·综述·

儿童青光眼诊断和治疗的研究进展

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【摘要】 儿童青光眼是严重危害儿童视觉健康的疾病，将伴随儿童患者终身，给家庭和社会带来沉重负担。青光眼所致儿童盲多数可预防或可治疗，近年的相关研究取得了新的进展。本文在世界青光眼协会达成新的共识基础上，基于国内外最新循证医学证据，针对儿童青光眼新的定义与分类体系、诊断、分子遗传学特点、病理和发病机制以及药物、手术等综合治疗等方面进行总结和分析，重点关注各类手术方法在儿童青光眼治疗中的应用进展，以期为临床及相关科研工作提供参考，共同提高儿童青光眼的临床诊疗水平。

【关键词】 青光眼；　诊断；　治疗学；　儿童

Research progress in the diagnosis and treatment of childhood glaucoma

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【Abstract】 Childhood glaucoma is a disease that seriously endangers children's visual health. It will accompany the patients throughout their lives and bring a heavy burden to families and society. Most childhood blindness caused by glaucoma is preventable or treatable. Relevant research has made progress in recent years. Based on the new consensus reached by the World Glaucoma Association and the latest medical evidence at home and abroad, this article summarizes the definition, classification, diagnosis, molecular genetics, pathogenesis and comprehensive treatments including drugs and surgery of childhood glaucoma, with a focus on the application of various surgical methods, so as to provide reference for clinical and scientific research and improve the clinical diagnosis and treatment of childhood glaucoma.

【Key words】 Glaucoma;　Diagnosis;　Therapeutics;　Child

儿童青光眼是导致儿童盲的主要原因之一[1-2]。儿童患者到预期寿命时间要远长于成人，视力丧失所带来的负面效果将伴随终身，给家庭和社会带来更为沉重的负担。青光眼所致儿童盲多数可预防或可治疗，应特别强调针对儿童青光眼所致儿童盲的预防性战略。本文基于国内外最新循证医学证据和世界青光眼协会专家共识意见，就儿童青光眼的分类、遗传学特点、发病机制、诊断和治疗等方面进行分析和总结。

一、儿童青光眼的定义和分类

世界青光眼协会新的定义与分类体系推荐使用名词为儿童青光眼。在我国儿童的定义为小于 18 岁，青光眼则符合一般青光眼的定义。儿童青光眼进一步分为原发性和继

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发性两大类。原发性青光眼包括原发性先天性青光眼（primary congenital glaucoma，PCG）和青少年开角型青光眼（juvenile open angle glaucoma，JOAG）。PCG与JOAG的区别在于后者不伴有高眼压下的眼球扩张，该区别在年龄方面存在个体差异，为此世界青光眼协会提出了一项国际共识，即JOAG发病的最早年龄为4岁，而最晚年龄可达30~40岁。与原发性开角型青光眼（primary open angle glaucoma，POAG）相比，JOAG的眼压升高幅度有所不同，典型的JOAG表现出极高的眼压，甚至超过40~50 mmHg（1 mmHg=0.133 kPa）。多数JOAG患者前节和房角结构正常，部分患者房角会出现梳状韧带较多、虹膜根部高位附着等现象以及虹膜纹理轻度异常，这与POAG不同[3]。

继发性青光眼可分为青光眼合并非获得性或获得性疾病以及白内障摘除手术后继发青光眼3个类型，其中获得性与非获得性根据伴发疾病或异常是否在出生时即存在加以分类，青光眼合并的获得性疾病包括葡萄膜炎、外伤、糖皮质激素诱发疾病、肿瘤等；而青光眼合并的非获得性疾病又可细分为2类，即青光眼合并非获得性眼部异常（如Axenfeld-Rieger异常、Peter异常、无虹膜症、先天性葡萄膜外翻等）和青光眼合并非获得性全身疾病或综合征（如Sturge-Weber综合征、多发性神经纤维瘤病、眼脑肾综合征等）。与旧的分类系统相比，新的分类系统囊括了更广范围的儿童青光眼疾病，将儿童青光眼人群统一在一起。新的分类体系已在国际交流、合作以及大规模临床研究中广泛使用[4]。

二、儿童青光眼的诊断

儿童青光眼的诊断，除了年龄外，须至少符合以下2项：（1）眼压>21 mmHg；（2）视盘凹陷（盘沿变窄）：视杯与视盘直径比（杯盘比）进行性增大（弥漫性盘沿变窄），双眼杯盘比不对称（差值≥0.2）或出现盘沿局部变窄；（3）角膜改变：出现Haab纹、角膜水肿，或者角膜直径≥11 mm（新生儿）、>12 mm（年龄<1岁儿童）、>13 mm（任何年龄）；（4）进展性近视或近视漂移合并眼轴增长速度大于正常生长速度；（5）与青光眼性视神经病变相对应、可重复检测到的视野缺损，并排除其他引起视野缺损的病变。

1.眼压：正常新生儿的眼压较成人平均值低，在少年时期达到成人水平。儿童眼压测量存在较多潜在影响因素，如眼压计种类、眼球运动、麻醉药物、使用开睑器及角膜情况（水肿、混浊、生物力学性能及中央角膜厚度等）。鉴于此，在评估儿童青光眼的所有参数中，眼压是最不精确且最易变化的指标，因此不能仅根据眼压升高作出儿童青光眼的诊断，而应根据总体临床表现和检查结果进行判断[5-7]。

2.结构和功能评估：视盘形态是儿童青光眼诊断和进展评价的最重要且敏感的指标。抗青光眼手术后，随着眼压下降，视杯的直径和深度一般会较术前缩小，但视网膜神经纤维层厚度（retinal nerve fiber layer thickness，RNFLT）不可逆转[8]。基于相干光层析成像术的研究结果显示，5~18岁健康人的平均RNFLT无明显差异，或随着年龄增长，RNFLT略微降低，其中颞侧RNFLT从出生至18个月下降35%，而后呈现22%上升，直至稳定[9]。儿童青光眼患者的RNFLT显著低于正常水平[10]。相干光层析血管成像术检查结果显示，PCG患者视盘周围以及黄斑区的血管密度、灌注密度、血流指数均低于健康人[11]。儿童青光眼患者在眼压控制不佳的情况下，眼部相关结构和功能呈现进展性损伤[12]。视野缺损的危险因素包括杯盘比增大和视网膜神经纤维层缺损[13]。

三、儿童青光眼的分子遗传学特点

PCG是儿童青光眼最常见类型，目前已知与PCG发病相关的基因包括CYP1B1、FOXC1、LTBP2和TEK[14]。其中CYP1B1是最早发现且研究最为深入的致病基因。PCG发病率存在明显的地域差异性，在PCG高发的种族或地区，家族性发病率高达40%，且多为常染色体隐性遗传模式，约50%~100%患者存在CYP1B1突变[15-16]。在我国，因为近亲结婚现象较少，大多数患者呈散发状态，家族性发病仅约为7%，存在CYP1B1突变者仅约占10%[17-19]。携带CYP1B1基因突变的患者，发病年龄更早，表型更严重，预后更差。此外，在中国PCG患者中已筛查到致病性TEK突变，但尚未筛查到致病性LTBP2突变[20-21]。

从疾病的发生层面而言，广义的眼前节发育不良（anterior segment dysgenesis，ASD）为一组疾病，包括PCG、Axenfeld-Rieger综合征、无虹膜、Peter异常以及其他ASD，这组疾病均是由于在胚胎期眼前节发育过程中基因发生突变，导致眼前节不同程度发育异常，在临床特征和致病基因方面有很多交叉重叠。临床特征包括角膜混浊、房角发育不良、虹膜根部前粘连、虹膜基质发育不良、虹膜缺损等。目前部分疾病或综合征存在较为明确的致病基因，如Axenfeld-Rieger综合征的致病基因包括PITX2、FOXC1、PAX6，无虹膜是因PAX6突变引起。而CYP1B1作为PCG的致病基因，在Peter异常、无虹膜患者中也可筛查到其致病性突变[22-25]（表1）。CPAMD8是近年新确定的ASD相关基因，与其他ASD相关基因相同，CPAMD8可引起广泛而多样的ASD表型[26]。

JOAG具有明显的遗传异质性。家族发病最常见的遗传模式是常染色体显性遗传，但少数患者也可表现为常染色隐性遗传。MYOC是JOAG最主要的致病基因，其次与JOAG相关的基因还有CYP1B1、LTBP2、OPTN、TBK1等[3]。

儿童青光眼合并非获得性全身疾病或综合征多数可检测出较为明确的致病基因突变，有助于疾病诊断。如合并Ⅰ型神经纤维瘤的致病基因是NF1，眼脑肾综合征的致病基因是位于X染色体的OCRL，马方综合征的致病基因是FBN1，Stickler综合征的致病基因有COL2A1、COL11A1等[27-30]。

分子遗传学检查可应用于遗传咨询和产前诊断，有助于最大限度降低患病者的出生概率。同时，基因检测可发现有患病风险的儿童，而早期发现疾病有助于确定监测策略并给予及时的治疗干预。然而，目前仅有约20%患者可

表 1　不同眼前节发育不良的临床特征及致病基因

眼前节发育不良	临床特征						致病基因				
	角膜混浊	角膜与虹膜或晶状体粘连	虹膜缺失	虹膜发育不良	瞳孔移位	房角发育异常或房角粘连	PITX2	FOXC1	PAX6	FOXE3	CYP1B1
原发性先天性青光眼				√		√		√			√
Axenfeld-Rieger 异常				√	√	√	√	√	√		
无虹膜	√		√	√		√			√		√
Peter 异常	√	√		√		√	√		√	√	√

注：√示阳性；表内空项示阴性

检测到确定的分子学病因，未来研究需要发现更多的致病基因，以提高儿童青光眼基因检测的诊断率[31]。

四、儿童青光眼的病理和发病机制

在前房角正常发育过程中，由于角巩膜与葡萄膜的生长速率不同，故对于角巩膜，睫状体出现相对后移。在胚胎 5 个月时，虹膜根部位于 Schwalbe 线水平；而在出生时，虹膜根部后移至巩膜突水平。PCG 患者的睫状体在后移过程中受到抑制而导致虹膜根部位置靠前，遮挡小梁网，形成未发育成熟的房角[32]。房角镜下可见高而平坦的虹膜，而无法窥见小梁结构，部分患者可看到密集的梳状韧带附着于 Schwalbe 线，这些均表明中胚叶组织尚未完全后退而残存。PCG 的病理学特征：(1)小梁网增厚、融合；(2)Schlemm 管发育不良；(3)Schlemm 管内壁、邻管组织的泡状结构减少或缺失；(4)小梁网细胞坏死、凋亡以及自噬[33-34]。

研究结果显示，34%JOAG 患者的房角结构正常，66%患者呈现不同类型的异常房角结构，包括虹膜根部前插、梳状韧带残留、房角结构不清，后者可能是因小梁网呈现紧致融合状态，导致小梁网结构难以辨认。此外，JOAG 患者可伴有虹膜纹理异常，如虹膜隐窝增多或减少[35-36]。

五、儿童青光眼的药物治疗

对于大多数类型的儿童青光眼，药物治疗难以维持长久疗效，一般仅作为等待手术过程中的控制眼压措施或手术后的辅助方法。而对于 JOAG、葡萄膜炎继发青光眼或白内障摘除手术后继发青光眼，药物治疗可作为一线治疗方法[37]。

目前缺乏足够的关于儿童青光眼药物治疗效果的临床试验证据。儿童患者可能发生成人罕见严重药物不良反应，且部分不良反应表现不典型。由于具有中枢性抑制高风险，α 肾上腺素能受体激动剂禁止婴儿使用，建议体重>18~20 kg 儿童使用。β 肾上腺素能受体阻滞剂可显著降低眼压，通常作为一线用药，但仍须谨慎防范 β 肾上腺素能受体阻滞剂可能导致的呼吸系统和心血管系统并发症，如夜间咳嗽、肺部感染加重等，用药前应与儿科医师沟通，以确保无心脏或呼吸系统禁忌证。碳酸酐酶抑制剂通常安全且耐受性良好，为了避免发生严重的代谢性酸中毒，局部用药优于全身用药。前列腺素类药物在婴幼儿青光眼早期的作用有限，但可作为辅助用药。前列腺素类药物可作为治疗 JOAG 的一线用药。缩瞳剂很少作为一线降眼压药物，但在房角相关手术后经常应用，以防止房角粘连[38-39]。

六、儿童青光眼手术治疗方法的选择

儿童青光眼大多需要进行手术治疗，甚至需要多次手术治疗，首次手术往往是获得长期成功最重要的机会。影响手术方法选择的因素很多，最为主要的因素是青光眼类型，其他因素包括青光眼的程度、手术医师的能力及经验、各种手术方法的适应证和特点、是否具备手术相关仪器设备以及患者的诉求等。

(一)房角手术

房角手术是 PCG 以及部分房角开放的继发性儿童青光眼的一线手术方法。

1. 房角切开术和传统小梁切开术：一直以来房角切开术和使用 Harms 小梁切开刀的传统小梁切开术是治疗 PCG 的主要手术方法。房角切开术依赖角膜透明度，在使用时受到限制，所以小梁切开术应用更为广泛[40]。小梁切开术的 10 年成功率可达 75.0%~76.5%，但往往需要多次手术[41-42]。

2. 微导管辅助或缝线辅助小梁切开术(全周切开或部切开分，经外路或经内路)：近 10 年微导管辅助小梁切开术(microcatheter-assisted trabeculotomy，MAT)广泛应用于治疗儿童青光眼。手术中发光微导管可持续提供导管位置信息，从而确保准确切开小梁网，可提高手术的成功率。作为首次手术方法，经外路 MAT 治疗 PCG 的成功率可达到 81%~90%，较传统小梁切开术更具有明显优势[43-46]。也可使用缝线对 Schlemm 管进行 360 °穿通后行全周小梁切开[47]，其手术成功率同样高于传统小梁切开术[48-50]。微导管辅助全周小梁切开与微导管辅助部分小梁切开具有同等效用。研究结果显示，抗青光眼手术失败患者，若可切开剩余未被破坏的小梁网，MAT 仍可获得较好的治疗效果，手术成功率可达到 77%~84%[51-52]。房角镜辅助下内路全周小梁切开术(gonioscopy-assisted transluminal trabeculotomy，GATT)与经外路 MAT 治疗角膜透明 PCG 患者的手术成功率相同，而 GATT 的手术操作创伤更小，不损伤结膜，最大限度保留了再次手术治疗的机会[53-56]。GATT 治疗 JOAG 同样具有较好的有效性和安全性，可作为首选手术治疗方法[57-60]。

3. 穿透性黏小管成形术：穿透性黏小管成形术是近年出现的手术方法，是在完成 Schlemm 管成形术的基础上于角巩膜缘开窗，使房水由前房或后房经开窗口直接进入 Schlemm 管断端，再经生理性房水流出通路进行引流。该

手术不受房角形态限制，在治疗原发性闭角型青光眼、虹膜角膜内皮综合征继发青光眼、外伤性房角后退继发青光眼等中取得了较好的手术效果[61-63]。在PCG和JOAG患者中，术后1年的相对手术成功率可达到89.19%[64]。

房角手术同样可用于治疗房角开放的白内障摘除手术后继发青光眼，但须谨慎防范相关并发症，如玻璃体积血、脉络膜脱离等[65]。房角手术还可用于治疗如早发型Sturge-Weber综合征相关青光眼等开角型儿童青光眼[66-67]。

以Schlemm管为基础的抗青光眼手术后常发生一过性高眼压，不同的研究者将其定义为术后眼压高于30 mmHg或高于21 mmHg。约74%JOAG患者在GATT术后出现一过性高眼压，且与手术失败具有相关性[60]。儿童青光眼较成人发生一过性高眼压的概率低[68]。

（二）小梁切开联合小梁切除术（combined trabeculectomy and trabeculotomy，CTT）

CTT可作为治疗儿童青光眼的首选手术方法[69-70]。对于原发性儿童青光眼，CTT具有确切的有效性和安全性[71-72]。然而，随着随访时间延长，手术成功率出现逐渐下降的趋势[73-75]。对于伴有前节发育异常的儿童青光眼，CTT的有效性显著降低[76]。比较性研究结果显示，CTT与单纯小梁切开术的手术成功率无明显不同[77-80]，而CTT较单纯小梁切除术的手术成功率高[81-82]；术中是否使用丝裂霉素，不影响手术成功率[83]。

（三）小梁切除术和引流管植入术

小梁切除术和引流管植入术主要用于治疗房角手术失败的儿童青光眼，也可用于首次手术治疗。由于儿童具有较强的生长能力和瘢痕反应，小梁切除术治疗儿童青光眼的效果并不十分理想，尤其对于年龄较小患者，手术成功率随时间延长而下降。应用丝裂霉素可一定程度增加手术效果，而相关并发症发生率也显著提高[76, 81, 84-86]。小梁切除术治疗继发性儿童青光眼的成功率与治疗原发性儿童青光眼相比，无明显差异[84, 87-88]。引流管植入术的整体成功率高于小梁切除术，5年手术成功率为59.0%~71.5%，对于需要进行有效外引流的患者，可作为首选手术方法，在治疗难治性儿童青光眼中具有重要地位。然而，引流管植入术相关并发症的发生率在26.7%~45.7%之间，主要包括引流管位置异常、低眼压、脉络膜脱离等。引流管植入术多用于治疗白内障摘除手术后继发青光眼、葡萄膜炎继发青光眼、Sturge-Weber综合征相关青光眼以及抗青光眼手术失败的青光眼[89-95]。

（四）非穿透深层巩膜切除术（non-penetrating deep sclerectomy，NPDS）

NPDS治疗儿童青光眼具有较好效果。与小梁切除术比较，NPDS具有相似的降眼压效果和手术成功率，而并发症较少[96-97]。CO_2激光辅助深层巩膜切除术（CO_2 laser-assisted sclerectomy surgery，CLASS）使用CO_2激光消融系统，可更精确、更容易制作巩膜池和消融Schlemm管，在降低眼压方面与传统NPDS相同[98]。CLASS在部分难治性儿童青光眼治疗中显示了良好的有效性，但治疗儿童青光眼的有效性和安全性尚需进行大样本观察研究[99-101]。

（五）睫状体手术

睫状体破坏性手术可作为各种治疗失败青光眼的最后治疗方法，或者用于手术风险高的患者，如白内障摘除手术后继发青光眼[102]。经巩膜途径的二极管激光睫状体光凝术很难做到精准定位，手术成功率较低，经常需要反复治疗和继续药物治疗。反复治疗可发生的并发症包括低眼压、严重的葡萄膜炎、巩膜软化、视网膜脱离等。内窥镜下睫状体激光光凝术可提高手术操作的准确性，用于治疗儿童白内障摘除手术后继发青光眼可获得中等手术效果[103]。微脉冲二极管激光睫状体光凝术是近年基于激光发射探针改良的新技术。传统的激光发射探针可输出连续高强度能量激光，而微脉冲激光是发出一系列重复的短脉冲能量，脉冲之间的休息时间称为热松弛时间。将连续的激光能量波分割成数个微脉冲以产生效果，可减轻不良反应。微脉冲二极管激光睫状体光凝术治疗抗青光眼手术失败的儿童青光眼，具有较好的有效性和安全性[103-104]。

（六）选择性激光小梁成形术（selective laser trabeculoplasty，SLT）

SLT可有效降低JOAG的眼压，其具有简单、安全、可重复等优势，可作为治疗JOAG的首选方法，或者药物治疗失败青光眼的手术治疗方法。SLT可使JOAG患者推迟行其他抗青光眼手术或减少手术次数[105]。

（七）微创青光眼手术

在微创青光眼手术中，部分新的手术方法可尝试用于治疗儿童青光眼，如Kahook双刃刀内路小梁切除术、XEN凝胶引流管植入术、Preserflo青光眼引流器植入术等，其有效性和安全性尚需要进行大样本长期观察，该类手术尚不能取代房角手术[106-112]。

七、儿童青光眼的综合治疗

儿童时期是视觉功能发育的重要时期，在积极控制眼压的同时，应重视儿童青光眼的综合治疗，及时矫正屈光不正，进行适当的弱视训练，验配视残儿童助视器等，最大限度改善视功能预后[113]。应从整体角度对儿童青光眼的视功能、视觉发育、视觉行为进行评估，如角膜瘢痕、眼球震颤、斜视、弱视等对视功能的影响。此外，伴有眼球扩张者终身有发生视网膜脱离的风险，应避免眼外伤，并定期进行详细的眼底检查，必要时行预防性治疗。

利益冲突　所有作者声明无利益冲突

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