

DOI: 10.14188/j.1671-8852.2025.0317

文章编号 1671-8852(2026)04-0557-07

脉络膜血管因素在闭角型青光眼发病中的机制研究进展

金心怡¹ 陈伟²

¹浙江中医药大学绍兴基地眼科 浙江 绍兴 312000;

²绍兴市人民医院眼科 浙江 绍兴 312000

摘要 原发性闭角型青光眼(PACG)是一种不可逆性导致失明的多因子视神经疾病,与房角关闭导致的急性或慢性眼压升高有关,发病机制较为复杂,涉及多种因素。脉络膜富含丰富的血管网络,是视网膜神经的主要血供来源,对视神经具有重要调节作用。近年来,PACG发病机制中的脉络膜血管扩张学说逐渐受到临床关注,本文将就PACG发病现状、脉络膜血管因素在PACG发病中的相关机制进行综述,并针对脉络膜血管因素的治疗策略及应用前景展开探讨,以期PACG的临床防治提供新的思路。

关键词 闭角型青光眼;脉络膜血管;视网膜神经

中图分类号 R775

文献标志码 A

Research progress on mechanism of choroidal vessel factor in the pathogenesis of angle-closure glaucoma

JIN Xinyi¹, CHEN Wei²

¹Department of Ophthalmology, Zhejiang University of Traditional Chinese Medicine Shaoxing Base, Shaoxing 312000, Zhejiang, China;

²Department of Ophthalmology, Shaoxing People's Hospital, Shaoxing 312000, Zhejiang, China

Abstract

Primary angle-closure glaucoma (PACG) is an irreversible multi-factor optic nerve disease that causes blindness, and it is related to acute or chronic intraocular pressure elevation caused by angle closure. Its pathogenesis is complex and involves many factors. The choroid has a rich vascular network, which is the main source of blood supply to the retinal nerve and plays an important role in regulating the optic nerve. In recent years, the theory of choroidal vasodilation in the pathogenesis of PACG has gradually attracted clinical attention. This paper reviews the current status of PACG and the related mechanisms of choroidal vascular factors in the pathogenesis of PACG, and discussed the treatment strategies and application prospects of choroidal vessel factor, in order to provide new insights for the clinical prevention and treatment of PACG.

Key Words Angle-Closure Glaucoma; Choroidal Vessel; Retinal Nerve

原发性闭角型青光眼(primary angle-closure glaucoma, PACG)好发于30岁以上人群,是临床上

导致失明的主要疾病之一,房角关闭是其发病的基本病理过程,但具体发病机制仍尚未完全阐明^[1]。

课题来源:2024年度浙江省医药卫生计划项目(编号:2024KY1718)。

作者简介:金心怡,女,2000—,医学硕士生,主要从事青光眼前临床治疗研究,E-mail:17857314687@163.com。

通信作者:陈伟,E-mail:chennwei@hotmail.com。

眼部成像技术的不断发展使得 PACG 眼部结构变化的临床研究有了新的进展,由此而发现的虹膜、晶状体、睫状体等结构变化为 PACG 的发病机理研究提供了便捷。目前,临床上已发现的致病机制包括瞳孔阻滞机制、高褶虹膜机制、晶状体诱发性机制等,为 PACG 的临床治疗提供了重要参考^[2-3]。随着研究的不断深入,脉络膜结构及功能在 PACG 发病中的作用逐渐受到关注。作为高度血管化的组织,脉络膜在视网膜供血中发挥主要作用,脉络膜血管扩张学说认为,脉络膜体积变化对玻璃体、晶状体的压迫是造成房角关闭的重要原因,然而,引起脉络膜体积增加的相关因素尚待进一步探究^[4]。

1 PACG 的发病现状及机制探究

PACG 是临床上不可逆的致盲性视神经疾病,其主要是由于房角关闭引起眼压急性升高所致,以视野缺损、视网膜神经纤维层缺失为主要病理特征,临床表现为眼睛胀痛、视力模糊、眼压升高、眼红等^[5-6]。目前,全球范围内约有超过 6 000 万的青光眼患者,其发病仍呈上升趋势,据世界卫生组织估计,到 2030 年约有超过 9 000 万人次发展为青光眼,2040 年则可能超过 1.1 亿人次患病^[7-9]。PACG 是青光眼的临床常见病理类型,亚洲女性被认为是 PACG 发病的高危人群,我国作为青光眼发病大国,农村人群中青光眼患病率为 1.7%,其中 PACG 占比高达 44.4%^[10]。目前,青光眼已成为威胁公众健康的重要疾病之一而备受临床关注,探究引起 PACG 发病的相关机制对临床疾病预防和治疗意义重大。关于 PACG 发病的机制猜想较多,涉及解剖机构、遗传因素、心理应激因素等,其中,解剖结构相关的机制探究较多,包括高褶虹膜机制、晶状体诱发性机制、玻璃体韧带假说、脉络膜膨隆学说等^[11-12]。根据《中国青光眼指南(2020 年)》^[13]分类,原发性闭角型青光眼的临床类型包括单纯瞳孔阻滞型、虹膜高褶型、睫状体前位型、晶状体位置异常型及脉络膜膨隆型。随着研究的不断深入,眼部血流因素对视网膜神经调节的作用逐渐受到重视,其中脉络膜因素是近年来探讨的热点。

2 脉络膜血管因素与 PACG 发病的关系

2.1 脉络膜的解剖结构与功能 脉络膜是位于视网膜和巩膜之间的一层柔软、光滑、具有弹性的棕色薄膜,其范围从视网膜锯齿缘延伸至整个视神经

周围,覆盖于眼球后部。脉络膜组织包括丰富的血管网络、黑色素细胞、成纤维细胞等,其中,脉络膜血管层是脉络膜的重要组成部分,包含毛细血管层、中型血管层和大血管层,整个血管网络与视网膜表面高度吻合,并在其表面形成密集的三维血管网络^[14-16]。脉络膜血流量占整个眼球总血流量的 85%,为视网膜外层提供能量和氧气,保证 Bruch 膜与视网膜之间的代谢交换^[17]。脉络膜也因其重要的解剖位置和丰富的血管系统在视神经功能中发挥着重要作用,如为视网膜外层提供营养、参与眼内液体的产生和排出、调节眼内温度等。以往由于研究未深入或疾病检测技术有限等原因,无法获取清晰的脉络膜断层图像,其对 PACG 发病的相关机制也未进行深入剖析,随着临床影像学技术的发展,扫频源光学相干断层扫描血管成像可清晰呈现脉络膜血管,以脉络膜膨隆学说为基础的 PACG 发病机理研究被广泛探讨^[18-19]。脉络膜膨隆学说的主要内容就是脉络膜血管在 PACG 发病中的变化情况,探究脉络膜血管因素影响 PACG 发病的潜在机制对早期 PACG 的诊断和治疗具有重要意义。

2.2 影响 PACG 发病的相关脉络膜血管因素 脉络膜是一个高度血管化的组织,其血管因素对脉络膜功能发挥起着决定性作用,临床上常见的血管因素包括脉络膜血流和脉络膜血管内皮功能两个方面。

2.2.1 脉络膜血流

(1)脉络膜血流灌注:脉络膜血流是视网膜血流灌注的主要来源,其充盈程度对眼球总供血量关系密切,影响脉络膜充盈程度的因素主要包括血管灌注压和血流阻力,脉络膜血管可根据灌注压的升高或降低进行血管收缩或舒张调节,以保证血流灌注在一定范围内稳定存在,这种现象称之为脉络膜血管的自我调节能力^[20-21]。SHIN 等^[22]研究显示,脉络膜血管密度减少、血流灌注降低可引起眼部自主神经功能障碍,进一步引起青光眼发病。脉络膜血流阻力与血管硬化有关,随着年龄增大脉络膜血管容易发生硬化,进而引起脉络膜血流阻力升高,血流量降低^[23]。研究^[24]显示,脉络膜血流量降低可引起青光眼患者眼压升高,改善脉络膜血流可缓解青光眼症状,提示脉络膜血流可通过影响眼压引起青光眼发病。

(2)脉络膜厚度:临床上评估脉络膜血流的常见指标是脉络膜厚度,脉络膜血管损伤、灌注异常均可导致脉络膜厚度变化,因而,脉络膜厚度可一

定程度上反映脉络膜血流灌注情况^[25]。此外,脉络膜还可根据脉搏变化同步增厚或变薄来辅助调节眼压,研究^[26]显示,PACG患者脉络膜厚度显著升高,且随着脉络膜厚度增加,患者眼压逐渐升高,预后不良风险增加。分析其原因,青光眼主要临床特点是视网膜神经纤维层变薄,患者脉络膜血流异常可引起眼压升高,而长期过高的眼压可压迫视网膜神经,引起视神经受损,导致视网膜神经纤维层变薄,进而加重青光眼病情^[27]。目前,关于脉络膜厚度与PACG发病之间的关系尚未完全阐明,有研究^[28-29]认为脉络膜厚度增加可压迫玻璃体、晶状体,造成晶状体向前移动引起瞳孔阻滞,进而造成房角关闭,最终导致PACG发病。此外,眼部不同位置的脉络膜厚度也存在差异^[30]。除分布差异外,脉络膜还受自身节律的影响,由于脉络膜和视网膜眼外段的血管有丰富的自主神经,刺激交感神经可引起脉络膜血管扩张,如交感神经受温度和光照刺激,可引起脉络膜血管扩张,导致脉络膜厚度增加,但正常生理状态下的眼压、血压波动并不能使脉络膜厚度明显变化,脉络膜根据眼压、灌注压力变化调节自身血流,以满足视网膜的血供需求,而当眼部处于炎症、创伤状态时,负责提供视网膜血流的脉络膜血管血流速度降低,循环血量及氧气供应无法满足视网膜正常的代谢需求,导致其功能下降,进而影响视觉神经^[31]。由此可见,脉络膜增厚推动晶状体前移引起的房角关闭可导致PACG;相反,PACG发病时的炎症、眼压升高等特征亦会反向导致脉络膜血流灌注降低,引起视网膜功能下降,加重患者病情,形成恶性循环^[32]。脉络膜厚度与眼灌注压之间并非单一的线性关系,因而,脉络膜血流变化在PACG发病过程中的机制尚待更多研究进一步探究。

但在PACG的研究中,脉络膜血流的变化存在一定争议。急性闭角型青光眼(acute primary angle-closure glaucoma, APAC)发作时,眼压急剧升高可能导致脉络膜血管代偿性扩张,相关研究^[33]通过增强深度成像光学相干断层扫描发现,APAC发作眼的脉络膜厚度显著高于正常眼及对侧未发作眼,提示脉络膜血管扩张或渗出导致血容量增加,与情绪应激或交感神经兴奋可能通过释放儿茶酚胺类物质,引发脉络膜血管短暂性充血有关,这种充血可能进一步导致玻璃体腔压力升高,推动晶状体-虹膜隔前移,加剧房角关闭。也有部分研究^[34]显示,PACG患者的视盘旁血管密度较正常组下降,

且与视野缺损程度呈正相关,这种改变可能与长期眼压升高引起的血管内皮损伤及自主神经调节失衡有关,且光学相干断层扫描血管造影(optical coherence tomography angiography, OCTA)定量分析表明,慢性闭角型青光眼患者黄斑区深层毛细血管丛的血流密度显著降低,提示脉络膜血流从急性期的充血状态向慢性缺血状态转化。因此,未来需通过纵向研究联合多模态影像技术[如OCTA+超声生物显微镜(ultrasound biomicroscopy, UBM)同步监测],明确血流变化的时空动态规律。

2.2.2 血管内皮功能障碍 PACG是以视神经损伤和视野缺损为主要特征的不可逆眼疾,脉络膜作为眼球的血管层,在视网膜血液循环、视神经功能中发挥重要的作用。近年来,除血流调节引起的眼压及供血问题外,血管内皮功能变化引起的血液灌注异常在PACG发病中的相关机制也日益受到关注^[35]。作为脉络膜血管的重要组成部分,脉络膜内皮细胞在维持脉络膜正常功能中发挥重要作用,如通过调节血管通透性、血流量及新生血管生成等过程确保视网膜可得到正常的血液供应,以维持其正常的视神经功能^[36]。血管内皮功能障碍主要是由于血管内皮细胞在病理因素的刺激下所表现出的功能紊乱状态,临床上血管内皮功能紊乱可引起血管通透性增加、血流灌注异常、血管新生失衡等,这些改变均可不同程度地影响脉络膜供血功能,造成眼内压变化及视网膜功能异常^[37-38]。脉络膜功能障碍引起PACG发病的机制较为复杂,大体上可分为以下几种途径:(1)脉络膜血管内皮损伤可引起眼内压升高。正常生理状况下,脉络膜通过参与眼内房水的生成和排出维持眼内压稳定,当脉络膜血管内皮损伤时,其通透性增加,对房水的重吸收功能降低,眼内房水排出受阻可引起眼压增高,进而压迫视网膜神经,造成房角关闭,引发PACG^[39-40]。(2)脉络膜血管内皮障碍可引起视神经损伤。炎症或外伤引起的脉络膜损伤可引起黄斑区水肿或出血,引发视物变形或颜色异常,脉络膜小血管炎症引起的出血还可引起玻璃体腔内积血,进而引起视网膜炎症。此外,脉络膜血管内皮损伤还可能引起视网膜血流灌注降低,导致其发生缺氧性损伤^[41-42]。(3)脉络膜内皮功能障碍可能促进视网膜神经节的凋亡。脉络膜主要为视网膜外层提供营养,促进视网膜神经细胞生长,而内皮细胞功能障碍可导致视网膜营养供给不足,进而加快神经节细胞的凋亡,引发视神经损伤和视野缺损,进而加快PACG发

病^[43-44]。目前,关于脉络膜血管内皮功能障碍的临床研究尚少,其在PACG发病中的作用机制尚待进一步探究。

2.2.3 其他因素影响脉络膜血流 尽管多数研究支持脉络膜血管因素参与PACG进展,但需警惕混杂因素的干扰:

(1)系统性高血压:系统性高血压可导致脉络膜血管壁增厚、管腔狭窄、血流阻力增加,相关研究^[45]发现,合并高血压的闭角型青光眼患者,其脉络膜毛细血管密度较无高血压患者更低,这可能掩盖疾病本身导致的缺血性改变。

(2)糖尿病:糖尿病患者的脉络膜血管通透性异常,表现为血管渗漏与血流分布不均,此类患者即使处于闭角型青光眼慢性期,其脉络膜厚度可能因渗出性增厚而被误判为充血状态^[46]。

(3)降眼压药物:部分降眼压药物或可产生双向影响,如前列腺素类药物(如拉坦前列素)可通过增加葡萄膜巩膜途径房水排出,间接减少脉络膜静脉回流压力,使血流速度降低; β 受体阻滞剂(如噻吗洛尔)可能通过抑制交感神经活性,减少脉络膜血管自主调节能力,导致昼夜血流波动幅度扩大^[47]。

(4)生活方式和环境因素:情绪波动、过度劳累、长时间处于暗室、大量饮水等生活方式和环境因素也可能诱发PACG,影响脉络膜血流^[48]。

由于上述多种因素的影响,临床上对脉络膜血流的测量和研究可能存在一定的局限性,不同的研究方法、样本大小、测量技术和时间点的选择都可能导致研究结果的差异。因此,未来可结合多种影像学技术,如超声多普勒、磁共振成像(Magnetic Resonance Imaging, MRI)和光学相干断层扫描(Optical Coherence Tomography, OCT)等,以获得更全面、准确的脉络膜血流信息,开展大规模的纵向研究,跟踪PACG患者不同阶段的脉络膜血流变化,以更准确地评估其在疾病发展中的作用。

3 针对脉络膜血管因素的早期筛查、治疗策略及应用前景

3.1 早期筛查 脉络膜血管因素在PACG的早期筛查中具有重要意义,通过监测脉络膜血管的解剖结构和血流动力学变化,可尽早发现潜在的房角关闭风险。目前,早期筛查方法主要包括以下几种:超声多普勒可用于评估脉络膜血流速度和血管阻

力,检测血流动力学异常;MRI提供高分辨率的脉络膜血管图像,帮助识别解剖异常;OCT用于评估脉络膜厚度和血管结构,检测早期的脉络膜改变;UBM用于评估睫状体厚度、脉络膜突形态。通过早期干预,可有效控制病情发展,减少视力损失的风险^[49-50]。对无症状高危人群(如有浅前房家族史者)进行脉络膜评估,可提前识别PACG风险,通过激光虹膜切开术使房角关闭发生率降低^[51]。

3.2 治疗策略 脉络膜血管因素在PACG发病中发挥重要作用,针对血管因素的治疗策略是目前及今后临床研究的重点。目前,关于PACG的治疗主要包括药物治疗、激光治疗、手术治疗三个方面。药物治疗方面,房角关闭导致脉络膜改变引发的PACG可通过使用前列腺素类药物、血管扩张剂等改善血流,降低眼内压,采用抗炎药(如皮质类固醇、非甾体抗炎药)等改善脉络膜炎症,进而降低脉络膜血管内皮损伤^[52-53]。激光治疗方面,主要针对房角关闭 $>180^\circ$ 的急性PACG患者,通过调节激光参数,可在不损伤视网膜的情况下有效降低眼压、加深前房、开放房角、改善脉络膜血管通透性和血液循环,如针对脉络膜血管通透性增加,可选择微脉冲激光疗法进行改善;针对脉络膜血流降低可通过激光小梁成形术进行改善^[54-55]。手术治疗方面,目前无直接针对脉络膜的手术,手术治疗主要通过改善眼压促进脉络膜血流恢复,如针对药物降眼压效果不明显者,可通过摘除透明晶状体降低其对视网膜的压迫改善眼压、加深前房深度,改善脉络膜血流,缓解PACG症状^[56]。针对小梁网与虹膜粘连的患者,可通过超声乳化摘除(phacoemulsification, Phaco)+人工晶状体(intraocular lenses, IOL)植入联合房角分离术解除粘连,缓解瞳孔阻滞及加深前房^[57]。虽然关于脉络膜血管的临床治疗尚在研究阶段,但阐明其在PACG发病中的相关机制将为PACG的临床治疗提供新的视野。

4 总结与展望

PACG是多种机制引起的不可逆眼部疾病,其发病涉及多种因素,不仅局限于眼球解剖结构,还涉及视神经、视觉通路、肌源性、血流等多个方面。脉络膜是近年来发现的新的致病因素,作为视网膜血供的主要来源,脉络膜血流、血管内皮功能在PACG的发生、发展中起着重要作用。目前,多数研究认为脉络膜血管因素引起的血流及血管通透

性改变是PACG的重要发病机制之一,但针对上述观点的研究仍处于进行状态,未来脉络膜血管因素能否作为PACG发病及病情变化的重要监测指标服务于临床诊断及临床治疗参考仍需进一步研究。



开放科学(资源服务)标识码(OSID): 

参考文献

- [1] WANG Y, DONG X X, HOU X W, et al. Risk factors for primary angle-closure glaucoma: A systematic review and meta-analysis of 45 studies[J]. *Optom Vis Sci*, 2023, 100(9): 606-613.
- [2] KADER M A, PRADHAN A, SHUKLA A G, et al. Lowering of intraocular pressure after phacoemulsification in primary open-angle and angle-closure glaucoma: Correlation with lens thickness[J]. *Indian J Ophthalmol*, 2022, 70(2): 574-579.
- [3] CHEN J W, ZHANG Y, HUANG Y Q, et al. Relatively anterior lens position in primary angle-closure glaucoma eyes with long axial length[J]. *Indian J Ophthalmol*, 2023, 71(5): 1941-1947.
- [4] AYUB G, CAMPOS B M, REZENDE T J R, et al. Measurement of retina/choroid complex perfusion with magnetic resonance imaging in eyes with acute primary angle-closure[J]. *Arq Bras Oftalmol*, 2021, 85(2): 166-173.
- [5] SONG Y H, ZHANG H L, ZHANG Y Z, et al. Minimally invasive glaucoma surgery in primary angle-closure glaucoma[J]. *Asia Pac J Ophthalmol*, 2022, 11(5): 460-469.
- [6] ALVAREZ-GUZMAN C, VALDEZ-GARCIA J E, RUIZ-LOZANO R E, et al. High prevalence of angle-closure glaucoma in Vogt-Koyanagi-Harada disease[J]. *Int Ophthalmol*, 2022, 42(12): 3913-3921.
- [7] VAN ZYL T, YAN W J, MCADAMS A, et al. Cell atlas of aqueous humor outflow pathways in eyes of humans and four model species provides insight into glaucoma pathogenesis[J]. *Proc Natl Acad Sci USA*, 2020, 117(19): 10339-10349.
- [8] KURYSHEVA N I, SHAROVA G A. Primary anterior chamber angle closure: Progression from suspect to glaucoma. Part 1. Frequency and rate of transition from suspected primary angle closure to true angle closure and primary angle closure glaucoma[J]. *Vestn Oftalmol*, 2022, 138(4): 101-107.
- [9] ABDELAAL A, HASSAN A R, KATAMESH B E, et al. The incidence and presentation features of glaucoma in Vogt-Koyanagi-Harada syndrome: A systematic review and meta-analysis[J]. *Ophthalmol Glaucoma*, 2024, 7(6): 587-601.
- [10] TEO Z L, SOH Z D, THAM Y C, et al. Six-year incidence and risk factors for primary angle-closure disease: The Singapore epidemiology of eye diseases study[J]. *Ophthalmology*, 2022, 129(7): 792-802.
- [11] CĂLUGĂRU D, CĂLUGĂRU M. Etiology, pathogenesis, and diagnosis of neovascular glaucoma[J]. *Int J Ophthalmol*, 2022, 15(6): 1005-1010.
- [12] LAM S S, AMARASEKERA D C, RAZEGHINE-JAD R. Chronic angle closure glaucoma with an atypical iris-pupillary membrane[J]. *Eur J Ophthalmol*, 2023, 33(4): NP37-NP41.
- [13] 中华医学会眼科学分会青光眼学组,中国医师协会眼科医师分会青光眼学组. 中国青光眼指南(2020年)[J]. *中华眼科杂志*, 2020, 56(8): 573-586. Glaucoma Group of Society of Ophthalmology of Chinese Medical Association, Glaucoma Group of Society of Ophthalmologist of Chinese Medical Doctor Association. China glaucoma guide (2020)[J]. *Chin J Ophthalmol*, 2020, 56(8): 573-586.
- [14] LIU Y L, WANG L J, XU Y Y, et al. The influence of the choroid on the onset and development of myopia: From perspectives of choroidal thickness and blood flow[J]. *Acta Ophthalmol*, 2021, 99(7): 730-738.
- [15] WANG Y M, HUI V W K, SHI J, et al. Characterization of macular choroid in normal-tension glaucoma: A swept-source optical coherence tomography study[J]. *Acta Ophthalmol*, 2021, 99(8): e1421-e1429.
- [16] GUO Y Z, HUANG S Y, XU S S, et al. Choroidal microvasculature dropout in glaucoma[J]. *Semin Ophthalmol*, 2023, 38(7): 610-616.
- [17] KIM J A, SON D H, LEE E J, et al. Intereye comparison of the characteristics of the peripapillary choroid in patients with unilateral normal-tension glaucoma[J]. *Ophthalmol Glaucoma*, 2021, 4(5): 512-521.
- [18] GU X W, ZHAO X Y, ZHAO Q, et al. Recent advances in imaging polypoidal choroidal vasculopathy with swept-source optical coherence tomography angiography[J]. *Diagnostics*, 2023, 13(14): 2458.
- [19] SHOJI T, KANNO J, WEINREB R N, et al. OCT angiography measured changes in the foveal avascular zone area after glaucoma surgery[J]. *Br J Ophthalmol*, 2022, 106(1): 80-86.
- [20] KIYOTA N, SHIGA Y, OMODAKA K, et al. The relationship between choroidal blood flow and glaucoma progression in a Japanese study population[J]. *Jpn J Ophthalmol*, 2022, 66(5): 425-433.
- [21] MUIR E R, CHANDRA S B, NARAYANAN D, et al. Effects of chronic mild hyperoxia on retinal and choroidal blood flow and retinal function in the DBA/2J mouse model of glaucoma[J]. *PLoS One*, 2022, 17(3): e0266192.

- [22] SHIN D Y, HONG K E, LEE N Y, et al. Association of choroidal blood flow with autonomic dysfunction in patients with normal tension glaucoma[J]. *Sci Rep*, 2022, 12(1): 5136.
- [23] KIYOTA N, SHIGA Y, TAKAHASHI N, et al. Progression in open-angle glaucoma with myopic disc and blood flow in the optic nerve head and peripapillary chorioretinal atrophy zone[J]. *Ophthalmol Glaucoma*, 2020, 3(3): 202-209.
- [24] KIYOTA N, SHIGA Y, NINOMIYA T, et al. The effect of β -blocker eye drops on pulse rate, ocular blood flow, and glaucoma progression: A retrospective longitudinal study[J]. *Adv Ther*, 2024, 41(2): 730-743.
- [25] BAYRAKTAR S, İPEK A, TAKMAZ T, et al. Ocular blood flow and choroidal thickness in ocular hypertension[J]. *Int Ophthalmol*, 2022, 42(5): 1357-1368.
- [26] HUANG H L, WANG G H, NIU L L, et al. Three-dimensional choroidal vascularity index and choroidal thickness in fellow eyes of acute and chronic primary angle-closure using swept-source optical coherence tomography[J]. *Int J Ophthalmol*, 2024, 17(1): 42-52.
- [27] ANSHUKITA, KHURUM F, KARKHUR S, et al. Comment on: Correlation between choroidal thickness and intraocular pressure control in primary angle-closure glaucoma[J]. *Indian J Ophthalmol*, 2022, 70(8): 3160-3161.
- [28] HUANG W B, LI X Y, GAO X B, et al. The anterior and posterior biometric characteristics in primary angle-closure disease: Data based on anterior segment optical coherence tomography and swept-source optical coherence tomography[J]. *Indian J Ophthalmol*, 2021, 69(4): 865-870.
- [29] LUO S R, YUAN G M, ZHAO C W, et al. Changes in anterior chamber angle and choroidal thickness in patients with primary angle-closure glaucoma after phacogoniosynechialysis[J]. *J Clin Med*, 2023, 12(2): 406.
- [30] NADEEM S. Choroidal thickness in juvenile open angle glaucoma: Insights from a south Asian case-control study[J]. *Graefes Arch Clin Exp Ophthalmol*, 2024, 262(10): 3295-3304.
- [31] ABDOLRAHIMZADEH S, DI PIPPO M, CIANCIMINO C, et al. Choroidal vascularity index and choroidal thickness: Potential biomarkers in retinitis pigmentosa[J]. *Eye*, 2023, 37(9): 1766-1773.
- [32] PEGU J. Response to comments on: Correlation between choroidal thickness and intraocular pressure control in primary angle-closure glaucoma[J]. *Indian J Ophthalmol*, 2022, 70(8): 3161.
- [33] TSUCHIYA S, MAKINO S. Transient optic disc swelling after laser iridotomy for the treatment of acute angle closure glaucoma[J]. *Cureus*, 2024, 16(3): e55765.
- [34] MOHAMMADI M, MOHAMMADI S S, TABATABAEI S M, et al. Comparison of vascular density and structural patterns between primary open angle glaucoma and primary angle closure glaucoma[J]. *J Glaucoma*, 2022, 31(8): 645-650.
- [35] VOIGT A P, MULLIN N K, MULFAUL K, et al. Choroidal endothelial and macrophage gene expression in atrophic and neovascular macular degeneration[J]. *Hum Mol Genet*, 2022, 31(14): 2406-2423.
- [36] NG D S C, FUNG N S K, YIP F L T, et al. Ranibizumab for myopic choroidal neovascularization[J]. *Expert Opin Biol Ther*, 2020, 20(12): 1385-1393.
- [37] BRINKS J, VAN DIJK E H C, KIELBASA S M, et al. The Cortisol response of male and female choroidal endothelial cells: Implications for central serous chorioretinopathy[J]. *J Clin Endocrinol Metab*, 2022, 107(2): 512-524.
- [38] DI STASO F, DI PIPPO M, ABDOLRAHIMZADEH S. Choroidal neovascular membranes in retinal and choroidal tumors: Origins, mechanisms, and effects [J]. *Int J Mol Sci*, 2023, 24(2): 1064.
- [39] BREWINGTON B Y, KONDAPALLI S, KOTHARI S S, et al. Choroidal effusion mimicking uveal melanoma: A novel presentation of idiopathic systemic capillary leak syndrome[J]. *Ocul Oncol Pathol*, 2021, 7(6): 390-395.
- [40] HOVEN E, MICHELET J T, VETTORE M V, et al. Choroidal thickness after anti-vascular endothelial growth factor in typical neovascular age-related macular degeneration – A systematic review and meta-analysis [J]. *Surv Ophthalmol*, 2025, 70(1): 86-95.
- [41] CARPEL E F, SCHEURER R A. Choroidal hemorrhage, preplaced sutures, and host-over-donor keratoplasty[J]. *J Cataract Refract Surg*, 2021, 47(12): e56-e59.
- [42] LIU B Y, LAI K B, MA Y, et al. Systemic risk factors for vitreous hemorrhage secondary to polypoidal choroidal vasculopathy[J]. *Ophthalmol Ther*, 2023, 12(5): 2769-2780.
- [43] YAMAGUCHI C, KIYOTA N, HIMORI N, et al. Differentiating optic neuropathies using laser speckle flowgraphy: Evaluating blood flow patterns in the optic nerve head and peripapillary choroid[J]. *Acta Ophthalmol*, 2025, 103(1): e49-e57.
- [44] KASHYAP S, SINGH L, KUMAR N, et al. Combined association of massive choroidal and optic nerve invasion as a prognostic relevance in primary retinoblastoma: A 10-year study[J]. *Asia Pac J Clin Oncol*, 2021, 17(2): e100-e108.
- [45] JUNG K I, KIM Y C, SHIN H J, et al. Nationwide cohort study of primary open angle glaucoma risk and

- cardiovascular factors among in Korean glaucoma suspects[J]. *Sci Rep*, 2025, 15(1): 1952.
- [46] ZHONG Z L, CHEN S. Indicating and predicting role of the horizontal C/D ratio in preclinical diabetic retinopathy associated with chronic angle-closure glaucoma[J]. *Int J Ophthalmol*, 2019, 12(2): 268-274.
- [47] YANG M C, LIN K Y. Drug-induced acute angle-closure glaucoma: A review[J]. *J Curr Glaucoma Pract*, 2019, 13(3): 104-109.
- [48] SUN J A, YUAN M, JOHNSON G E, et al. Comparison of structural and functional features in primary angle closure and open angle glaucomas[J]. *J Glaucoma*, 2024, 33(4): 254-261.
- [49] 程天, 郭瑞萍, 郭安琪, 等. 超声乳化白内障摘除联合 IOL 植入术后眼前节结构变化及相关性分析[J]. *国际眼科杂志*, 2024, 24(11): 1721-1727.
- CHENG T, GUO R P, GUO A Q, et al. Structural changes in anterior segment after phacoemulsification combined with intraocular lens implantation and correlation analysis[J]. *Int Eye Sci*, 2024, 24(11): 1721-1727.
- [50] 赵会芳, 赵春梅, 刘湘云, 等. mGCL 厚度、pRNFL 厚度及 BMO-MRW 对早期原发性闭角型青光眼的诊断价值[J]. *眼科新进展*, 2022, 42(3): 230-233.
- ZHAO H F, ZHAO C M, LIU X Y, et al. Significance of macular ganglion cell layer thickness, peripapillary retinal nerve fiber layer thickness and Bruch membrane opening-minimal rim width in diagnosis of early primary angle-closure glaucoma[J]. *Recent Adv Ophthalmol*, 2022, 42(3): 230-233.
- [51] 梁远波, 江俊宏. 我国青光眼防治问题与展望[J]. *浙江医学*, 2020, 42(22): 2377-2382.
- LIANG Y B, JIANG J H. Problems and prospects of glaucoma prevention and treatment in China[J]. *Zhejiang Med J*, 2020, 42(22): 2377-2382.
- [52] CHEN G, PENG X M, LI J, et al. Efficacy of brinzolamide in the initial management of acute primary angle closure: A randomized controlled trial[J]. *J Clin Pharm Ther*, 2022, 47(6): 792-797.
- [53] KUSUHARA S, NAKAMURA M. Ripasudil hydrochloride hydrate in the treatment of glaucoma: Safety, efficacy, and patient selection[J]. *Clin Ophthalmol*, 2020, 14: 1229-1236.
- [54] CHANG D S, JIANG Y Z, KIM J A, et al. Cataract progression after Nd: YAG laser iridotomy in primary angle-closure suspect eyes[J]. *Br J Ophthalmol*, 2023, 107(9): 1264-1268.
- [55] RAJ S, THATTARUTHODY F, YADAV D, et al. Long-term efficacy of selective laser trabeculoplasty in primary angle-closure disease after laser peripheral iridotomy[J]. *Semin Ophthalmol*, 2024, 39(3): 235-241.
- [56] COSTA V P, LEUNG C K S, KOOK M S, et al. Clear lens extraction in eyes with primary angle closure and primary angle-closure glaucoma[J]. *Surv Ophthalmol*, 2020, 65(6): 662-674.
- [57] BAYRAKTAR S, ERDAL B D Y, ALTAŞ F B, et al. The effects of lens extraction surgery on intraocular pressure and anterior segment parameters in primary angle-closure glaucoma[J]. *Turk J Ophthalmol*, 2024, 54(1): 32-37.

(2025-04-01 收稿)

编辑 沈建国