·指南与共识解读·

《超重/肥胖多囊卵巢综合征患者体重管理内分泌专家共识(2025 版)》解读*

蔡汭[1,2] 高睿[1,2] 秦朗[1,2]

(四川大学华西第二医院 1. 妇产科生殖医学中心; 2. 出生缺陷与相关妇儿疾病教育部重点实验室, 四川 成都 610041)

【摘要】 目前,超过半数的多囊卵巢综合征(PCOS)患者合并超重/肥胖,超重/肥胖不仅显著增加 PCOS 的发生风险,还可通过加重胰岛素抵抗和高雄激素状态,进一步恶化代谢异常、生殖功能障碍及心理健康问题,并提高 2 型糖尿病、心血管疾病及子宫内膜病变等远期并发症风险。有效的体重管理是治疗关键。然而,既往针对该人群的体重管理策略在国内外缺乏统一的专家共识,临床实践中存在评估标准不一、干预路径不清及缺乏个体化等问题。2025 年发布的《超重/肥胖多囊卵巢综合征患者体重管理内分泌专家共识》就超重/肥胖与 PCOS 的交互影响、超重/肥胖 PCOS 患者的临床特征、评估诊断、个体化治疗及管理目标进行了归纳及更新,为此类患者的诊断和体重管理提供了建议。本文通过对该共识进行详细解读,旨在提升超重/肥胖 PCOS 的规范化诊疗水平。

【关键词】 多囊卵巢综合征;肥胖;体重管理;专家共识;解读

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Interpretation of the *Endocrinology expert consensus on weight management in overweight/obese patients with polycystic ovary syndrome* (2025 *edition*)

CAI Rui[1,2], GAO Rui[1,2], QIN Lang[1,2]

(1. *Reproductive Medical Center, Department of Obstetrics and Gynecology, West China Second University Hospital, Sichuan University, Chengdu* 610041, *China*;
2. *Key Laboratory of Birth Defects and Related of Women and Children of Ministry of Education, West China Second University Hospital, Sichuan University, Chengdu*, 610041, *China*)

【Abstract】 Currently, more than half of patients with polycystic ovary syndrome (PCOS) are overweight or obese. Overweight and obesity not only significantly increase the risk of developing PCOS, but also exacerbate insulin resistance and hyperandrogenism, thereby worsening metabolic abnormalities, reproductive dysfunction, and psychological health problems, and increasing the long-term risk of complications such as type 2 diabetes mellitus, cardiovascular disease, and endometrial disorders. Effective weight management is therefore a cornerstone of treatment for this population. However, until recently, there has been a lack of systematic and unified expert consensus on weight management strategies for overweight/obese patients with PCOS, resulting in heterogeneity in clinical assessment, unclear intervention pathways, and insufficient individualization in practice. In 2025, the *Endocrine Expert Consensus on Weight Management in Overweight/Obese Patients with Polycystic Ovary Syndrome* was released, providing updated and comprehensive recommendations on the interaction between overweight/obesity and PCOS, the clinical characteristics of overweight/obese PCOS

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通信作者:秦朗,主任医师,教授,博士研究生导师,四川大学华西第二医院生殖医学大平台副主任。从事专业和研究方向:生殖医学。担任生殖领域多学会委员,作为课题负责人主持或参与国家级、省部级科研课题多项,累计作为第一作者或通信作者发表 SCI 论文及核心期刊 50 余篇;多本国内和国际杂志的编委、审稿人,如 *International Journal of Obstetrics and Gynecology*, *Chinese Medical Journal*,《西部医学》《中国计划生育和妇产科》《西南医科大学学报》等。长期关注生殖医学尤其是生殖免疫学的研究,主要从事生殖医学健康、人工智能在生殖医学的应用、女性和男性的不孕不育治疗、辅助生育技术、各种规范化促排卵治疗、复发性流产、抗磷脂综合征等疾病的基础及临床研究。

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across the life course, assessment and diagnostic approaches, individualized treatment strategies, and short- and long-term management goals. This article provides a detailed interpretation of the consensus, aiming to promote standardized clinical management and improve overall outcomes in overweight/obese patients with PCOS.

【Key words】 Polycystic ovary syndrome; Obesity; Weight management; Expert consensus; Interpretation

多囊卵巢综合征(Polycystic ovary syndrome, PCOS)是一种具体病因不明,好发于育龄期女性的生殖内分泌代谢疾病,以月经紊乱、高雄激素血症和/或高雄激素临床表现、超声提示多囊卵巢形态为主要特征。PCOS 患者还常伴有胰岛素抵抗(Insulin resistance, IR)和各种代谢综合征,远期发生 2 型糖尿病和心血管疾病的风险增加。全球育龄女性中,PCOS 的患病率大约在 10%~13%[1]。在中国,这一比例约为 7.8%[2],而在超重/肥胖的女性中,患病率可达到 28.3%[3]。这些流行病学数据提示超重/肥胖女性是罹患 PCOS 的高危人群。此外,超半数 PCOS 患者合并超重/肥胖,且比例逐年上升[1]。而合并超重/肥胖的 PCOS 患者较正常体重患者更容易发生代谢紊乱、生殖异常、心理问题以及远期并发症,如何有效管理体重成为改善这类患者身心健康的关键[4]。鉴于既往指南对超重/肥胖 PCOS 患者的诊治缺乏系统性指导,中华医学会内分泌学分会于 2025 年制定了翔实的《超重/肥胖多囊卵巢综合征患者体重管理内分泌专家共识》(以下简称本共识)[5]。本共识结合了国内外相关指南、最新研究成果以及专家的临床经验,针对临床实践中可能遇到的难点,提出了相应的指导和建议,帮助临床医生为超重/肥胖 PCOS 患者制定个性化的体重管理方案。而本文基于国内外的相关文献,对本共识的核心内容进行了分析和解读,并更新补充了相关内容,旨在为超重/肥胖 PCOS 患者的临床诊疗提供有力的参考。

1 PCOS 与肥胖的共病机制及交互影响

虽然肥胖既不是 PCOS 的必要条件,也不是充分条件,但现有流行病学、遗传学及机制研究一致表明,肥胖与 PCOS 之间存在密切的共病关系,并在疾病发生发展中形成相互促进的恶性循环[6]。基于高质量研究的系统评价显示,无论采用何种诊断标准,人群平均体重指数(Body Mass Index, BMI)或肥胖率均与 PCOS 患病率呈显著正相关[7],且该关联在青春期女性中尤为突出[8]。孟德尔随机化研究进一步支持儿童及青春期肥胖可显著增加成年期 PCOS 发生风险[9]。此外,PCOS 与成人及儿童 BMI 共享多种易感位点,涉及类固醇生成、卵泡发育及脂质代谢等关键通路,其中 FTO 基因多态性被认为是连接肥胖与 PCOS的重要遗传纽带[10-12]。机制上,肥胖通过促进腹型脂肪堆积和胰岛素抵抗,加重高胰岛素血症和卵巢高雄激素状态,进而导致排卵障碍和代谢异常;而 PCOS 相关的高雄激素水平和心理行为因素又可反过来抑制脂肪分解、促进脂肪蓄积,进一步加重肥胖,形成持续放大的病理性因果循环[13-15]。因此,本共识强调应将肥胖及相关代谢异常视为 PCOS 重要的病理组成部分,在疾病的早期识别、风险分层和综合管理中给予充分重视。

2 超重/肥胖 PCOS 患者临床特征及风险

本共识强调,PCOS 的临床与代谢影响贯穿女性整个生殖生命周期,而超重/肥胖是加重不同阶段 PCOS表型和远期不良结局的关键因素。青春期是 PCOS 发病高峰期[16],生理性胰岛素抵抗会与PCOS相关代谢异常叠加[17-18],同时肥胖可进一步加重高雄激素血症、月经紊乱及胰岛素抵抗,升高三酰甘油、低密度脂蛋白胆固醇,高敏 C 反应蛋白等心血管疾病风险标志物,显著增加 2 型糖尿病、阻塞性睡眠呼吸暂停等疾病风险[19-21]。此外,青春期女性因身体发育和社会化需求面临心理适应问题。肥胖 PCOS 女孩抑郁风险更高(为对照组 2.26 倍),且抑郁指数与 BMI 正相关[22]。育龄期肥胖 PCOS 患者表现为更为严重广泛的临床表型和代谢紊乱[23-26],是 2 型糖尿病、高血压、血脂异常及代谢相关脂肪性肝病的高危人群[27-30]。同时肥胖可加剧生殖内分泌紊乱,降低自然受孕率和辅助生殖活产率,并增加妊娠并发症发生风险[31-34]。进入围绝经期及绝经后,PCOS 的典型生殖内分泌特征逐渐减弱,但体重增加和腹型肥胖普遍存在,研究发现 PCOS 女性仍较非 PCOS 人群更易肥胖,但两者间代谢风险差异缩小,这可能与对照组年龄相关代谢风险累积有关[35]。而在校正 BMI 后,仅高密度脂蛋白胆固醇这一指标仍有差异,可见肥胖仍是决定心血管与代谢风险的核心因素[36];但目前尚无明确证据表明绝经后 PCOS 患者心血管疾病及女性生殖系统恶性肿瘤的发生风险增加[1,37-38]。总体而言,超重/肥胖在 PCOS 各阶段均与更严重的代谢负担和不良远期结局密切相关,体重管理应作为 PCOS 全生命周期管理的核心策略。

3 超重/肥胖 PCOS 的诊断筛查和风险评估

3.1 PCOS 的诊断标准 根据 2018 年中国 PCOS 诊断标准[39],育龄期及围绝经期女性的 PCOS 诊断遵循

“疑似—确诊”流程：首先，根据月经稀发/闭经和高雄激素表现或超声下多囊卵巢形态（Polycystic ovary morphology，PCOM）可拟诊为疑似 PCOS；确诊需排除其他引起高雄激素或排卵障碍的疾病，如皮质醇增多症、先天性肾上腺皮质增生、高催乳素血症、甲状腺疾病、雄激素分泌性肿瘤、功能性下丘脑性闭经、早发性卵巢功能不全等。此路径强调 PCOS 作为排他性诊断，有助于避免误诊，特别是在围绝经期症状不典型时。2023 年国际循证指南指出抗苗勒管激素（anti-Müllerian Hormone，AMH）可作为 PCOM 替代指标，但因受年龄、BMI 等因素影响，尚未制定统一标准[1]，因此本共识尚未将 AMH 列为 PCOM 的定义指标。对于青春期女性，依据 2018 年指南，诊断 PCOS 需同时满足初潮后 2 年内的月经稀发/闭经、高雄激素表现和超声 PCOM，且需排除其他疾病。而国际指南强调 AMH 和超声不应用于青春期 PCOS 诊断[40]。

3.2 超重/肥胖的诊断标准 育龄期和绝经期女性参照中国《成人体重判定》标准（2013 版）[41]及《肥胖患者的长期体重管理及药物临床应用指南（2024 版）》[42]。其中，成人女性超重定义为 BMI 24.0～27.9 kg/m^2，肥胖为 BMI≥28.0 kg/m^2；中心型肥胖可依据腰围（前期 80～85 cm，确诊≥85 cm）、腰臀比≥0.85、腰高比≥0.50 或内脏脂肪面积≥80 cm^2 进行判定，体脂率≥30% 亦可作为肥胖诊断标准。青春期 12～18 岁女孩则采用年龄别 BMI 界值判定超重和肥胖，如 12 岁超重/肥胖界值分别为 21.5/23.9 kg/m^2，随年龄逐年上调至 18 岁 24.0/28.0 kg/m^2[43]。

3.3 心血管疾病与代谢紊乱风险评估 在 PCOS 诊断确立后，应立即启动系统代谢与心血管风险评估，并实施分层动态随访。糖代谢与 IR 评估为首要环节，推荐采用 5 点法口服葡萄糖耐量试验（oral glucose tolerance test，OGTT）（0、30、60、120、180 min）同步胰岛素释放试验（必要时可简化为 0、120 min 2 点法），以识别早期餐后高血糖及 IR 模式；根据检查结果、用药及妊娠需求，每 3 个月至 3 年重复评估。脂代谢评估要求同步筛查总胆固醇、甘油三酯、高密度脂蛋白胆固醇和低密度脂蛋白胆固醇，初期每 1～3 个月复查，达标后延长至每 3～6 个月。其他心血管危险因素筛查包括血压、血尿酸、脂肪肝影像学检查，肥胖者需加做睡眠呼吸监测以早期发现阻塞性睡眠呼吸暂停（初筛正常但体重未达标者每年重复 1 次），随访频率同血脂管理。对于围妊娠期患者[44]，应在孕前、孕早期及孕 24～28 周全程进行 OGTT 与胰岛素释放试验，并动态监测血压血脂，将代谢风险管控前移。

4 超重/肥胖 PCOS 患者的个体化治疗策略

4.1 治疗管理手段概述

本共识明确超重/肥胖 PCOS 的治疗以减重、改善 IR 为核心，联合调经、降低雄激素及促排卵等综合干预。目前，生活方式干预是治疗的基石，若疗效不佳，可进阶至药物或手术治疗。

4.1.1 生活方式 减重 5%～15%是首要目标，需通过腰围（<80 cm）、腰臀比（<0.8）等核心指标管理中心性肥胖，制定个体化且可持续的体重管理方案[45]。饮食方面，应在适度限制能量、优化营养结构的基础上，减少碳水化合物与饱和脂肪摄入，增加膳食纤维与健康脂肪，优先推荐地中海饮食或得舒（Dietary approaches to stop hypertension，DASH）饮食模式，关键在于实现能量负平衡并确保长期依从性[46-48]。运动干预需结合有氧与抗阻训练，基于患者偏好与减重目标进行个体化设计，同时注重关节保护。此外，应通过健康教育提升疾病认知，保障优质睡眠，疏导负面情绪，避免使用可能加重 PCOS 症状的药物，并提供全面的家庭支持系统，以帮助患者平稳度过青春期至成年期的关键过渡阶段。

4.1.2 纠正代谢的药物和手术 二甲双胍作为经典胰岛素增敏剂，通过改善外周胰岛素敏感性、抑制肝糖异生并间接降低高胰岛素驱动的雄激素分泌，是合并胰岛素抵抗、糖耐量异常或糖尿病患者的一线用药，推荐小剂量起始，常见胃肠道反应，长期使用需警惕维生素 B12 缺乏[1]；奥利司他通过抑制胃肠道脂肪酶减少脂肪吸收，适用于脂肪摄入较高、难以调整饮食结构的患者，注意服药期间脂肪泻、腹泻及脂溶性维生素缺乏风险[49]；胰升糖素样肽 1 受体激动剂（Glucagon like peptide-1 receptor agonist，GLP-1RA）通过中枢抑制食欲并改善胰岛素分泌，可显著降低体重、腰围及胰岛素抵抗，推荐用于合并糖尿病或心血管危险因素者，常见胃肠道不良反应，甲状腺髓样癌或多发性内分泌腺瘤病 2 型患者禁用[49]；钠葡萄糖协同转运蛋白 2（Sodium dependent glucose transporters 2，SGLT-2）抑制剂通过促进尿糖排泄发挥减重及改善胰岛素抵抗作用，适用于合并糖代谢异常或高尿酸血症者，需注意泌尿生殖道感染风险并监测肾功能[50]；小檗碱作为二甲双胍不耐受者的替代选择，可改善内脏脂肪分布和胰岛素敏感性，但证据有限，仅作为补充治疗[1]；阿卡波糖通过抑制 α-糖苷酶减少餐后血糖波动，适用于以餐后高血糖为主的糖耐量异常或 2 型糖尿病患者，不良反应为胃肠道胀气[51]；噻唑烷二酮类（Thiazolidinediones，TZDs）虽不减重，但可改善胰岛素抵抗和高雄激素状态，适用于合并血脂异常或脂肪肝的特定人群，需警惕水钠潴留

和心衰风险[52]；肌醇作为胰岛素信号第二信使，对部分代谢指标和排卵功能有轻度改善，安全性良好，但证据有限，仅可作为辅助选择，不能替代生活方式干预或二甲双胍[53-55]。若上述单药疗效不足，可在二甲双胍基础上联合 GLP-1RA、SGLT-2 抑制剂或吡格列酮，以增强减重和代谢改善效果[56-58]。对于重度肥胖、合并明显代谢紊乱或生育需求且药物治疗失败者，代谢手术（以袖状胃切除为主）可显著改善体重、代谢及生殖结局[59-60]。BMI≥35 kg/m^2 者无论是否合并代谢性疾病均可考虑手术[61]，若合并 2 型糖尿病、高血压等代谢性疾病，BMI≥32.5 kg/m^2 即可选择手术[62]。

4.1.3　合并代谢紊乱的处理　本共识强调对于合并代谢并发症的超重/肥胖 PCOS 患者，在强调生活方式及纠正代谢紊乱的一线治疗基础上，应根据具体危险因素选择相应专科规范化管理。高脂血症者依甘油三酯或胆固醇类型选择贝特类或他汀类药物（他汀虽可降低雄激素，但不推荐用于无血脂紊乱及心血管疾病高危因素者）；高尿酸血症者先保证足量饮水，无效后碱化尿液（推荐尿 pH 值维持在 6.2～6.9），仍未达标则启动降尿酸药物；高血压患者需心血管风险分层，特定血压阈值合并高危因素者首选钙通道阻滞剂；代谢相关脂肪性肝病者优选二甲双胍、吡格列酮、SGLT-2 抑制剂或 GLP-1RA 等具有肝脏获益的药物，合并肝功能损伤可加用保肝药，相关药物选择与疗程均参照最新中国指南[63-65]。必要时联合专科医师或全科医师规范治疗。

4.1.4　复方口服避孕药的应用　对于月经稀发（每年＜4 次）的超重/肥胖 PCOS 患者，需药物干预以保护子宫内膜并调整周期，可予以孕激素周期撤退、雌孕激素序贯或复方口服避孕药（combined oral contraceptive pill，COCP）对症治疗，常规疗程为 3～6 个月多毛症治疗至少需 6 个月以上。其中指南首选含低剂量雌激素（20～30 μg）的 COCP，而炔雌醇环丙孕酮片（达英-35）因增加体重、血栓及肝损伤风险，不推荐作为一线用药；仅当痤疮或多毛症状显著时，可在充分沟通后短期使用[1]。低剂量雌激素搭配第二代孕激素（如左炔诺孕酮）的 COCP 致血栓风险较低[66]。COCP 可能加重代谢风险，联合二甲双胍在改善 IR、降雄及升高性激素结合球蛋白方面优于单药，可抵消其代谢副作用[67-68]。COCP 禁忌者可单用二甲双胍，而高代谢风险人群（BMI＞30 kg/m^2 或糖耐量异常）优选联合治疗[1]。用药前应行血栓风险分层评估，并建立用药后 3 个月复查糖脂代谢、6 个月评估肝功能的监测体系。

4.2　不同时期的治疗方案策略　本共识基于现有证据，针对不同生命周期的超重/肥胖 PCOS 女性提出分期管理策略，强调不同时期、不同治疗策略。青春期治疗以生活方式干预为核心，强调均衡限能饮食和规律运动。饮食上按同年龄正常体重儿童的能量需求适当减少摄入，三大营养素供能比为碳水化合物 50%～60%、脂肪 20%～30%、蛋白质 15%～25%，严格避免含糖饮料、高脂等高能量密度食品[69]；运动干预方面建议每周至少进行 3 次、每次 60 min 的中高强度运动（含力量训练）[70]；药物选择应高度谨慎，除二甲双胍（1 500～2 000 mg/d）和 COCP 外，不推荐其他代谢调节药物[71]。育龄期仍以强化生活方式干预为基础，必要时联合药物或代谢手术以控制体重和代谢异常。饮食上建议每日减少 500～750 kcal 或总热量 30%，维持 1 200～1 500 kcal/d 摄入，推荐 DASH 饮食模式[1,72]；运动方面建议减重需每周≥250 min 中等强度或≥150 min 高强度有氧运动，每周 2 天力量训练[73]；二甲双胍（最大2 500 mg/d）依然是合并胰岛素抵抗或糖代谢异常者的首选药物[74]。相较青春期，纠正代谢紊乱的药物及手术手段应用更为广泛。围妊娠期管理强调用药安全性和妊娠时机控制，孕前应系统评估并优化体重和代谢状态。妊娠期尽量选择 FDA A/B 类药物，确有指征时优先选择安全性证据相对充分的药物。BMI＞30 kg/m^2 者需补充高剂量叶酸，并充分告知超重对妊娠结局的不利影响[1]。二甲双胍属妊娠 B 类药，可通过胎盘但无明确致畸证据。本指南指出，若未合并 2 型糖尿病或妊娠期糖尿病，建议确诊妊娠后停用二甲双胍[75]。但实际临床应用中，妊娠期使用二甲双胍并未见明确母胎不良事件报告，属常规应用。代谢手术后应待体重稳定后再计划妊娠[76]；绝经期治疗重点由生殖转向心血管和代谢风险防控，应长期坚持健康饮食和安全运动，药物及术式选择可参照育龄期方案，但需更加审慎评估血栓及其他不良反应风险[77]。总体而言，超重/肥胖 PCOS 的治疗应贯穿全生命周期，依据不同阶段的主要健康目标实施个体化管理。

5　超重/肥胖 PCOS 患者的管理目标

本共识将超重/肥胖 PCOS 患者的管理目标分为近期和远期两个层面。近期目标（3～6 个月）以减重和纠正代谢紊乱为核心：超重患者减重 5%～10%、肥胖患者减重 10%～15%；空腹血糖≤6.1 mmol/L、餐后 2 小时血糖≤7.8 mmol/L、HbA1c＜6.5%；如伴有高血压，血压＜130/80 mmHg；使血脂、尿酸、肝功能及甲状腺功能维持在正常范围；合并代谢异常和月经紊乱者，应在减重纠代谢的同时同步调节月经周期并

降低雄激素水平。围妊娠期女性在受孕或辅助生殖助孕前应达到上述代谢控制目标，合并甲状腺功能减退者需将促甲状腺激素控制在≤2.5 mU/L。远期目标侧重长期体重管理和并发症防控：超重者 BMI≤23 kg/m^2，肥胖者≤25 kg/m^2，体脂率＜30%；系统预防 IR、糖尿病、心血管疾病及代谢相关脂肪性肝病，并定期监测子宫内膜，关注心理健康问题。

6 超重/肥胖与 PCOS 的诊疗研究进展

本文在解读现有共识诊疗建议的基础上，进一步梳理了肥胖 PCOS 患者诊疗新策略的最新研究进展。AMH 虽未被指南纳入 PCOS 诊断标准，但研究提示其在特定 BMI 范围内可反映减重疗效，唯在重度肥胖（BMI≥35 kg/m^2）人群中价值受限[78]。还有研究发现，中国肥胖伴高胰岛素血症 PCOS 患者血清及卵泡液中果糖水平显著升高，联合睾酮检测可提高诊断性能，提示其成为诊断标志物的潜能。此外，脂肪因子和肠道通透性相关蛋白的水平也可能作为评估 PCOS 代谢紊乱的辅助指标[79]。肥胖 PCOS 患者的肠道菌群测序多项研究揭示，菌群失调可能是高雄激素血症及胰岛素抵抗的关键机制，且 DHEA 硫酸盐等粪便代谢物具有辅助诊断价值[80-81]。因此有望通过益生菌/合生元来改善 PCOS 患者的代谢和激素紊乱[82-83]。

7 结语

本共识的发布标志着我国超重/肥胖 PCOS 体重管理进入循证与个体化并行的新阶段，强调根据患者年龄、生育需求及代谢表型，制定覆盖青春期至绝经期的全生命周期管理方案。共识系统梳理了超重/肥胖 PCOS 的评估、诊断、治疗与随访路径，填补了国内相关领域的规范空白，为临床提供了可操作、可推广的管理框架，并明确以生活方式干预为基础、药物治疗为辅助、代谢手术为补充的分层策略。临床实践中，应依据共识动态监测代谢与生殖指标，合理选择饮食、运动、药物及手术时机，实现体重下降 5%～15%的关键目标，从而改善胰岛素抵抗和高雄激素状态，优化生殖结局，并降低远期代谢及肿瘤相关风险，全面提升超重/肥胖 PCOS 患者的长期预后与生活质量。

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