Advances in Clinical Medicine 临床医学进展, 2026, 16(3), 200-208
Published Online March 2026 in Hans. https://www.hanspub.org/journal/acm
https://doi.org/10.12677/acm.2026.163780

Hans汉斯

胆囊切除术后胆道重构：结构、功能与临床结局研究进展

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收稿日期：2026年1月27日；录用日期：2026年2月22日；发布日期：2026年2月28日

摘　要

腹腔镜胆囊切除术(laparoscopic cholecystectomy, LC)已成为治疗胆囊良性疾病的主流方式。随着手术量的持续增加，大量“无胆囊人群”的长期结局逐渐受到关注。切除胆囊并非单纯的器官减法，而是牵动了解剖结构、流体动力学、神经－内分泌调控以及全身代谢的一系列连锁反应。胆囊切除术后，胆道系统由“储存－脉冲式排放”转为更连续的单出口排放模式；胆囊作为“压力缓冲器”与胆汁浓缩、调节器的缺失，可能改变胆道压力动力学、Oddi括约肌－胆道协调、胆汁酸肠肝循环与肠道生态，从而影响临床症状与远期结局，部分患者在适应过程中出现胆囊切除术后综合征、胆总管结石、胆汁酸性腹泻及非酒精性脂肪性肝病等远期问题。本综述围绕“解剖－功能－症状”三条主线展开讨论，总结了胆囊切除术后胆道结构重构与机体代谢改变的相关证据，梳理了其与临床结局之间的关联，同时，我们还讨论了胆囊切除术后胆管结石复发的主要机制。

关键词

胆囊切除术，胆道重构，Oddi括约肌，胆汁酸，胆囊切除术后综合征，胆总管结石

Advances in Research on Post-Cholecystectomy Biliary Remodeling: Structural, Functional Alterations and Clinical Outcomes

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文章引用: 夏静娴, 李俊. 胆囊切除术后胆道重构: 结构、功能与临床结局研究进展[J]. 临床医学进展, 2026, 16(3): 200-208. DOI: 10.12677/acm.2026.163780

Received: January 27, 2026; accepted: February 22, 2026; published: February 28, 2026

Abstract

Laparoscopic cholecystectomy (LC) has become the standard treatment for benign gallbladder diseases. With the continuously increasing surgical volume, growing attention has been directed toward the long-term outcomes of the expanding post-cholecystectomy population. Cholecystectomy is not merely "organ subtraction"; rather, it can trigger a cascade of changes involving anatomy, fluid dynamics, neuroendocrine regulation, and systemic metabolism. After gallbladder removal, the biliary system shifts from a "storage-pulsatile discharge" pattern to a more continuous, single-outlet drainage mode. Loss of the gallbladder as a "pressure buffer" and as a concentrator and regulator of bile may alter biliary pressure dynamics, sphincter of Oddi-biliary coordination, enterohepatic circulation of bile acids, and the gut ecosystem, thereby influencing clinical manifestations and long-term outcomes. During this adaptive process, a subset of patients may develop late complications such as post-cholecystectomy syndrome (PCS), recurrent choledocholithiasis (common bile duct stones, CBDS, CBD stones), bile acid diarrhea (BAD), and nonalcoholic fatty liver disease (NAFLD). This review is organized around three interrelated themes—anatomy-function-symptoms—and summarizes current evidence regarding post-cholecystectomy biliary structural remodeling and metabolic alterations, delineates their associations with clinical outcomes, and further discusses the principal mechanisms underlying recurrence of bile duct stones after cholecystectomy.

Keywords

Cholecystectomy, Biliary Remodeling, Sphincter of Oddi, Bile Acids, Post-Cholecystectomy Syndrome, Common Bile Duct Stones

1. 引言

腹腔镜胆囊切除术(laparoscopic cholecystectomy, LC)是胆囊结石及其他良性胆囊疾病的标准治疗之一，已成为最常见的腹部外科手术之一[1]。随着微创技术普及及适应证拓展，胆囊切除术量在全球范围内持续增加；我国相关专家共识亦明确 LC 在良性胆囊疾病中的重要地位并被广泛采用[2] [3]。但胆囊切除并不意味着症状与风险终止：部分患者术后出现持续或新发腹痛、消化不良、腹泻、反流等表现，统称为胆囊切除术后综合征(post-cholecystectomy syndrome, PCS)，其报道发生率约 5%~47%，反映了定义与随访窗口差异带来的异质性[4]-[6]。此外，胆管损伤(post-cholecystectomy bile duct injury, BDI)虽发生率较低，但指出 LC 后胆管损伤发生率约 0.4%~1.5%，可导致胆漏、脓毒症及远期胆管炎、狭窄等不良结局[7]。因此，术后胆道系统可能存在适应性重塑与病理性改变并存的复杂过程。

基于此，本文围绕胆道重构展开讨论。本文所称胆道重构并非指胆肠吻合等外科重建术式，而是指胆囊切除后胆道系统在结构、功能与微环境层面的持续性变化：结构层面包括肝外胆道生理性扩张与狭窄、损伤等病理性分支，Park 等人的研究提示术后胆总管可轻度增宽，且在无症状、肝功能正常背景下扩张至 10 mm 以内可视作生理范围[8]；功能层面涉及 Oddi 括约肌相关障碍(sphincter of Oddi dysfunction, SOD)与胆汁排空失调，胆囊切除后胆道样疼痛可见于部分患者，其中一部分符合 SOD 诊断谱系，且内

镜治疗并非对所有分型患者都有效[9] [10]；微环境层面则包括胆汁酸池与肠道菌群–黏膜屏障互作的改变，Ang 等人的研究也提示胆囊切除与肠道菌群改变及 PCS、腹泻等结局存在关联，但现有研究对具体结构与功能改变仍缺乏一致结论[11]。从临床结局看，胆道重构的外显表型既包括 PCS 与胆道样疼痛，也包括胆汁酸相关腹泻(bile acid diarrhea, BAD)及与功能性肠病的交织。综述与指南也强调，胆囊切除后可发生 BAD，且在腹泻型肠易激综合征(irritable bowel syndrome with diarrhea, IBS-D)人群中并不少见，临床上要避免长期将该病归类于功能性腹泻，并尽量采用 SeHCAT 或血清 C4 等手段进行阳性诊断与分层管理[12] [13]。在更长期结局层面，Zhao 等人本研究对 413,472 名未患 IBS、炎症性肠病、癌症或常见良性消化道疾病的参与者进行前瞻性分析，发现胆囊切除术与较高的 IBS 风险相关，尤其是伴有腹泻的 IBS。提示“胆汁酸–动力学–菌群”重构可能与功能性肠病发生有关[14]。因此，本文主要围绕“结构–功能–临床结局”的视角，对胆囊切除术后胆道重构相关证据进行系统梳理，明确可接受的生理适应与可干预病理过程的边界，为临床决策与随访策略提供依据。

2. 解剖重构：胆道结构与几何形态改变

胆道系统由肝内外胆管、胆囊与胆囊管、胆总管(CBD)远端及壶腹括约肌装置共同构成连续管路网络，负责胆汁的汇集、储存、浓缩与按需排空[15]。胆汁经肝内胆管汇入左右肝管并形成肝总管(CHD)，与胆囊管汇合后形成胆总管(CBD)，远端在胰头区开口于十二指肠乳头，多数情况下与主胰管汇合形成肝胰壶腹，由 Oddi 括约肌(sphincter of Oddi, SO)调控胆汁、胰液排入肠腔内并减少反流[16] [17]。生理状态下，空腹期 SO 张力较高，胆汁更易分流入胆囊储存并被浓缩；进食后在胆囊收缩素(CCK)等作用下胆囊收缩、SO 松弛，胆汁经 CBD 进入十二指肠参与消化，分泌素等亦可促进胆汁流动并维持肠腔碱化环境[18] [19]。因此，胆道系统不仅是解剖通道，更是受神经体液调控的动力学系统。

胆囊切除术后，胆道改变主要体现为胆汁动力学重塑与肝外胆管形态学适应：胆囊的储库、压力缓冲功能消失后，胆汁更倾向持续经胆道向十二指肠排空，胆道压力–流量调控模式随之改变。Luman [20]和 Tanaka [21]的经典测压研究提示，术后早期 SO 可出现短暂张力增高或节律紊乱，部分患者因此出现一过性胆道样疼痛；随后 SO 逐渐适应性调整，张力下调、开放时间延长，与上游胆总管的代偿性扩张共同维持胆汁外流[20] [21]。影像学研究亦显示，胆囊切除后 CBD 可出现轻度、渐进性增宽，且个体差异明显；在无症状且肝功、胆汁淤积指标正常的背景下，这种轻度扩张多被视为生理性适应[8] [22]。在结局关联方面，系统综述提示胆管显著扩张、狭窄以及胆道几何形态异常等与复发结石风险升高相关，提示胆道通畅性与几何形态改变可能通过胆汁淤积机制参与结石再发[23]。

对于 CBD 生理性扩张上限的临床判定，核心不仅仅在单一管径方面，而在综合因素的分层判断。AJR 专家组建议按年龄与术后状态采用可操作的判定框架：一般人群 CBD > 6 mm、≥60 岁 > 8 mm、既往胆囊切除者>10 mm 可定义为胆道扩张；但在缺乏症状或肝功能异常的情况时，单纯扩张通常不代表梗阻，需结合临床与实验室综合判断[24]。因此，提示需要进一步评估的“危险信号”包括：胆道样腹痛、发热、黄疸等症状；胆汁淤积型肝功能异常如胆红素、ALP、GGT 升高；以及影像学可疑征象如肝内胆管扩张、狭窄、占位、明确结石等[24] [25]。如果没有上述危险信号并且扩张可由术后状态或高龄来解释，多数情况下可选择观察或影像随访。

几何形态的改变：角度与曲率

与口径扩张相比，胆道的几何形态更直接决定胆汁流速、滞留与结晶环境。术后在胆总管逐渐扩张、胆管壁纤维化、炎症修复、以及远端胆总管受胰腺段固定等因素共同作用下，可出现胆总管走行迂曲、局部折角加深或呈“S 形、折线形”等形态学改变；这些改变被认为与胆汁淤积、细菌定植和十二指肠–

胆道反流形成协同，从而增加了复发结石与反复胆管炎的风险。临床研究最常用的指标是远端 CBD“最小角度”，通常在 ERCP 造影或 MRCP 上，以近端 CBD 长轴与远端 CBD 长轴(或在最大折点两段切线)夹角表示，既往研究常以 145˚或 135˚作为低角度的判定阈值。常见的测量手段与 MRCP、ERCP 等，MRCP 更像是在“自然状态”下把胆道当作一条立体管路来观察：它主要依赖重 T2 序列让胆汁呈高信号显示，因此不需要插管、也不依赖造影剂加压，整体走行更接近患者真实的胆道形态，更适合做形态分型(直型、S 型、折线型)与整体迂曲程度评估，但也容易受到呼吸运动伪影或投影叠加等因素的影响。ERCP 则更像在透视下给胆管显影：它通过内镜插管后注入造影剂，在 X 线透视下显示胆胰管腔，清晰度高且可同步治疗，但对几何测量天然更“情境依赖”，但 ERCP 时插管深度、注射速度、压力以及镇静相关的括约肌松弛，都可能改变管腔充盈与张力，从而使 ERCP 下的“直径、形态”与非侵入影像存在系统差异[26] [27]。因此，我们临床上需要根据患者的实际情况进行考量，选择对患者最佳的检查临床测量手段。Ryu 等人的研究提示：CBD 角度 ≤145˚与结石复发显著相关，提示“锐角化”可作为可量化的高危局部因素之一[28]。Keizman、Seo 等人的回顾性研究也观察到在成功取石后，存在胆总管急性折角者复发风险明显升高[29] [30]。在“已行胆囊切除”的人群中，Yoo 等进一步证实远端 CBD 角度 ＜145˚是复发的独立预测因子之一[31]。然而，单一角度指标难以完全体现复杂的胆道弯曲形态。近年来有研究以胆总管中心线的曲率、迂曲指数，或以简化形态分型(直型、S 型、折线型等)进行风险分层。Ji 等人的研究显示：胆总管形态中的 S 型、折线型与复发风险显著相关，再加上如果胆总管直径 ≥ 1.5 cm，这种风险就更值得临床医师注意。把这两点放在一起，其实就能作为一个很实用的随访分层标准：符合的人群要更密切复查、管理得更积极[32]；在胃切除(Billroth II)等解剖改变人群中，S 型与折线型同样提示更高的结石复发倾向[33]。除了结石复发外，胆道折角还可能通过加重胆汁淤积与反流而增加反复性胆管炎的发病几率。Chong 等人也报道 CBD、CHD 角度 ≤130˚与胆总管角度 ≤125˚与复发性胆管炎显著相关[34]，以上研究提示胆管的正常角度上限在结石复发、胆管炎中可能存在差异。

因此，在临床上实际情况中，影像科医师如果能在 MRCP 或 ERCP 报告中对 CBD 几何形态进行结构化描述，至少包含远端 CBD 最小角度、形态分型(直型、S 型、折线型等)，将对患者的临床决策提供更加有利的依据。对急性折角(≤145˚或≤135˚) + CBD 扩张(≥15 mm)的患者，应考虑严密的随访与个性化治疗[23] [28] [32]。

3. 功能重构：Oddi 括约肌功能障碍与机体代谢的改变

正常情况下，胆囊与 Oddi 括约肌之间存在精密的神经协同：胆囊－括约肌反射。胆囊收缩时，SO 舒张以利胆汁排出；胆囊舒张充盈时，SO 收缩以维持胆道压力。胆囊切除术切断了这一反射弧，迫使 SO 进入一种“自律性适应”状态[18] [19]。胆囊切除术不仅改变胆道几何形态，更重要的是打破了胆囊－胆总管-Oddi 括约肌－十二指肠这一压力－流量系统的动态平衡：胆囊储存、浓缩与节律性排空消失后，胆道系统从间歇性、受控排放转向单出口、连续排放，胆汁酸(bile acids, BAs)的肠腔暴露模式与肠肝轴信号随之也发生了重排，从而形成患者表现出来的临床症状如腹痛、腹泻、胆汁反流相关上消化道症状等[21]。胆囊作为胆道的压力库、缓冲器，经典胆道动力学研究提示：胆囊并非被动囊袋，而是胆道系统的顺应性容器。在禁食与进食周期中，胆囊通过储存与节律性排空，参与平滑胆道压力波动、降低胆总管与乳头端的瞬时负荷；胆汁酸库在禁食期也并非静止，而是持续参与肠肝循环[35]。胆囊切除术后胆道压力、流量关系发生改变，同样的胆道出口阻力，更容易导致胆道压力升高，Scott 等人的压力－流量实验显示，胆囊切除后胆道系统的流动表现更依赖乳头端阻力(Oddi 括约肌张力)与胆总管本身的被动顺应性[36]。Tanaka 等人进一步用直接压力监测提出“失去胆囊作为压力库(loss of gallbladder as a pressure reservoir)”的概念：在乳头端短暂痉挛、阻力上升时，术后胆道压力反应更剧烈，因此更容易因胆道壁张力上升而

出现胆源性疼痛与胆道扩张的连锁反应。结构–功能的相互塑形临床影像学上，术后胆总管轻度扩张较常见，其意义需要结合症状与实验室指标解释[21]。国内《胆囊切除术后常见并发症的诊断与治疗专家共识(2018 版)》也强调：胆囊切除后胆囊与 Oddi 括约肌之间的协调被破坏，括约肌痉挛、胆汁排出受阻与胆总管扩张、壁张力升高可共同参与疼痛发生[37]。

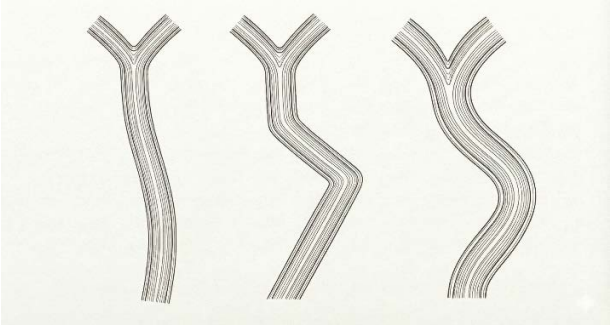

Figure 1. Right: S-shaped; middle: kinked type; left: straight type
图 1. 右侧：S 型；中间：折线型；左侧：直线型

胆囊切除术后，胆汁酸储存库消失，胆汁酸更持续进入肠道并使肠肝循环周转加快。Berr 等研究提示，循环次数增加可使初级胆汁酸(CA, CDCA)更频繁暴露于肠道细菌，从而促进次级胆汁酸(DCA, LCA)生成[38]。次级胆汁酸比例升高具有潜在病理生理意义：以 DCA 为代表的疏水性胆汁酸在高浓度条件下与黏膜损伤及结直肠肿瘤相关机制(如氧化应激与 DNA 损伤)有关。与此同时，胆汁酸负反馈调控轴(回肠 FXR-FGF19-肝 CYP7A1)在部分患者中可能出现反应不足：Barrera 等人发现，胆囊切除后可见 FGF19 水平下降并伴随 C4 升高，提示胆汁酸合成增加[39]。当过量胆汁酸未被回肠完全重吸收而进入结肠，可通过胆汁酸受体介导的分泌与动力增强诱发胆汁酸性腹泻(BAD)，这被认为是 LC 术后慢性腹泻的重要机制之一；Borup 等人建议对术后腹泻患者常规检测 SeHCAT 或血清 C4 测定，以明确胆汁酸重吸收障碍并指导治疗[40]。此外，胆囊切除与代谢相关结局的关联亦受到关注：Luo 等纳入约 2700 万人群的系统评价与荟萃分析提示，胆囊切除与非酒精性脂肪性肝病(nonalcoholic fatty liver disease, NAFLD)、代谢功能障碍相关脂肪性肝病(metabolic dysfunction-associated steatotic liver disease, MASLD)等慢性肝病风险增加存在统计学关联，可能与胆汁酸信号及脂质、葡萄糖代谢调控改变有关[41]。

谈到胆囊切除术后 NAFLD、MASLD 风险升高时，我们应该认识到：研究里看到的往往只是有关系，并不等于手术就是罪魁祸首。术后风险上升可能和患者原本的代谢问题、生活方式等因素有关，不能简单下结论说做了胆囊切除手术就会得病。LC 的主要指征是胆囊结石、胆囊炎，而胆石症本身与肥胖、胰岛素抵抗、2 型糖尿病、血脂异常等高度同源，因而 LC 人群天然携带更重的基线代谢负荷与“共同土壤”偏倚。Latenstein 等人的研究显示：LC 人群 MASLD 与超声 NAFLD 患病率确实更高，但在将 BMI、高血压、糖尿病、生活方式/能量摄入等代谢变量纳入多变量模型后，LC 与 MASLD、NAFLD 的独立关联不再显著，这提示共同土壤可能解释了一定程度的表观风险差异[42]。另一方面，采用倾向性评分方法的研究提示“手术效应”可能并非完全为混杂所致，但更像是在高代谢风险背景下的效应放大：韩国全国队列在对多类心代谢危险因素与生活方式进行 PSM 并排除基线脂肪肝高风险后，LC 与新发 MASLD 仍呈正相关，且风险主要集中在合并≥3 项心代谢危险因素者，低风险亚组关联趋于不显著[43]。因此，我们可以理解为，做了腹腔镜胆囊切除术(LC)的人，往往本身就更容易处在代谢不太健康的人群里，LC 更像是一个风险提示信号，而不是直接把人“切”出脂肪肝。手术之后胆汁酸信号和肠–肝轴可能会发生变化，在那些本来代谢负担就重的人身上，这种变化可能加快病情进展，让问题更容易出现或发展得更

快。但就目前的观察性研究证据来说，还不能把 LC 当成脱离其他因素的唯一“罪魁祸首”。

4. 临床结局：症状轨迹与胆道并发症

胆囊切除术后的症状结局具有显著差异，部分患者症状完全缓解，部分患者出现原有症状持续或新发的上腹痛、腹胀、腹泻、烧心、反酸及上消化道不适，临床上常被归入 PCS。PCS 并非单一疾病，而是胆道器质性问题、功能性胃肠病(functional gastrointestinal disorders, FGIDs)、胆汁酸相关代谢改变共同作用的表型集合。PCS 发生率在不同研究中差异较大，近年综述与队列研究仍提示其可影响相当比例患者，并强调术前筛查与术后分层评估的重要性[4]。PERFECT 前瞻性多中心观察研究显示：约三分之一拟行胆囊切除的胆石症腹痛患者，术后典型胆绞痛显著减少，但非胆源性腹痛持续比例仍高[44]。这就更进一步强调对胆囊切除术后患者的远期随访观察，以减少 PCS 的发生。

另外，胆囊切除后胆汁酸缺乏储存与餐后脉冲式释放，从而持续进入肠道；当回肠重吸收与反馈调控不足，胆汁酸到达结肠可刺激水与电解质分泌并增强动力，从而形成胆囊切除术后腹泻(post-cholecystectomy diarrhea, PCD)或胆汁酸腹泻(bile acid diarrhea, BAD) [12]。对此，BSG 慢性腹泻指南建议：对持续不明原因慢性腹泻，应主动检测胆汁酸腹泻(如 SeHCAT、血清 C4 等) [13]。上消化道症状与胆汁反流：胆囊切除可能增加十二指肠内容物反流入胃的概率，形成胆汁反流性胃病、胃炎等。Othman 等人的回顾性队列研究在“难治性上腹痛、上消化道症状”就诊人群中发现，胆囊切除组患者胆汁反流性胃炎检出率明显高于对照组，并提示肥胖、糖尿病、胃内胆红素升高与胃 pH 升高等风险特征；需要强调的是该研究基于症状人群，存在选择偏倚，不能直接外推至普通术后人群的总体患病率[45]。Lake 等人的研究提出：功能性消化不良的患者胆汁反流性胃病与更重腹痛相关，并且胆囊切除病史与胆汁反流性胃病显著相关，提示胆囊切除可能是重要易感因素[46]。临床上，胆汁反流相关诊断多依赖胃镜下胆汁潴留、黏膜充血、糜烂及病理“化学性胃病”改变；治疗方面高质量随机证据仍不足，临床多采取生活方式、促动力与黏膜保护等综合策略。

胆囊切除术后胆道并发症中，胆总管结石(common bile duct stone, CBDS)具有较高的临床负担，可表现为腹痛、梗阻性黄疸、胆管炎，甚至诱发胰腺炎。术后早期发现的胆管结石往往与“残余结石”相关，而术后较晚期再发则更多被视为“复发结石”。既往研究多将“6 个月”作为残余与复发的分界：6 个月内发现者更倾向于残余，6 个月及以后发现者更倾向于复发[47]。在“术后胆管结石复发”的危险因素方面，既往研究与系统评价提示其形成往往是多因素叠加：除年龄、既往胆道手术史等一般因素外，肝外胆管扩张、乳头旁憩室、胆道感染、炎症、结石负荷以及内镜治疗相关因素(如气胆、碎石、是否一次完全清除等)均被反复报道与复发风险相关，同时，越来越多证据强调胆道解剖几何特征的重要性，例如 CBD 走形、形态(如 S 型形态)、胆管成角与局部淤胆倾向可能参与结石再形成过程[48] [49]。因此，明确胆囊切除术后胆道形态与通畅性变化，并识别与术后结石相关的危险因素，对临床风险分层与随访策略制定具有重要意义。

此外，与胆囊切除术直接相关、且越来越受关注的因素是胆囊残端结构。Burckhardt 等人的 MRCP 病例–对照研究显示：存在胆道结石者的胆囊管残端更长并且当残端长度 ≥15 mm 时结石风险可增加至约 2 倍以上，≥45 mm 时风险进一步升高[50]。这提示我们，胆囊切除术后胆道重构不仅是关系有没有胆囊，还包括胆囊管残端这一“盲端”对胆汁淤积与结石成核的贡献。其次，夹子迁移所致的胆管结石也有研究提出，应作为临床上远期结石复发的鉴别诊断之一，夹子可进入胆道并成为结石“核心”，表现为反复胆管炎、黄疸或胰腺炎，常需行 ERCP 取出[51]。

5. 结语

胆囊切除不是终点，而是机体新平衡建立的起点。解剖结构的代偿性扩张与几何学改变，为胆汁动

力学与结石复发奠定了基础；肝–肠–菌群轴的再平衡，则将局部胆道事件扩展为全身代谢与肝病结局的长期议题。症状层面，胆囊切除术后综合征、胆汁酸性腹泻、功能性胃肠病及 NAFLD 等表型交织出现，使得单一学科难以充分应对。真正具有临床价值的术后管理，应以“结构–功能–症状”三位一体的理念为基础，通过多模态影像学评估、代谢与菌群组学分析以及心理–行为状态的系统测量，构建可视化、可预测、可干预的长期随访体系。在这一综合框架下，胆囊切除术的意义也将由单纯“解决当前结石问题”延伸至围绕胆道及肝–代谢轴的终身风险评估与健康维护，从而更全面地提升无胆囊人群的长期预后与生活质量。

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