

尿道及其周围组织结构变化对女性压力性尿失禁影响的研究进展

李元智*, 郑斌峰*, 汤武, 杨涛, 方克伟#

昆明医科大学第二附属医院 泌尿外科, 云南 昆明

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摘要

女性压力性尿失禁(Female urinary stress incontinence, SUI)是女性常见疾病, 其发病率随年龄增长而增加。疾病对患者生活、心理带来巨大压力及沉重经济负担。本综述对尿道及其周围组织结构在尿控及对女性SUI影响方面的相关文献进行综述、分析, 探讨尿道及其周围组织结构在SUI进程中的重要性, 为未来对SUI疾病研究及治疗方案提供参考。

关键词

女性压力性尿失禁, 尿道, 阴道前壁, 盆腔支持结构, 尿控

Research Progress on the Impact of Changes in Urethral and Periurethral Tissue Structure on Female Stress Urinary Incontinence

Yuanzhi Li*, Binfeng Zheng*, Wu Tang, Tao Yang, Kewei Fang#

Department of Urology, The Second Affiliated Hospital of Kunming Medical University, Kunming Yunnan

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Abstract

Female urinary stress incontinence (FUSI) is a common gynecological disorder, with its incidence

*共同第一作者。

#通讯作者。

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increasing with age. The disease imposes significant psychological stress and economic burden on patients' lives. This review summarizes and analyzes relevant literature on the structural relationship of the urethra and its surrounding tissues to urinary control and their impact on female SUI, exploring the importance of the urethra and its surrounding tissue structures in the pathogenesis of SUI, so as to provide reference for future research and treatment strategies of SUI.

Keywords

Female Urinary Stress Incontinence, Urethra, Anterior Vaginal Wall, Pelvic Support Structures, Urinary Control

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1. 引言

压力性尿失禁(Urinary stress incontinence, SUI)是指在腹压增加时,如咳嗽、打喷嚏或运动时,不自主地从尿道漏出尿液[1]。女性多见,其发病率为25%~45% [2] [3],随年龄增长而增加[4] [5]。SUI对患者的生活质量造成多方面的影响,不仅影响日常活动,还导致心理困扰和社会孤立[6]-[10]。同时,SUI也是一个严重的公共卫生问题,为家庭和社会带来巨大的经济负担[11]。SUI的发生与多方面的因素密切相关[1] [2] [12]。本文将聚焦于尿道及其周围组织在SUI疾病进展中解剖结构变化、病理生理表现进行综述和讨论。

2. 尿道解剖结构与 SUI

女性尿道由上皮、平滑肌、骨骼肌和结缔组织组成,这些组分共同维持其结构和功能的完整。

2.1. 尿道正常结构与尿控

女性尿道相对较短,长度平均约3~5厘米,几乎完全由膜部组成[13]。尿道内层通常由移行上皮和复层柱状上皮组成[13]。尿道壁由内外两层肌肉组成,内层为平滑肌,构成内尿道括约肌;外层为骨骼肌,形成外尿道括约肌[14] [15]。其中,平滑肌层又分为内层的纵向平滑肌和外层相对较薄的环形肌[14]。尿道平滑肌通过收缩和舒张来调节尿道管腔的阻力,从而控制尿液的流动。尿道平滑肌的收缩是维持尿控的重要机制之一[14]。在膀胱充盈期间,膀胱逼尿肌保持松弛以适应尿量增加,而尿道则保持收缩以防止尿液泄漏[16]。尿道平滑肌的这种持续性收缩活动,有助于维持尿道内压,抵抗膀胱内压,从而实现尿液的有效储存[14] [17] [18]。女性尿道外括约肌主要由骨骼肌组成,围绕尿道。在胎儿发育过程中,外部尿道括约肌从一种未分化肌肉纤维的同心括约肌演变为横纹肌的瞬时环,在出生后第一年内,当括约肌呈现 Ω 形结构时,该环在后尿道中从尾部向颅部退化[13] [19]。Venema等人在成人女性尿道括约肌系统的解剖研究表明,它并非简单的环形结构,而是由多个部分组成,包括括约肌尿道肌(sphincter urethrae)、尿道阴道括约肌(urethrovaginal sphincter)和尿道压迫肌(compressor urethrae)等(图 1) [20]。这些肌肉协同作用,确保尿道的闭合[20] [21]。

尿道外括约肌是维持尿控的关键组成部分,它通过收缩使尿道闭合,防止尿液漏出[15] [22]。膀胱充盈期间,尿道外括约肌的持续收缩有助于保持尿道压力高于膀胱压力,从而维持尿液的储存[23]。排尿时,尿道外括约肌必须放松,允许尿液顺畅排出。这种协调的尿道放松与膀胱逼尿肌的收缩共同完成排尿过

程[23][24]。综上,尿道自身生理结构通过改变尿道开闭情况从而改变尿道闭合压力来达到尿液控制的目的。

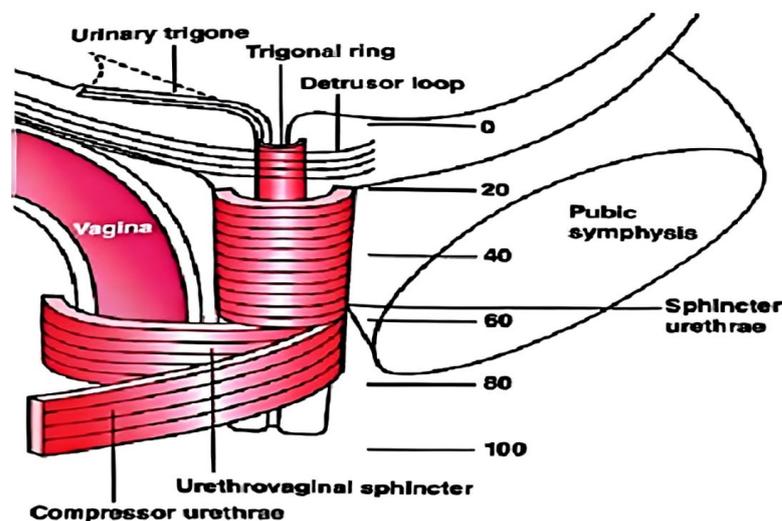


Figure 1. Shows a detailed anatomical diagram of the female lower urinary tract and pelvic floor structures, which include key muscles such as the sphincter urethrae, sphincter urethrae (vaginal part), and compressor urethrae. These structures collectively maintain urethral integrity and urinary control [20]

图 1. 显示了女性下尿路和盆底结构的详细解剖图, 其中包含尿道括约肌(sphincter urethrae)、尿道阴道括约肌(sphincter urethrae)和尿道压迫肌(compressor urethrae)等关键肌肉。这些结构共同维持尿道的完整性和尿液控制[20]

2.2. 尿道括约肌结构性异常与 SUI

尿道的闭合机制是维持排尿控制的关键, 主要由尿道括约肌系统实现[25][26]; 内尿道括约肌(平滑肌)与外尿道括约肌(骨骼肌)共同维持尿道闭合, 在膀胱充盈和排尿过程中发挥作用[15]。尿道括约肌结构性异常(括约肌纤维组织减少、肌肉退变等)导致的尿道的闭合机异常是 SUI 的重要病理因素之一。Morgan 等人通过磁共振成像也证实了 SUI 女性尿道括约肌具有解剖异常, 包括其长度和厚度可能与尿道功能和盆底功能具有相关性[27]。尿道括约肌的结构异常可能由多因素引起: ① 组织退化和年龄相关性变化: 随着年龄增长, 尿道括约肌结构发生异常, 如胶原纤维增生、肌肉密度降低, 都可能削弱尿道的闭合能力, 从而导致尿失禁[28][29]。Klauser 等人通过动态尿道内超声发现尿道括约肌的厚度和回声模式的变化与年龄相关[30]。② 经阴道分娩: 阴道分娩过程中, 胎头通过产道对盆底组织施加巨大的压力, 可能导致尿道括约肌的机械性损伤(肌肉过度伸展、撕裂), 这种损伤可能导致尿道横纹括约肌(EUS)的功能障碍, 从而影响尿道闭合机制导致 SUI 的发生[31][32]。③ 肥胖: Yung-Chin Lee 等在 Zucker 肥胖(ZF)大鼠模型中, 发现肥胖会引起尿道括约肌(包括平滑肌和横纹肌)的结构性改变[33]; 肥胖大鼠的尿道括约肌尽管体积或厚度可能无显著变化, 但其收缩功能受损, 这提示了这种改变是内在的结构性缺陷而非单纯的肌肉萎缩或肥大[33]。肥胖可能导致尿道括约肌组织内结缔组织和脂肪浸润的增加, 而正常的肌细胞比例可能下降。这种改变可能破坏肌纤维的排列和收缩效率, 影响括约肌的整体力学性能, 增加 SUI 风险。④ 尿道结构异常: 如尿道憩室等的尿道异常结构的存在会对尿道及其周围的括约肌组织施加机械压力; 这种压力可能导致括约肌功能的退化或紊乱, 使得括约肌无法有效地闭合尿道, 从而在腹压增加(如咳嗽、打喷嚏、运动)时发生尿液不自主渗漏[26]。其次, 尿道憩室容易发生感染或炎症[34]。反复的感染和炎症反应可能导致局部组织的纤维化, 进一步影响括约肌的完整性和弹性[35]。纤维化会使括约肌组织变得僵硬, 降低其顺应性, 从而影响其正常功能[35]。

2.3. 局限性与争议

尿道自身解剖结构与 SUI 虽然女性尿道的解剖层次及其括约肌复合体的组成已通过大体解剖学得到确认[20] [21],但在临床转化与机制研究中仍存在显著局限:① 微观功能量化的缺失:目前的尿动力学测试虽能评估尿道压,但仍无法在活体状态下精准量化尿道各解剖结构的实时闭合贡献[33]。② 影像学评估的宏观性:现有的高分辨率 MRI 和超声手段能观察尿道长度(3~5 cm)及肌肉萎缩情况[27],但难以准确量化尿道横纹肌内神经支配密度的微小变化,而这正是区分内在括约肌功能障碍(ISD)的关键。③ 内在因素与外在支持的权重争议:目前的治疗手段多集中于加强“吊床”样的外在支持(如中段尿道悬吊术),但对于如何恢复尿道自身黏膜密封性及括约肌张力的争议依然存在,这导致部分 ISD 患者在接受常规手术后疗效不佳。

3. 尿道周围支持结构与 SUI

3.1. 正常尿道周围支持结构与尿控

女性尿道并非一个简单的管道器官,而是由多层结构和复杂的支持结构组成;其功能和自身尿道属性和周围盆腔支持结构紧密相关[36]。女性尿道主要通过其阴道前壁的筋膜与提肛肌相连,形成一个“吊床”样的支持系统,即所谓“吊床假说”[36] [37]。该“假说”认为,尿道置于一个由阴道壁及其连接物组成的支撑层上,在腹内压增高时,尿道会被压向耻骨联合,从而关闭尿道口,防止漏尿[26] [36] [37]。

3.1.1. 阴道前壁与尿控

阴道前壁位于膀胱与尿道后方,构成尿道支持系统的核心解剖基础。其与尿道的空间关系呈“叠瓦状”分布,近端 1/3 与膀胱颈紧密相邻,中段与尿道平行走行并通过致密结缔组织相连,远端 1/3 则延伸至尿道外口下方[38]。这种特殊的解剖排列使阴道前壁在腹压增加时能够为尿道提供动态支撑,是维持尿控功能的重要结构基础[38] [39]。阴道前壁由黏膜层、肌层、外层筋膜三层组织构成,各层在维持尿道支持功能中发挥协同作用[38] [39]。黏膜层由复层鳞状上皮和固有层构成[21] [38],上皮细胞富含糖原,对雌激素水平的变化高度敏感[40] [41]。这有助于维持阴道的湿润环境和弹性[21]。同时,这种激素依赖性的形态学变化不仅影响黏膜的分泌功能,还对其屏障保护作用产生影响[40]。固有层含丰富的弹性纤维网络和微血管丛,其独特的“波浪状”胶原排列赋予组织在应力状态下的延展性[42]-[44]。肌层由内环外纵的平滑肌束组成[45] [46],肌纤维密度呈现梯度分布(近端 32.5 ± 4.2 束/mm²,远端 18.7 ± 3.6 束/mm²) [45]。特殊的是,其与尿道括约肌通过“肌-筋膜桥”结构相连,可将阴道壁的张力变化直接传递至尿道[47]。外层筋膜为盆内筋膜的延续,主要由 I 型(70%)和 III 型(30%)胶原纤维构成[48],形成“吊床样”支撑结构[49],从而维持尿道正常生理位置,防止漏尿。该层含有大量成纤维细胞,成纤维细胞的数量和功能对于维持胶原的正常代谢至关重要[50]。其分泌的基质金属蛋白酶抑制剂(TIMP-1)可维持胶原代谢平衡,有助于维持和修复细胞外基质(ECM) [51]。这是支持力持久性的关键。

3.1.2. 盆腔支持结构与尿控

盆腔支持结构与尿控之间存在密切且复杂的关系,其正常功能对于维持尿液和粪便的持续排泄以及盆腔器官的正确解剖位置至关重要[52]-[56]。盆腔支持结构由肌肉、韧带、筋膜和神经网络共同构成,共同为盆腔脏器(如膀胱、肠道和子宫)提供主动和被动支持(图 2) [55] [57]-[62]。形成一个类似于“吊桥”的结构[48]。在“吊桥”系统中,韧带和筋膜提供了主要的被动支持[21] [57]。这些结构在静态时维持盆腔器官的解剖位置。例如,子宫骶韧带(USL)、骶棘韧带(SSL)和耻骨尿道韧带等对盆腔器官的固定和尿控机制起到关键作用[63] [64]。这与吊桥的缆绳类似,在没有外部载荷时保持桥面的稳定。盆底筋膜,特

别是盆筋膜腱弓(ATFP)与前阴道壁的连接,对于尿道的正常支持至关重要[21][57]。这可以类比为吊桥的桥面结构,为器官提供了基础承托。盆底肌肉提供了动态的、主动的支持;女性盆底肌肉形成一个横跨整个盆腔的隔膜,主要由髂尾肌和肛提肌组成[57][61]。肛提肌作为盆底最重要的肌肉群之一,肛提肌及其筋膜共同为尿道、阴道和直肠提供支持,并通过收缩控制尿道、阴道和肛门开口[57]。当腹腔内压升高时(如咳嗽、打喷嚏或举重物),盆底肌肉会主动收缩,通过增加尿道闭合压来防止尿液渗漏[57]。这相当于吊桥中的可调节部件,能够根据需要提供额外的支撑。盆腔支持系统是一个相互依赖的整体,任何一部分的薄弱都可能影响到整体的稳定性。

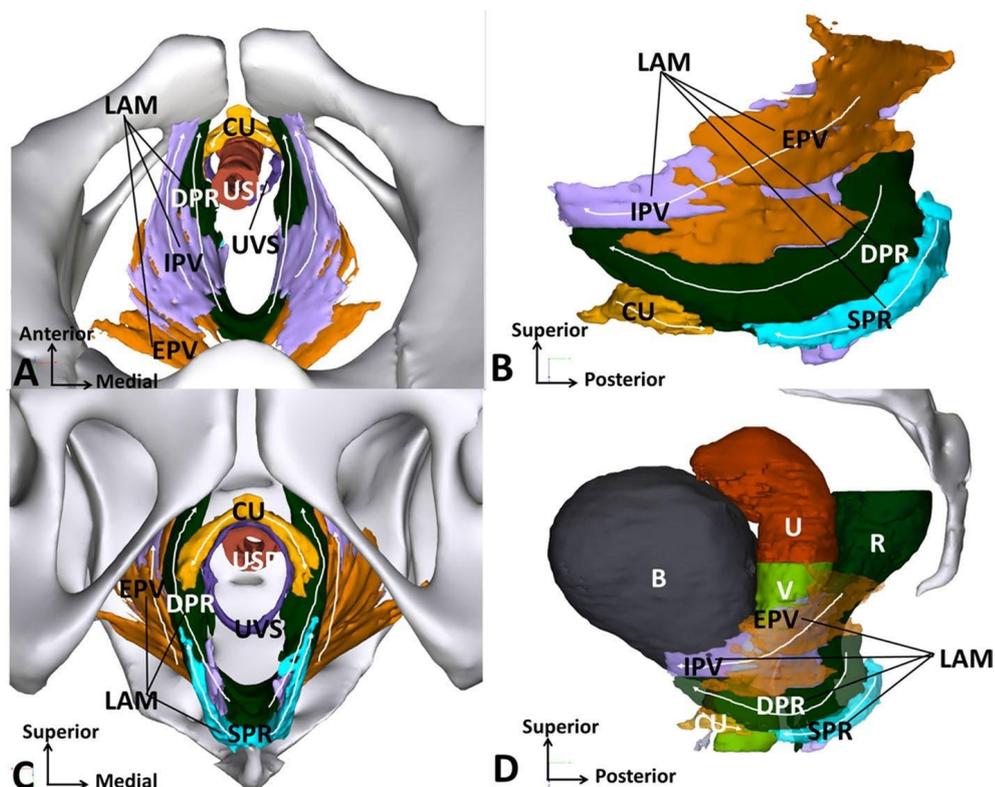


Figure 2. (Figures A-D) presents detailed anatomical views of pelvic-related structures, displaying the pelvic floor muscles, ligaments, and annular structures from different angles, including the levator ani muscle (LAM), coccygeus muscle (CU), deep perineal ring (DPR), urethral sphincter complex (USP), urethrovaginal sphincter (UVS), inferior pubovesical ligament (IPV), and superior pubovesical ligament (EPV) [21]. These structures collectively form the complex anatomical network of the pelvic floor, which is crucial for the support and function of pelvic organs [21]

图 2. (图 A-D)展示了盆腔相关结构的详细解剖视图,从不同角度呈现了盆底肌肉、韧带和环状结构,包括肛提肌(LAM)、尾骨肌(CU)、深会阴环(DPR)、尿道括约肌复合体(USP)、尿道阴道括约肌(UVS)、耻骨膀胱下韧带(IPV)和耻骨膀胱上韧带(EPV)等[21]。这些结构共同构成盆底的复杂解剖网络,对盆腔器官的支撑和功能至关重要[21]

3.1.3. 局限性与争议

尽管“吊床假说”为理解尿控机制提供了直观的物理模型[36][37],但在临床转化中仍存在争议。目前的影像学手段,如动态MRI或超声,虽能清晰显示盆底器官的解剖位移和韧带的宏观形态(如变薄或拉长)[21],但仍难以在活体状态下精准量化微观组织结构的物理特性(如阴道前壁固有层中弹性纤维的实时张力或胶原交联的化学稳定性)。此外,虽然已知雌激素水平通过影响黏膜层来维持阴道环境[40][41],但激素替代疗法在恢复组织生物力学强度方面的直接贡献比例仍是学术界讨论的热点,尚未形成统一的量化标准。

3.2. 尿道周围支持结构异常与 SUI

SUI 的病因复杂且多因素, 其中尿道支持结构的完整性是关键因素之一[65]。尿道周围支持结构异常会导致压力传递机制受损和尿道闭合功能减弱, 进而引发 SUI [66]。

3.2.1. 阴道前壁结构异常与 SUI

压力性尿失禁发生时, 阴道前壁结构呈现特征性的病理改变, 主要表现为组织结构损伤、尿道过度活动性增加[67][68]。这些病理改变共同导致尿道支持系统功能衰退, 在腹压增高时无法维持有效的尿道闭合压力, 从而引发不自主漏尿症状[67][68]。阴道前壁的组织学结构在 SUI 患者中呈现多层次的退行性改变, 直接影响其力学支持功能: ① 胶原纤维重组: a. I 型胶原占比从正常 70% 降至 50%~55%, III 型胶原比例异常增高, 纤维排列从规则交叉(55°~65°)变为紊乱分布[69]; b. 胶原交联密度降低 $40\% \pm 8\%$, 导致拉伸强度从 3.2 MPa 下降至 1.8 ± 0.3 MPa [70][71]; c. 基质金属蛋白酶 MMP-1 活性升高 3 倍, 加速胶原降解[67]。② 弹性纤维减少: a. 弹性蛋白含量减少 50%~60%, 断裂伸长率下降至健康组织的 40% [72][73]; b. 弹性纤维再生障碍与 LOXL1 基因表达下调相关(表达量降低 $65\% \pm 12\%$) [74]。③ 细胞外基质改变: 细胞外基质中透明质酸、纤维连接蛋白、硫酸软骨素等成分降低[75], 可能导致组织水合度下降、细胞粘附能力减弱以及组织压力缓冲功能降低[75]-[77]。上述病理改变导致阴道前壁弹性及对尿道支持能力下降, 无法有效传递盆底肌收缩力至尿道。阴道前壁与尿道间的剪切力传递效率下降 60%~70%, 无法形成有效的“吊床支撑” [36][78]; 膀胱颈下移超过正常位(>30 mm), 尿道倾斜角从静息状态 15°~20° 增大至 45°~60° [79]。阴道前壁支持功能下降导致尿道过度活动[68][80]。以上病理过程形成恶性循环, 共同促进 SUI 的进展和持续状态。

3.2.2. 盆腔支持结构异常与 SUI

SUI 的发生常常与盆腔支持结构的功能或结构损伤相关[81]。盆腔韧带, 例如耻骨尿道韧带、骶棘韧带、子宫骶骨韧带等, 在固定盆腔脏器和维持尿道位置方面发挥着不可替代的作用[63][64][82]。当这些韧带因各种原因受损或松弛时, 尿道及其周围结构可能失去足够的支撑, 导致尿道高活动度或内在括约肌功能障碍[82]; Li Shi 等人利用三维重建融合技术结合静态和动态 MRI 对初产妇盆底结构进行评估, 可以发现 SUI 患者韧带的拉长、变薄或附着点异常[82]。这些形态学改变直接影响韧带的功能, 导致尿道支持不足, 进而引发 SUI。Madill 和 McLean 提出的 SUI 结构缺陷模型强调了盆腔筋膜和盆腔肌肉在 SUI 发病中的重要性, 该模型指出 SUI 涉及盆底肌肉撕裂导致盆腔筋膜张力增加、盆底肌肉去神经化引起的筋膜撕裂、撕裂导致的筋膜薄弱以及盆底肌肉收缩效率低下四个主要的盆腔支持结构缺陷[81]。SUI 患者常表现出盆底肌肉力量、耐力或协调性的下降[83]。这种肌肉功能的不足使得尿道在腹压增加时无法获得足够的支撑和闭合压力, 从而导致尿液渗漏[83]。盆底肌肉的功能障碍(如撕裂或力量不足)会直接影响盆腔筋膜的张力[81]。当盆底肌肉无法提供足够的支撑时, 盆腔筋膜可能会承受过度的张力, 导致其过度拉伸或损伤, 从而影响其正常功能[81]。长期慢性腹内压增高(如肥胖、慢性咳嗽、便秘)也会对盆腔筋膜施加持续的应力, 逐渐导致筋膜的过度拉伸和损伤, 最终影响其支持功能[84]。以上各种病理改变可能导致膀胱颈和尿道中段在腹压增加时缺乏必要的支撑, 进而引起尿液的非自主渗漏[37]。

综上, 多因素导致的尿道及其周围组织正常解剖结构异常或受损, 会通过影响尿道闭合机制从而诱发或者造成女性压力性尿失禁的发生(表 1)。

3.2.3. 局限性争议

针对 SUI 的组织工程修复策略(如生物支架或干细胞疗法)正成为研究前沿, 旨在修复受损的胶原网络和细胞外基质。然而, 目前的最大瓶颈在于: ① 生物力学匹配性: 人工合成或天然生物材料难以完全

模拟阴道前壁非线性、大变形的弹性特征[68] [80], 易导致应力遮蔽或二次损伤; ② 神经肌肉整合: 简单的结构填充难以恢复盆底肌肉的“动态收缩”效率, 无法解决 Madill 模型[81]中提到的去神经化问题。此外, 实验室检测到的基因表达下调(如 LOXL1 表达降低 65%左右[74])如何精准对应到临床漏尿的严重程度, 仍缺乏大规模的纵向队列数据支持。

Table 1. Key Histological Differences between Urethral and Surrounding Tissues in SUI Patients and Healthy Women
表 1. SUI 患者与健康女性尿道及周围组织的关键组织学差异

组织成分	健康女性特征	SUI 患者特征	相关病理影响
胶原纤维	I 型占 70%, 排列规则	I 型降至 50%~55%, 排列紊乱	组织拉伸强度显著下降
弹性纤维	含量丰富, LOXL1 表达正常	含量减少 50%~60%, LOXL1 表达降低	组织回弹能力障碍
平滑肌束	近端密度 32.5 ± 4.2 束/mm ²	密度显著降低, 伴随去神经化	“肌-筋膜桥”张力传递失效
MMP-1 活性	代谢平衡	活性升高 3 倍	加速细胞外基质降解

4. 临床启示

尿道及其周围组织结构的完整性是维持女性尿控功能的基石。尿道作为排尿通道, 其周围环绕着复杂的解剖结构, 包括尿道外括约肌、盆底肌肉群以及支撑尿道的筋膜和韧带等, 这些结构共同构成了一个精密的“防漏系统”。当这些组织结构因各种原因出现损伤、退化或功能异常时, 尿控功能便会出现障碍。因此, 针对压力性尿失禁(SUI)的临床诊疗应从传统的通用管理模式转向基于解剖缺陷的精准分型治疗。临床医生可利用三维超声或动力学 MRI 明确患者的结构损伤类型并选择与之对应的治疗方案: 对于由阴道前壁筋膜松弛或耻骨尿道韧带受损引起的尿道过度活动型患者, 应首选尿道中段悬吊术(MUS)以重建“吊床”样的被动支撑; 若患者伴有严重的阴道前壁松弛、I 型胶原比例显著下降及盆腔脏器脱垂, 则需考虑执行尿道悬吊术联合阴道前壁修补术(Colporrhaphy)或使用生物材料支架进行整体筋膜修复; 而针对内在括约肌功能障碍(ISD)导致的闭合压不足, 单纯加强支撑效果有限, 应优先选择尿道黏膜下填充剂注射或张力稍高的耻骨后路径吊带手术。此外, 手术重建的被动支撑必须结合盆底肌训练(PFMT)以修复受损肛提肌的动态闭合功能, 并辅以围绝经期激素管理以优化胶原代谢平衡, 最终形成一个涵盖影像诊断、针对性术式匹配及围手术期康复的个体化综合管理体系(表 2)。

Table 2. The impact of different anatomical defects (pathological changes) on clinical symptoms (features) and treatment options
表 2. 不同解剖缺陷(病理改变)对临床症状(特征)及治疗选择的影响

解剖缺陷类型	关键解剖/病理改变	对临床症状/特征的影响	对应治疗策略/具体术式
尿道过度活动/支持结构失效	耻骨尿道韧带拉长或变薄、阴道前壁筋膜(吊床结构)松弛、盆筋膜腱弓(ATFP)撕裂。	腹压增加时尿道中段缺乏有效支撑; 尿道下移过度(>30 mm), 倾斜角显著增大。	尿道中段悬吊术(MUS): 通过 TVT/TOT 等术式重建被动支撑, 恢复“吊床”功能。
严重阴道前壁松弛(伴盆腔脏器脱垂)	I 型胶原占比显著降至 50%~55%; 基质金属蛋白酶 MMP-1 活性升高 3 倍导致胶原加速降解。	表现为明显的阴道前壁膨出, 盆底整体支撑力缺失, 单纯悬吊术复发风险高。	联合修补术: 尿道悬吊术联合阴道前壁修补术(Colporrhaphy), 必要时使用生物材料支架加强。
内在括约肌功能障碍(ISD/自身结构受损)	尿道横纹肌密度下降、闭合压不足; 尿道黏膜密封功能失效或神经支配受损。	即使在无尿道高活动度的情况下, 轻微腹压即可导致严重尿失禁。	功能增强疗法: 尿道黏膜下填充剂注射、高张力耻骨后路径吊带术或人工尿道括约肌植入。

续表

动态支持缺陷 (盆底肌损伤)	肛提肌(LAM)发生撕裂、去神经化或废用性萎缩。	压力增加时缺乏反射性主动闭合压力，盆底肌收缩效率低下。	主动康复策略： 盆底肌训练(PFMT)及电刺激，激活残余肌肉的动态闭合功能。
组织质量与代谢缺陷	绝经后雌激素水平下降，导致黏膜变薄及胶原交联密度降低。	组织水合度及弹性下降，应力缓冲功能减弱，影响术后组织修复能力。	围手术期内分泌管理： 局部补充雌激素以改善黏膜厚度及胶原代谢平衡。

5. 总结与展望

女性压力性尿失禁的发生与发展与尿道自身解剖结构和尿道周围支持结构的生理病理改变密切相关。同时，各个解剖结构之间密切联系、相互影响。其中某个结构发生异常可能会导致其他多结构乃至整体受影响。因此，我们在对压力性尿失禁疾病研究过程中，既要关注局部改变，也要掌握整体。应从多个方面，作具体全面的考量。同时，我们对于女性压力性尿失禁的诊疗与预防可考虑从其具体的病理过程入手，延缓甚至逆转其病理过程发生发展，从而达到非手术干预治疗或延缓疾病进展甚至达到早期预防的目的。

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