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病理性近视后巩膜葡萄肿的研究进展

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[摘 要] 病理性近视相对高度近视而言，更强调眼底并发症的存在。后巩膜葡萄肿(posterior staphyloma，PS)被认为是病理性近视的标志性特征，是眼球后巩膜壁的局部扩张，通常认为与巩膜变薄和脉络膜萎缩等因素相关。近年来研究认为PS的形成可能与局部炎症、Bruch膜缺失等因素密切相关。伴随OCT等检查技术的快速革新以及PS治疗手段的探索，诊断和治疗更加完善。

[关键词] 病理性近视；后巩膜葡萄肿；诊断进展；形成机制；治疗；综述

Research progress of posterior staphyloma in pathological myopia

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Abstract Pathological myopia emphasizes the existence of fundus complications. Posterior staphyloma, considered as a hallmark of pathological myopia, is a partial extension of the posterior scleral wall, which is believed to be related to the scleral thinning and choroidal atrophy. In recent years, studies have indicated that the formation of posterior staphyloma may closely connected with localized inflammation and defects of Bruch's membrane. With the rapid innovation of examination technologies such as optical coherence tomography (OCT) and the exploration of treatment for posterior staphyloma. The diagnosis and therapy will be more comprehensive in the future.

Keywords pathologic myopia; posterior staphyloma; diagnosis of progress; formation mechanism; treatment; review

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后巩膜葡萄肿(posterior staphyloma，PS)被定义为后巩膜壁变薄，局部扩张，扩张部分较较周围巩膜壁的曲率半径减小[1]。研究[2-3]认为病理性近视(pathologic myopia，PM)并发症中的脉络膜新生血管(choroidal neovascularization，CNV)、黄斑裂孔(macular hole，MH)、视网膜脱离(retinal detachment，RD)等和PS密切相关。当然，并非病理性近视都并发PS。Shinohara等[4]使用3DMRI检查方法报道病理性近视的PS检出率为12%~51%，差异可能与研究对象的屈光度、眼轴、年龄等有关。随着OCT等诊断技术革新，PS的检出率会有所变化。研究[5]认为PS与屈光度、眼轴、脉络膜萎缩程度呈正相关。近年来，对于PS的研究颇多，也是关注的热点，本文就PS的诊断进展、形成机制、治疗策略进行综述。

1 PS 诊断进展

PS可通过眼部B超、CT/MRI等手段诊断，其二维图像不能显示PS全貌，存在局限性[5]。随着三维MRI(3DMRI)成像技术的兴起，可观察整个PS的形状，比较直观[6]。但是3DMRI分辨率有限，不能区分PS的视网膜、脉络膜及巩膜组织，更不能显示各自的厚度，尤其位于视乳头及鼻侧的轻度PS，分辨能力有限。目前使用扫描源/光谱域OCT(swept-source/spectral-domain-OCT，SS/SD-OCT)可以清晰的显示PS部位的曲率变化以及眼球后部的大部分组织结构，1 310 nm SS-OCT相对840 nm光谱域OCT在检测较深组织结构上有优势，然而在分辨相对透明组织的结构时，2种不同的波长OCT表现均良好[7]。但由于激光穿透性的限制，不能显示PS的全部厚度，虽有不足，但可称上是对PS检查技术上的突破[5]。最近开发的广域扫描源OCT弥补了传统OCT的不足[8]，它使用多条扫描线，可三维重建23 mm ×20 mm ×5 mm深度的PS区域结构，且保持较高的分辨率，可以分辨出PS内组织间的异常变化程度[9]，更易对PS存在的位置及类型进行诊断，有取代3DMRI的趋势。

2 PS 的形成机制

PS形成机制较为复杂，至今仍不清楚。既往认为是巩膜本身发生病理性改变。眼部组织其他部位病理改变可能与PS的形成相互调控[10-11]。

2.1 PS 的脉络膜、眼轴因素

组织病理学证实病理性近视的PS区域的脉络膜明显变薄，血流量显著下降，脉络膜循环障碍导致巩膜缺氧，纤维细胞凋亡，巩膜外基质分解加速[5,12]。此外，有学者[13]推测脉络膜变薄导致缓冲作用减弱，眼内压对巩膜造成更直接和更大的压力负荷，与局部其他因素结合导致PS的形成。动物实验研究[14]也证实：单眼高度近视小鸡患眼较健眼的脉络膜明显变薄，可见脉络膜病变可能参与高度近视的形成。Fledelius等[15-18]研究了高度近视患者，认为随着年龄增长，屈光度增加，脉络膜变薄、萎缩更明显。

PS通常发生在双眼，认为单眼PS是严重的巩膜病理改变[19]。Wang等[20]报道发生PS的高度近视患者眼轴≥26.5 mm。PS多是后极部向后扩张，也可发生在中心凹外其他区域，发生在视轴以外的PS，眼轴有可能相对正常[5]。Ohno-Matsui等[6]研究了198名眼轴为30.0±2.3 mm (26.0~40.0 mm)的高度近视患者，约50%患者未检查出PS。提示眼轴和PS有时候是独立存在的[11,21]。

2.2 Bruch 膜因素

Bruch膜位于视网膜与脉络膜之间薄膜组织，前至锯齿缘，后至视乳头。Bruch膜病变、缺失可能与PS形成相关。Ohno-Matsui等[22]报道病理性近视出现PS后，黄斑区Bruch膜丢失。Jonas等[23-24]发现高度近视圆顶状黄斑附近可以检测到Bruch膜缺失，其他正常区域Bruch膜的厚度等特征无改变。同样伴有漆裂纹(Bruch膜破裂引起)的病理性近视与巩膜葡萄肿显著相关[2]。Jonas等[25]观察到非高度近视患者视网膜周边区域BM缺损后发生PS现象，推测玻璃膜具有稳定眼球壁形状的作用。Wang等[26]通过动物实验进一步说明了Bruch膜的作用，他们通过猪眼爆破试验发现Bruch膜破裂前能够承受82 mmHg(1 mmHg=0.133 kPa)的眼压，Bruch膜破裂后即使正常眼压，也观察到轻度巩膜葡萄肿的形成。

Cheng等[27]报道病理性近视(pathologic myopia，PM)并发黄斑部葡萄肿后易形成脉络膜新生血管(choroidal neovascularization，CNV)。这和伴有PS的PM患者具有较高的CNV发病率报道相一致[2]。推测黄斑部Bruch膜的缺失与PS的形成及CNV生长密切相关。

2.3 炎症因素

研究[28]显示：高度近视眼白细胞介素(IL-6)和基质金属蛋白酶-2(MMP-2)显著高于非高度近视眼，提示炎症因素可能和巩膜葡萄肿形成有关。Xu等[4]观察到视网膜色素膜炎患者存在PS现象，推测炎症因素与PS形成存在关联。Bhola等[29]报道3例外伤患者经睫状体部位激光光凝后，1例出现巩膜葡萄肿，1例巩膜变薄现象。组织学检查显示葡萄肿表面覆盖薄的结膜上皮、胶原和玻璃体冷凝体。随后Morales等[30]报道9例不同类型难治性青光眼，给予睫状体光凝，发现11只眼出现巩膜变薄，3只眼出现巩膜葡萄肿。Prata等[31]报道1名42岁男子在经过3次二极管激光透镜环形光电凝后，发生巩膜葡萄肿，随后在巩膜葡萄肿区域给予硬膜贴片加固后病情稳定，推测激光刺激巩膜产生炎症反应，导致Bruch膜受损，巩膜抗压作用较弱。随后有研究[32]在一些炎症性眼病(如Vogt-Koyanagi-Harada病)的治疗和随访中监测到巩膜葡萄肿的发生。总之，巩膜葡萄肿的形成和炎症存在关联，需要进一步研究，尤其在病理性近视患者开展研究。

2.4 眼压因素

PS形成可能和眼压有关。Park等[33-34]用SS-OCT检测伴有高眼压的高度近视眼经小梁切除术后，视乳头周围巩膜的形态随眼压降低而改变。Saka等[35]认为眼内压(intra-ocular pressure，IOP)可能是导致眼睛巩膜拉伸的机械因素。在伴有严重PS的病理性近视患者中，降低眼内压可导致PS的不均匀减轻，这表明PS形成可能受到IOP的影响。动物实验[36]也证实眼内压对病理性近视相关PS形成的影响，在诱导高度近视的豚鼠模型中，通过拉坦前列素滴眼液降低单眼眼内压，和对侧诱导眼比较，可显著减轻高度近视形成的程度及减少PS的发生。眼内压可能参与病理性近视PS的形成。

2.5 后巩膜解剖因素

PS的形成与巩膜的病理改变密切相关，正常巩膜以胶原纤维和板层纤维组成，胶原纤维占优势，PS巩膜以板层纤维为主，胶原纤维明显减少、稀疏，纤维间隙增宽[37]。有研究[18]认为几乎所有哺乳动物的巩膜都有肌成纤维细胞，且细胞数量相对稳定，肌成纤维细胞作为巩膜细胞的一部分，随着年龄增长而增加。肌成纤维细胞是由成纤维细胞分化而来，成纤维细胞可分泌巩膜基质参与巩膜的重塑。其中分化过程极其复杂，受巩膜发育过程中的张力及相关信号分子刺激调控，其中可明确的转化生长因子(transforming growth factor β，TGF-β)在转化中发挥重要作用。病理性近视的PS主要发生在后极部，与后极部的巩膜纤维排列有关，围绕视神经头和中央凹的巩膜胶原束大部分呈环状排列，会削弱局部组织对正常眼压的抵抗，增加巩膜后扩张的敏感性。而眼底其他区域，巩膜胶原纤维束的各向交叉排列可以缓冲眼内压力[28]。动物实验[5]证实高度近视豚鼠出现后巩膜变薄，胶原纤维排列稀疏等病理性改变。

有学者[4,38]根据病理性近视的PS发生部位及形状改变，将PS分为多种类型，位于黄斑部的PS最常见。巩膜葡萄肿位于眼底颞侧波及黄斑部，可导致黄斑与视盘间巩膜变薄，视盘不稳定，位置容易改变，出现视盘倾斜[39]。

2.6 PS和遗传因素

遗传因素在病理性近视中可能发挥重要作用，目前已发现超过20个染色体100个以上突变基因和人类高度近视相关联[40]。对于PS的独立遗传基因报道极少，PubMed以“posterior staphyloma”和“gene”为关键词搜索了12篇文章(时间为2021年3月30日)。8篇介绍与突变基因相关的综合症出现PS，例如：*RHO*基因的c.886A+G突变分离相关的视网膜色素膜炎病例中发现了PS的形成[41]；一个特定变异(BEST1-V239M)与MRCS(微角膜、杆状锥体营养不良、白内障和PS)综合征相关，这种突变可能和PS疾病相关[32,42]。2篇研究[43-44]分别介绍DNA杂交技术干扰巩膜纤维蛋白表达从而导致PS形成和包括PS体征在内的家族性综合征的相关DNA研究，其余则与PS形成无明显关联的介绍。总的来说，未检索到与PS形成直接相关联的独立遗传突变基因。

Ohno等[25]报道母亲(61岁)与女儿(35岁)的PS形态极其相似。PS多发于年龄较大的患者，如果通过突变基因预测PS风险及类型，可以在早期给予高危患者制订预防措施。同时为后期手术干预提供理论保障。可能为病理性近视PS的预测提供极其重要的防控策略。

3 PS治疗进展

PS的形成机制不明确，治疗方法通常是采取对症处理，国内学者[45-50]多采用后巩膜加固术。Zhu等[45]采用异体巩膜加固后巩膜，术后眼轴减少(2.15 ± 0.56) mm，巩膜形状趋于圆顶状。Li等[46]对16名单眼高度近视儿童接受后巩膜加固术治疗随访(术后3年)发现，巩膜加固组[(27.38±1.30) mm]与对照组[(28.29±0.74) mm]的巩膜轴长差异有统计学意义(P=0.03)，同时指出既往研究中，没有关注后巩膜加固术对PS本身的影响，有待进一步研究。

治疗方法除了后巩膜加固术外，有报道[22,24,51]用巩膜缩短术可以平复PS，目前还不清楚该种手术方式能将凸起的PS平复维持多久。巩膜胶原交联术主要是加强胶原纤维之间的结合能力，达到巩膜胶原硬化。目前巩膜胶原交联术的有效性已经在体外实验中得到报道[52-53]。Karl等[54-55]利用兔子模型实现巩膜胶原交联术可以稳定眼轴增长的作用，巩膜胶原交联对于人类明显变薄的巩膜是否有影响仍不清楚。近年来，Shinohara等[56]在大鼠近视模型中，将人成纤维细胞移植到大鼠后巩膜上后合成了I型胶原纤维。目前还不清楚这种方法是否可以用于加强PS区域的巩膜。总之，病理性近视PS的手术方式在不断的探索、完善。

4 结语

病理性近视PS的形成可引发一系列严重并发症，给患者的视功能带来不可逆性损伤，最终可能致盲。虽然PS的形成机制复杂，相关因素较多，但是人类对这一致盲眼病的研究从未停止。未来形成机制的进一步阐明，独立遗传基因的明确，可能对PS预防和控制带来更有力的保障。

开放获取声明

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