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·综述·

息肉状脉络膜血管病变的临床病理研究现状

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文章亮点

1. 关键发现

- 息肉状脉络膜血管病变 (polypoidal choroidal vasculopathy, PCV) 病灶的层次：部分病理学研究认为 PCV 病灶位于 Bruch 膜内，但实际上 PCV 病灶更准确地位于视网膜色素上皮 (retinal pigment epithelium, RPE) 基底膜下。
- BVN 的起源：尸体眼标本的病理分析表明，BVN 起源于脉络膜动脉，且动脉穿过 Bruch 膜后，转变为薄壁毛细血管形成 I 型脉络膜新生血管。少数研究指出 PCV 可能由静脉扩张形成。
- 血管内皮生长因子 (vascular endothelial growth factor, VEGF) 在 PCV 病灶中的表达：VEGF 是新生血管性 AMD 的关键致病因子，一些研究表明 PCV 病灶中 VEGF 表达升高，提示 PCV 可能与新生血管性年龄相关性黄斑变性的 (age-related macular degeneration, AMD) 具有相似的发病机制，但也有研究发现 PCV 病灶中的 VEGF 表达为阴性，提示 PCV 的机制可能不完全依赖于 VEGF。

2. 已知与发现

- 近年来的研究表明，除了传统的玻璃膜疣驱动机制外，PCV 可能与肥厚脉络膜机制相关，尤其在亚洲人群中更为常见。
- 与之相对应，PCV 的病理特征具有复杂性，既有与新生血管性 AMD 相似的表现，也有肥厚脉络膜的特征。

3. 意义与改变

- 随着眼球捐献意识的提高，未来有望获得更多宝贵的眼球标本。深入的病理学探索将有助于揭示 PCV 的发病机制，并探索 PCV 与其他脉络膜疾病之间的内在联系。

[摘　要]　息肉状脉络膜血管病变(polypoidal choroidal vasculopathy, PCV)是中国人新生血管性年龄相关性黄斑变性的(age-related macular degeneration, AMD)主要亚型。PCV与典型的新生血管性AMD在流行病学、临床表现、影像学特征和自然病程方面存在一定差异。近年来的研究表明，除了传统的玻璃膜疣驱动机制外，PCV可能与肥厚脉络膜机制相关，后者在亚洲人群中更为常见。深入的病理学探索将有助于揭示PCV的发病机制，并探索PCV与其他脉络膜疾病之间的内在联系。由于PCV患者眼球标本的稀缺，现有的病理学研究较少，且结果之间存在一定差异。文章通过介绍笔者最新的临床病理研究结果，并结合历年来国内外的研究，总结了关于PCV病灶所在的层次、起源及血管内皮生长因子(vascular endothelial growth factor, VEGF)表达水平的争议问题，阐明了PCV的临床病理研究现状。第一，PCV病灶的层次。

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电子全文

临床上，OCT成像显示PCV病灶位于视网膜色素上皮(retinal pigment epithelium, RPE)与Bruch膜的高反射线之间，属于I型脉络膜新生血管的特殊亚型。部分病理学研究认为PCV病灶位于Bruch膜内，但实际上PCV病灶更准确地位于RPE基底膜下。第二，异常分支血管网(branching vascular networks, BVN)的起源。尸体眼标本的病理分析表明，BVN起源于脉络膜动脉，且动脉穿过Bruch膜后，转变为薄壁毛细血管形成I型脉络膜新生血管。少数研究指出PCV可能由静脉扩张形成，并存在脉络膜静脉的淤滞。第三，VEGF在PCV病灶中的表达。VEGF是新生血管性AMD的关键致病因子，一些研究表明PCV病灶中VEGF表达升高，提示PCV可能与新生血管性AMD具有相似的发病机制，但也有研究发现PCV病灶中的VEGF表达为阴性，提示PCV的机制可能不完全依赖于VEGF。综上，PCV的病理特征具有复杂性，既有与新生血管性AMD相似的表现，也有肥厚脉络膜的特征。随着眼球捐献意识的提高，未来有望获得更多宝贵的眼球标本，为进一步探索PCV的发病机制提供支持,并为其临床诊断和治疗提供更有效的策略。

[关键词] 息肉状脉络膜血管病变；临床病理；玻璃膜疣；肥厚脉络膜

[中图分类号] R774.5 **[文献标志码]** A

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Research status of clinicopathologic studies of polypoidal choroidal vasculopathy

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Abstract Polypoidal choroidal vasculopathy (PCV) is the main subtype of neovascular age-related macular degeneration (AMD) in China. PCV differs from typical neovascular AMD in terms of epidemiology, clinical presentation, imaging features, and natural disease course. Recent studies suggest that, in addition to the traditional drusen-driven mechanism, PCV may also be associated with pachychoroid mechanism, which is particularly more common in Asian populations. In-depth pathological research will help uncover the pathogenesis of PCV and explore the intrinsic connections between PCV and other choroidal diseases. Due to the rarity of eye specimens from PCV patients, there is limited pathological research, and results can vary. Herein, this article summarize the controversial issues regarding the location level, origin, and the vascular endothelial growth factor (VEGF) expression of PCV lesions by introducing our latest clinicopathologic study on PCV and combining with previous studies in China and worldwide. First, the layer of PCV lesions. Clinically, OCT imaging shows that PCV lesions are located between the retinal pigment epithelium (RPE) and the hyperreflective line of Bruch membrane, making them a special subtype of type I choroidal neovascularization. Some pathological studies suggest that PCV lesions are located within Bruch membrane, but in fact, PCV lesions are more accurately located beneath the RPE basement membrane. Second, the origin of the branching vascular networks (BVN). Pathological analysis of postmortem eye specimens indicates that BVN originates from choroidal arteries, and after passing through Bruch membrane, they transform into thin-walled capillaries, forming type I choroidal neovascularization. A few studies suggest that PCV may result from dilation of choroidal vein, accompanied with vein stasis. Third, VEGF expression in PCV lesions. VEGF is a key pathogenic factor in neovascular AMD. Some studies show increased VEGF expression in PCV lesions, suggesting that PCV may share a similar pathogenic mechanism with neovascular AMD. However, other studies have found negative VEGF expression in PCV lesions, indicating that the mechanism of PCV may not be entirely dependent on VEGF. In conclusion, the pathological features of PCV are complex, showing both similarities to neovascular AMD and characteristics of pachychoroid. With the increasing awareness of eye donation, more valuable

eye specimens are expected to be obtained in the future, providing support for further exploration of the pathogenesis of PCV and offering more effective strategies for its clinical diagnosis and treatment.

Keywords polypoidal choroidal vasculopathy; clinicopathology; drusen; pachychoroid

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年龄相关性黄斑变性(age-related macular degeneration, AMD)是全球失明的主要原因，预计到 2040 年将影响 1.7 亿人，仅亚洲就有 1.1 亿人[1]。息肉状脉络膜血管病变 (polypoidal choroidal vasculopathy, PCV)被认为是新生血管性 AMD 的一个特殊亚型。PCV 和典型的新生血管性 AMD 在流行病学、临床和影像学特征以及自然病程方面存在一定差异。PCV在亚洲人群和男性中的患病率较高，且通常发生在比典型新生血管性AMD更年轻的群体中。PCV患者的眼睛更容易出现黄斑下出血，导致大范围的浆血性视网膜脱离，甚至在严重病例中出现玻璃体积血。在眼底检查中可能会发现视网膜下橙色结节。重要的是，玻璃膜疣(通常认为是AMD的标志)在PCV患者中不太常见，而部分PCV患者曾有中心性浆液性脉络膜视网膜病变的病史。近期研究指出，除了玻璃膜疣驱动的机制外，PCV也可能由肥厚脉络膜驱动的机制引起，后者在亚洲人群中更为常见。因此，PCV被认为属于肥厚脉络膜谱系。肥厚脉络膜是一个相对较新的概念，其实质是脉络膜毛细血管缺血，其影像学特点是Haller层静脉异常扩张、脉络膜高渗透性和通常脉络膜厚度增加[2]。随着对PCV的研究不断深入，病理学的进一步探索将有助于揭示其发病机制，并探索PCV与其他脉络膜疾病的内在联系。然而，由于PCV患眼眼球材料的稀缺，目前关于PCV的病理研究较少，并且研究之间缺乏一致性的结论。本文旨在根据笔者所在的Curcio研究团队近期发表的关于PCV临床病理研究的结果[3]，结合已有的文献，总结及厘清以下大家关注的PCV病理问题。

1 PCV病灶所在的层次：RPE下、Bruch膜内、还是脉络膜?

临床上，我们可以利用OCT观察到，PCV病灶位于RPE与Bruch膜的两条高反射线之间，是I型脉络膜新生血管的特殊亚型[2, 4]。然而，在一些病理学研究中PCV却被描述为Bruch膜内(intra-Bruch membrane)的病变[5-9]。究其原因，是各研究间采用的Bruch膜组织学分层定义不同。

Bruch膜在组织学上可分为3层或5层。在高分辨率病理学切片中，可观察到3层的Bruch膜(内胶原层、弹力纤维层、外胶原层)，并显示I型黄斑新生血管在视网膜色素上皮(retinal pigment epithelium, RPE)基底膜下空间生长。而低分辨率病理学切片，无法区分3层Bruch膜和两个基底膜(RPE和脉络膜毛细血管)，因此把RPE基底膜和脉络膜毛细血管基底膜归入Bruch膜的5层结构中，并推断PCV病灶位于Bruch膜内，而其实际为RPE基底膜下[10]。Gass(眼科专家)和Sarks(眼病理学家)等均认为，如果将Bruch膜定义为仅由3层结构组成，而不是5层，则可以最好地理解AMD的病理表现[11]。这3层可以被认为是血管性Bruch膜，与动脉内膜和血管壁的转运功能具有分子、结构和功能上的共性。此外，3层的Bruch膜有助于认识和理解位于RPE基底膜的基底膜沉积(basal laminar deposit, BLamD)的病理外观，BLamD可反映AMD的存在和严重程度(图1)[3, 12-15]。尽管薄而斑块状的 BLamD 在老年人眼中也很常见，但 AMD 中的 BLamD 是连续的、较厚的。因此，关于PCV病灶的层次，更准确的说是，位于病变的RPE基底膜，即BLamD下。

2 异常分支血管网的起源：动脉、还是静脉?

在BVN起源的研究上，完整的尸体眼较手术标本更可靠，因其判断动静脉是基于血管的连续性，而非血管的形态。Li等[3]及Rosa等[8]采用PCV患者的尸体眼进行病理切片，两项研究均指出，BVN起源于脉络膜动脉(含弹力纤维层，图2A、C)。并且，由于PCV病灶均位于视盘旁，根据脉络膜动脉所在的位置推断其为睫状后短动脉的分支，睫状后短动脉分支穿过Bruch膜缺口后，即转变为薄壁的毛细血管(即I型脉络膜新生血管)。此外，Li等[3]及Nakashizuka等[9, 16-18]均观察到脉络膜动脉的透明样变性(hyalinization)，见图2C。所谓透明样变性，是指平滑肌成分被性质不明的无定形假胶原组织广泛替代。透明样变性血管的特征是血浆蛋白外渗和基底膜样物质沉积。透明样变性是动脉硬化的变化之一，不仅在脉络膜中可见，而且在身体的其他部位也可见，例如大脑、肾脏和胰腺。

此外，另有两个切除黄斑区PCV病灶的手术标本

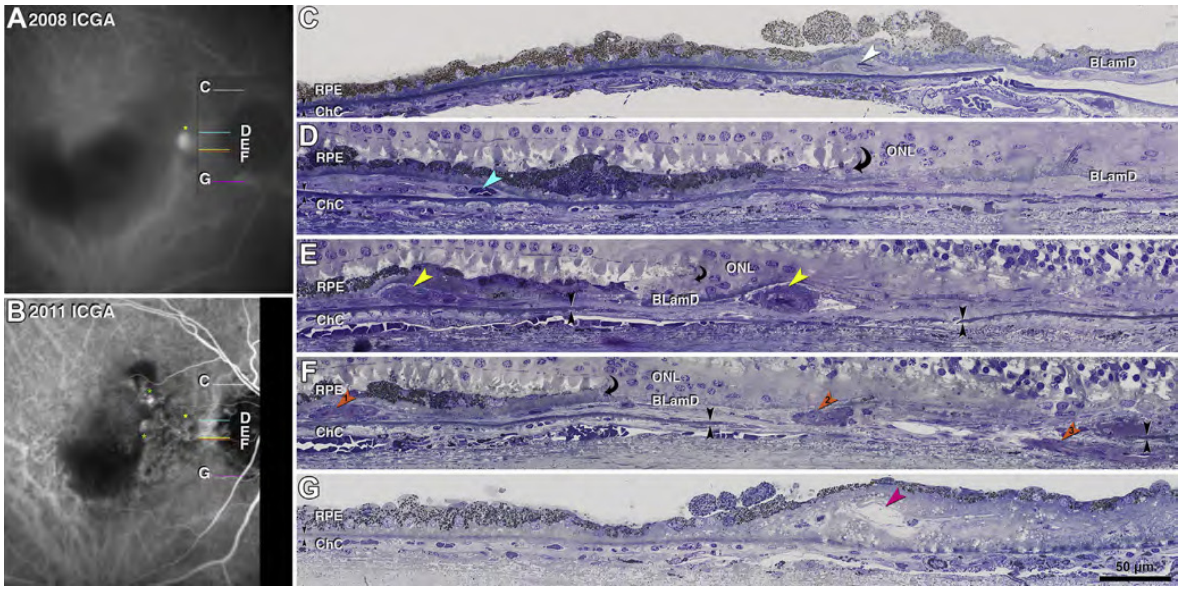


图 1　异常分支血管网和视盘周围脉络膜新生血管

Figure 1　Branching vascular network (BVN) and peripapillary choroidal neovascularization

Bruch 膜位于黑色箭头之间。在高分辨率视网膜病理切片中，可以清晰地区分RPE基底膜/BLamD(AMD)、Bruch膜和脉络膜毛细血管基底膜，Bruch膜由内胶原层、弹力纤维层、外胶原层3层结构组成。(A、B)吲哚菁绿血管造影 (ICGA)，分别于2008 年通过眼底照相机(A)和 2011 年通过扫描激光检眼镜拍摄(B)。黄色星号表示 2008 年出现的息肉状病灶，绿色星号表示2011 年出现的息肉状病灶。(C)视盘上方的薄壁脉络膜新生血管。(D、E、F)厚壁的异常分支血管网(BVN)。(D)中的蓝色箭头、(E)中的黄色箭头和(F)中的橙色箭头为可能伴有的平滑肌细胞。 Bruch 膜的断裂即(E) 和 (F) 中的黑色箭头间，较为明显。(G)视盘下方的薄壁脉络膜新生血管。黑色弯箭头表示外界膜下降。 BLamD 为基底膜沉积；ChC为脉络膜毛细血管；ONL为外核层 。引用自Li等文章[3]，图片已得到版权所有人授权。

Bruch membrane is located between the black arrows. In high-resolution retinal pathological sections, the RPE basement membrane/BLamD (AMD), Bruch membrane, and choriocapillaris basement membrane can be clearly distinguished. Bruch membrane consists of three layers: inner collagen layer, elastic fiber layer, and outer collagen layer. (A, B) Indocyanine green angiography (ICGA), taken by fundus camera in 2008 (A) and scanning laser ophthalmoscope in 2011 (B). Yellow asterisks indicate polypoidal lesions that appeared in 2008, and green asterisks indicate polypoidal lesions that appeared in 2011. (C) Thin-walled choroidal neovascularization above the optic nerve head (ONH). (D, E, F) Thick-walled BVN. Blue arrow in (D), yellow arrow in (E), and orange arrow in (F) may be the accompanied smooth muscle cells. The rupture of Bruch membrane is obvious between the black arrows in (E) and (F). (G) Thin-walled choroidal neovascularization inferior to the ONH. The black curved arrow indicates the descent of the external limiting membrane. BLamD, basal laminar deposit; ChC, choriocapillaris; ONL, outer nuclear layer. Cited from Li et al.[3]. The image has been authorized by the copyright owner.

病理研究指出[5, 18]，PCV病灶为囊样扩张的静脉，并推测与临床影像中的息肉状病灶相对应。其中一个研究还观察到脉络膜静脉的淤滞[5, 18]，而临床上部分PCV患者亦观察到可能由于涡静脉淤滞所致的肥厚脉络膜特征[19]。

3　血管内皮生长因子在PCV病灶中的表达水平：阳性，还是阴性?

血管内皮生长因子(vascular endothelial growth factor, VEGF)在PCV病灶中的血管内皮细胞的表达水平之所以重要，是因为VEGF是新生血管性AMD的重要致病因子，其升高与否反映了PCV是否具有与新生血管性AMD相同的发病机制。然而，各项研究的结论并不一致，观察到VEGF升高的研究认为[9]，PCV病灶实质为脉络膜新生血管，是新生血管性AMD的特殊亚型；此外，支持PCV为新生血管性AMD亚型的依据还包括，Li[3]及Okubo等[5, 7, 18]在PCV标本中观察到AMD的病理标志，即BLamD(图1)和基底膜线性沉积[20-28](basal linear deposit)。观察到VEGF表达为阴性的研究则认为，PCV与新生血管性AMD不同，VEGF可能不是导致其发病的主要因素[17,29]。

综上，鉴于临床上PCV既可表现为新生血管性AMD的特殊亚型，亦可呈现肥厚脉络膜的改变，PCV病理表现的多样性也就可以理解了。PCV是国人新生血管性AMD的主要亚型[30]，随着人们眼球捐献意识的提高，未来有望获得更多宝贵的眼球标本，为进一步探索PCV的发病机制提供支持,并为其临床诊断和治疗提供更有效的策略。有望为其病理研究提供得天独厚的条件，从而进一步加深对其发病机制的认识。

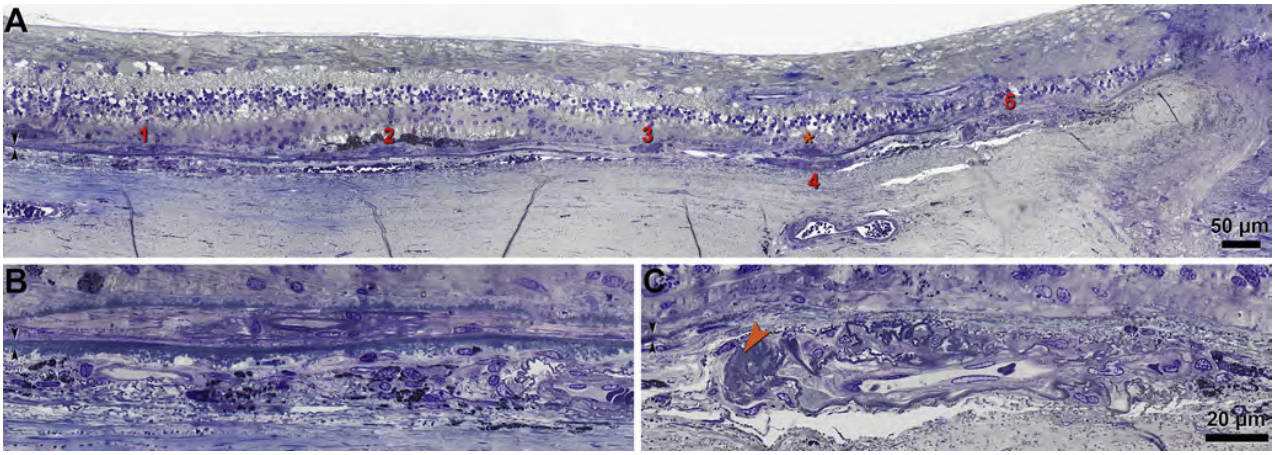


图 2 异常分支血管网及其起源的全景和细节图(来自图1患者)

Figure 2 Panoramic and detailed images of the branching vascular network (BVN) and its origin (from the same patient in Figure 1)

(A)显示了五个血管横截面(编号 1-5)，其中编号 2、3 和 4 如图1F 所示。视盘位于图的右侧缘，Bruch 膜(黑色箭头之间)的止端清晰可见。(B、C)分别放大了血管 1 和 5 及周围的区域。1 和 2为2 个毛细血管横断面，可见薄壁的由内皮细胞组成的非扩张管腔；3为RPE基底膜下的紫染血管，呈裂隙状开口，内皮细胞排列不清晰；4为脉络膜中类似的紫染血管横断面。星号表示类似的紫染物质。 5为脉络膜动脉，可通过皱褶的弹力纤维层识别。(B)毛细血管 1 的管壁很薄，仅由内皮细胞组成。(C)该脉络膜动脉可能与 4 和 3 处的血管相连。橙色箭头所指的紫染物质为动脉的透明样变性。引用自Li等[3]文章，图片已得到版权所有人授权。

(A) Five cross-sections of vessels (numbered 1-5) are shown, of which numbered 2, 3, and 4 are shown in Figure 1F. The optic disc is located at the right edge of the image, and the end of Bruch membrane (between the black arrows) is clearly visible. (B, C) Magnifications of vessels 1 and 5 and the surrounding area are shown, respectively. 1 and 2 are cross-sections of 2 capillaries, showing thin-walled non-dilated lumens composed of endothelial cells; 3 is a purple-stained vessel beneath the RPE basement membrane, with a slit-like opening and unclear endothelial cell arrangement; 4 is a similar cross-section of a purple-stained vessel in the choroid. The asterisk indicates similar purple-stained material. 5 is the choroidal artery, which can be identified by the wrinkled elastic fiber layer. (B) The wall of capillary 1 is very thin and composed only of endothelial cells. (C) This choroidal artery may be connected to the vessels at 4 and 3. The purple-stained material indicated by the orange arrow is hyaline degeneration of the artery. Cited from Li et al. [3]. The image has been authorized by the copyright owner.

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