 BMI 正常的 PCOS 患者存在胰岛素抵抗(IR)，而合并肥胖的 PCOS 患者 IR 发生率更高[17]。PCOS 女性发生胰岛素抵抗(IR)的主要机制是丝氨酸磷酸化水平升高，从而引发胰岛素信号的结合功能障碍。此外，胰岛素受体和胰岛素受体底物-1 的酪氨酸磷酸化缺陷也会导致骨骼肌、脂肪细胞和卵巢的代谢紊乱。皮下脂肪细胞中葡萄糖转运蛋白 4 (GLUT4)减少、肝脏胰岛素清除受损、线粒体功能异常以及丝裂原活化蛋白激酶/细胞外信号调节激酶(MAPK-ERK)通路中的丝氨酸激酶刺激，都可能导致胰岛素抵抗，这些机制也会增加 2 型糖尿病的风险[18]。一项采取 BMI 匹配对照的研究表明，与健康人群相比，PCOS 患者的瘦素水平呈现升高趋势，并且不受 BMI 影响[19]。另有研究证明血清瘦素水平与空腹高胰岛素血症存在关联，进一步佐证了瘦素水平与 PCOS 患者胰岛素抵抗的相关性[20]。遗传学研究揭示，PCOS 和 2 型糖尿病的易感区域在 IRNS、THADA 和 HMGA 的基因座上呈现重叠变异的特点[21]。黄佳等人研究发现，PCOS 患者发生糖耐量异常的概率约为 11.7%，7.5%~10%的患者有 2 型糖尿病[22]。近年研究发现，PCOS 可增加非肥胖女性的糖尿病罹患风险[23]。并且，无论是瘦型还是肥胖的 PCOS 患者，其胰岛素敏感性均降低，且其胰岛素抵抗程度达到最大[24]。由于 IR 是肥胖的关键致病因素，合并中心性肥胖的 PCOS 患者会进一步加剧胰岛素相关的代谢并发症，从而显著增加心脑血管疾病的发生风险。另一方面，PCOS 患者存在雄激素分泌过早的特征，且该异常状态可在疾病早期阶段诱发胰岛素抵抗，构成 PCOS 代谢紊乱的重要病理生理基础[25]。已有研究证实，雄激素可损伤肌肉与脂肪组织中的胰岛素信号通路，进而降低细胞葡萄糖摄取效率，并引发胰岛素功能障碍[26]。在 PCOS 患者中，胰岛素水平与雄激素水平相关。研究表明，不同表型的 PCOS 患者的胰岛素抵抗程度存在差异，伴有高雄激素血症的无排卵型 PCOS 患者的胰岛素抵抗程度最高[27]。对具有高雄激素血症的女性予以抗雄激素干预，可降低其基础胰岛素水平及口服葡萄糖耐量试验刺激后的胰岛素水平[28]。雄激素过度分泌亦与胰岛功能障碍相关，可损害胰腺代谢，进而诱发高胰岛素血症，这直接增加 2 型糖尿病(T2DM)发病风险[29]。毫无疑问，胰岛素抵抗与高雄激素血症是连接内分泌异常与代谢改变的核心机制，在疾病发病进程中发挥关键作用。一些典型的胰岛素增敏药物，如二甲双胍，已被用于减少 PCOS 患者的胰岛素抵抗和高胰岛素血症。胰高血糖素样肽-1 受体激动剂(GLP-1RA)属于肠促胰岛素类激素，其核心作用机制为葡萄糖依赖性的胰岛素分泌刺激效应，同时可通过延缓胃排空和抑制胰高血糖素生成，协同发挥血糖调节作用[30]

[31]。近年来，GPL-1 受体激动剂和类似物在临床上被用于治疗肥胖型 PCOS [32]。有研究发现短期联合二甲双胍和艾塞那肽在改善 PCOS 患者糖脂代谢指标、恢复规律月经、提高体外受精妊娠率方面有一定作用[33] [34]。然而，GLP-1 受体激动剂的长期应用安全性仍有待全面评估，其不良反应以胃肠道症状为主，主要表现为恶心、呕吐和腹泻等。值得注意的是，GLP-1 类似物治疗肥胖症通常需要更高剂量，因此该类患者的胃肠道副反应风险可能相应增加[35]。有安全性分析表显示，低血糖、注射部位反应、胰腺炎、肿瘤及胆囊相关不良事件的发生风险总体较低[36]。在启动 GLP-1 受体激动剂治疗前，需对患者开展全面筛查，重点排除肝炎、胰腺癌、甲状腺癌等疾病的既往病史，同时排查可能影响进食的心理障碍。基于 PCOS 的高度异质性，临床中不同的 PCOS 患者对该药物的疗效反应存在个体差异。因此，需要开展更多针对性研究，筛选适用于 PCOS 的特异性药物。

(2) 血脂异常

PCOS 与一系列血症异常相关，表现为低密度脂蛋白、极低密度脂蛋白水平升高和高密度脂蛋白水平降低，以及血脂异常总体患病率的升高。PCOS 患者发生血脂异常的核心机制主要涉及胰岛素抵抗、极低密度脂蛋白过量生成、脂蛋白脂肪酶介导的脂解功能障碍，以及由 PI3KR1 基因过表达导致的胰岛素信号通路缺陷；此外，睾酮可通过双重途径诱导血脂异常，一是经雄激素受体介导加重胰岛素抵抗；二是上调参与高密度脂蛋白分解代谢的相关基因表达[37]。PCOS 患者中约 22%~70%存在血脂异常。王等人进行了一项前瞻性研究发现，与健康女性相比，PCOS 患者的高脂血症的发病率约增加 2.5~5 倍[38]。一项荟萃分析表明，与 BMI 匹配的同龄健康女性相比，患有 PCOS 的女性低密度脂蛋白水平高出 12.6 mg/dl，甘油三酯水平高出 26.4 mg/dl，高密度脂蛋白水平低 6.5 mg/dl [39]。高密度脂蛋白(HDL)可分为多个亚类，其中 HDL2 被证实是最具有抗动脉粥样硬化作用的亚型[40]。Conway 等人发现，即使是偏瘦的 PCOS 女性，其血清 HDL 和 HDL2 浓度也有所降低[41]。另外，有研究表明，小而致密低密度脂蛋白(LDL)导致动脉粥样硬化性的潜能更强，且与冠状动脉疾病密切相关[42]；PCOS 患者体内具有该种亚型比例升高或平均颗粒尺寸减小的特点[43] [44]，此变化与心血管疾病风险升高密切相关。远期心血管代谢疾病的防控是 PCOS 临床管理的核心重点。针对依靠饮食和生活方式干预难以达到理想疗效的 PCOS 患者，他汀类药物已逐步应用于临床治疗。但目前该类药物对 PCOS 患者胰岛素水平、血脂代谢影响尚存在争议，存在样本量小、证据质量低等局限，故尚未纳入标准化治疗。

(3) 肥胖/超重

体重异常在 PCOS 女性中极为普遍，其中超重(BMI 25~29.9 kg/m^2)和肥胖(BMI ≥ 30 kg/m^2)者比例高达 80%；且相较于年龄匹配的健康女性，PCOS 患者的体重指数(BMI)和腰臀比(WHR)均显著升高[45]。PCOS 患者的体重增加及肥胖的发生，与雄激素对脂肪细胞脂解功能的调控密切相关。一项荟萃分析指出，与 BMI 匹配的对照组相比，PCOS 患者表现出更为显著的腹型肥胖特征[46]。肥大的脂肪细胞更易发生炎症、凋亡、纤维化及释放非酯化的脂肪酸，进一步阻碍脂肪因子的合成和脂肪细胞分化，最终导致局部脂肪组织异常堆积[47]。Anagnostis P 等人[48]则提出，肥胖可损害子宫内膜容受性，导致胚胎着床困难，由此引发受孕延迟、流产率升高等不良生殖结局。AhmedB 等人[49]认为，脂肪组织的异常增多可诱导巨噬细胞聚集，并促使其向促炎表型极化。过量的游离脂肪酸以异位脂肪形式沉淀，产生脂毒性，引起全身炎症。全身慢性炎症状态可干扰胰岛素的生物学效应，破坏机体葡萄糖代谢稳态，进而诱发内分泌功能紊乱。在 PCOS 患者中，较高的 BMI 可使胰岛素抵抗程度加重约 15% [50]。但 BMI 与胰岛素抵抗的关联性在非 PCOS 对照组中同样存在[51]，这提示 BMI 并非导致 PCOS 患者心脏代谢危险因素发生率升高的唯一诱因。此外，采用匹配对照设计的观察性研究亦证实，无论 BMI 水平高低，PCOS 患者的心脏代谢危险因素发生率均显著高于对照组[52] [53]。针对 BMI 正常的 PCOS 患者开展的观察性研究也得出了相似结论[54] [55]。综合以上发现，单纯依靠体重及 BMI，不足以解释 PCOS 患者中心脏代谢危

险因素高发的现象。但从总体来说，肥胖会进一步加重 PCOS 患者的生殖和代谢问题是较为明确的，因此体重管理被提议作为 PCOS 患者的初始治疗策略[56]。

(4) 高血压

美国心脏病协会将高血压的诊断标准界定为收缩压大于或等于 130 mmHg，舒张压大于或等于 80 mmHg [57]。与健康女性相比，PCOS 患者的高血压患病率高出 24%，且其高血压发病呈现年轻化趋势，该关联在排除 BMI 影响后仍存在[58]。既往有研究明确 PCOS 与远期心血管疾病具有关联性[59]。合并高血压的 PCOS 患者较血压正常者呈现出更高的血脂、血糖及胰岛素水平[60]。多项研究证实，PCOS 患者的醛固酮水平显著高于年龄及 BMI 匹配的对照组，提示通过激活肾素－血管紧张素系统导致 PCOS 患者高血压的发生[61] [62]。另有研究认为，高雄激素可调控肾素－血管紧张素－醛固酮系统(RASS)，其功能异常会诱发内皮功能障碍，进而导致动脉血压升高[63]。此外，自主神经系统失衡引起的肾脏钠的重吸收增加、一氧化氮合成减少等病理改变，均与 PCOS 患者高血压的发生发展密切相关[58]。口服避孕药是 PCOS 的临床一线治疗用药，其药理作用可能诱发凝血酶原、白介素和其他炎性因子表达激活，从而增加 PCOS 患者心血管疾病的总体患病风险[64]。此外，有研究指出，螺内酯治疗或可逆转 PCOS 所引发的内皮功能障碍[65]。总之，并发高血压的 PCOS 患者可能更易发生恶性心血管疾病，如动脉粥样硬化和心肌梗死，这将增加发病率及死亡率。

(5) 肠道微生物群

有研究证实，PCOS 女性与对照组(包括 BMI 匹配人群)的肠道微生物组群落结构及组成具有显著差异[66]。这些研究揭示了 α-多样性改变、短链脂肪酸生成、胆汁酸代谢及内毒素信号传导的异常。机制研究表明，“肠道-PCOS 轴”可能通过代谢产物(如短链脂肪酸)调节胰岛素敏感性、低度炎症及雄激素代谢。在小鼠模型中，将健康对照小鼠的粪便微生物移植至 PCOS 小鼠可使其恢复发情周期。除代谢层面外，在普通人群中肠道菌群失调与重性抑郁障碍、焦虑及应激反应改变相关。鉴于 PCOS 女性抑郁和焦虑患病率亦较高[67]，阐明肠道微生物组在 PCOS 心理共病中的作用至关重要。总体而言，微生物组不仅提供了连接 PCOS 患者代谢、生殖与心理健康的合理因果通路，亦为潜在的治疗新靶点。

3. 瘦型 PCOS 患者的独特代谢特征

临床中 PCOS 可分为肥胖型和瘦型两大表型，其中瘦型 PCOS 的体重指数处于 18.5~23.9 kg/m^2 正常范围内[68]。传统观点认为 PCOS 的代谢异常多与肥胖相关，但近年研究证实，瘦型 PCOS 患者并非“代谢健康”人群，尽管其体重正常，仍存在体脂分布异常(如内脏脂肪堆积、腰臀比升高)、胰岛素抵抗(HOMA-IR 异常)及血脂谱改变等潜在代谢问题，且这类异常的发生机制与肥胖型 PCOS 存在显著差异[69] [70]。PCOS 患者摄入饱和脂肪，可激活介导炎症反应的分子通路，而该通路已被证实能够持续推动动脉粥样硬化的发生发展[71]。上述效应仅在瘦型 PCOS 女性中观察到，提示该效应的产生与超重状态无明显关联。与对照组相比，瘦型 PCOS 患者组在低剪切速率条件下的血液黏度呈现显著升高的特征[72]。内皮功能障碍、冠状动脉钙化及静脉血栓栓塞等，均被认为是增加 PCOS 患者心血管代谢不良事件发生风险的重要危险因素[73]。多次线性回归分析结果显示，总睾酮、天冬氨酸转氨酶(AST)及丙氨酸转氨酶(ALT)水平与瘦型 PCOS 的发生存在独立关联，这提示瘦型 PCOS 女性在疾病早期阶段即可能存在潜在肝脏病变风险[74]。因此，临床中一旦确诊 PCOS，应及时对患者开展肝脏相关指标的早期评估与针对性干预，从而有效预防或延缓肝脏病变的发生发展。由于瘦型 PCOS 的代谢紊乱表现相对隐匿，临床关注度不足，其心血管代谢特征的独特性及潜在风险尚未被完全阐明。

对于瘦型 PCOS 女性。努力保持体重稳定、摄入多种微量营养素至关重要[75]。早餐高热量摄入与晚餐低热量摄入可提升胰岛素敏感性指标，缓解高雄激素血症并增加排卵频率。Agar 等人[76]近期的一项

研究探讨了维生素 D 替代疗法对瘦型与肥胖型 PCOS 女性血清核因子 kβ (NF-kβ)水平的调控作用。结果表明，该疗法可通过下调血清 NF-kβ 水平，改善 PCOS 患者的生育力低下状态及代谢紊乱差异。此外，联合补充必需脂肪酸等关键营养素，可进一步放大均衡膳食模式的干预获益。

4. PCOS 心血管代谢疾病的管理

对于患有 PCOS 的女性，其管理工作着重于关注其特定的临床症状表现，并依据个人具体情况进行个性化安排。根据近期发布的国际多囊卵巢综合征(PCOS)指南[1]，患有 PCOS 的女性应常规对自身的心血管代谢风险进行评估。在初次就诊时，应评估空腹血糖或糖化血红蛋白水平，并且后续随访时，需根据是否存在糖尿病风险因素再次进行评估。伴随心血管代谢风险因素的个体，如肥胖、IGT 病史、妊娠期糖尿病、糖尿病家族史，应通过口服葡萄糖试验进行筛查[77]。此外，每年应监测血脂、血压和体重。

生活方式和体重的管理被认为是改善 PCOS 患者生殖健康的一线疗法。相关研究表明，通过运动实现 5%~10%的体重减轻，既能有益调节代谢风险，还能促进排卵功能改善[78]。对于患有严重肥胖(BMI ≥ 40)或中度肥胖(BMI ≥ 35)且伴有合并症的女性，可以考虑进行减重手术。一项针对 PCOS 女性的随机对照试验表明，与饮食管理组和药物治疗组(二甲双胍)相比，手术组(袖状胃切除术)的体重减轻幅度更大，且自然排卵率更高[79]。另有研究表明，实施胃切除术后的 PCOS 女性代谢异常也得到一定程度的改善[78]。此外，如血脂异常和阻塞性睡眠呼吸暂停等心血管代谢风险，仍需根据适当的治疗标准进行管理。未来研究应明确不同 PCOS 亚组对特定生活方式干预的获益差异，同时探索能改善生活方式、控制体重的最佳干预方案，从而在生殖、代谢及心理层面为 PCOS 患者带来综合获益。

5. 小结

与普通人群相比，PCOS 女性的远期心血管代谢风险显著升高。PCOS 年轻女性中，通常伴随心血管代谢的多种危险因素，这些都增加 PCOS 患者长期的健康风险及相关的经济和医疗成本。因此，临床在诊疗中，在解决排卵功能障碍和不孕等首要就诊问题的基础上，还应重视并加强远期并发症的预防和管理。另外，国内外对 PCOS 远期心血管代谢风险增加的机制尚未完全阐明，为提高我们对危险因素的认识，还需进行更多的长期人群研究。为有效缓解心血管代谢疾病日益加重的全球负担，完善风险分层尤为关键[80]。生活方式的调整已经证明有利于改善 PCOS 患者的生活功能和相关心血管代谢异常，减少其社会及心理压力。未来研究需明确何种 PCOS 亚型可从针对性生活方式干预中获益，并厘清生活方式相关疾病中 PCOS 依赖性因素与非依赖性因素的作用。同时，研究还应着力明确最优干预方案的剂量与形式，通过优化生活方式，进而改善患者的生殖功能及心血管代谢水平。

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