

# 近视牵拉性黄斑病变发病机制及治疗研究进展

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## 摘要

病理性近视是世界范围内视力下降的主要原因, 在东亚和东南亚的发达国家最为普遍。病理性近视可引致多种并发症, 例如近视性黄斑病变、近视性脉络膜新生血管、视网膜脱落、白内障、开角型青光眼, 严重者可引致失明。病理性近视中视力丧失的主要与近视性黄斑病变有关。近年来, 越来越多的研究者开始关注近视牵拉性黄斑病变的发病机制。尽管病理性近视背后的发病机制尚未阐明, 但这一过程被认为是由两组不同的作用于视网膜的力引起的: 视网膜前和视网膜下因素, 从而导致视网膜上受到了切向牵引力和垂直牵引力。当视网膜前和视网膜下的作用力超过视网膜弹性时, 视网膜就会发生近视牵拉性黄斑病变的变化。目前, 还没有公认的关于近视牵拉性黄斑病变的治疗指南。本篇综述中, 我们的目的是回顾近视牵拉性黄斑病变的分期, 危险因素, 发病机制和治疗, 更好地了解近视牵拉性黄斑病变的发生发展过程, 有助于根据发病阶段制定治疗方案。

## 关键词

病理性近视, 近视牵引性黄斑病变, 玻璃体切除术, 黄斑扣带术, 内限膜剥离术

# Advances in the Pathophysiological Mechanism and Treatment of Myopic Traction Maculopathy

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## Abstract

Pathological myopia is the main cause of vision loss worldwide and is most common in developed countries in East and Southeast Asia. Pathological myopia can cause a variety of complications, such as myopic macular lesions, myopic choroid neovascularization, retinal detachment, cataract, open-angle glaucoma, and blindness in severe cases. Vision loss in pathological myopia is mainly related to myopic macular lesions. In recent years, more and more researchers have begun to pay attention to the pathophysiological mechanism of myopic traction macular lesions. Although the pathophysiological mechanism behind pathological myopia has not been clarified, this process is believed to be caused by two different groups of forces acting on the retina: pre-retinal and subretinal factors. Preretinal factors cause centrifugation and perpendicular traction on the retina, while subretinal factors cause perpendicular traction on the retina. When the force before and under the retina exceeds the elasticity of the retina, myopic traction macular changes will occur in the retina. At present, there are no recognized guidelines for the treatment of myopic traction macular lesions. In this review, our purpose is to review the staging, risk factors, pathophysiological mechanisms, and treatment of myopic traction maculopathy.

## Keywords

Pathological Myopia, Myopic Traction Maculopathy, Pars Plana Vitrectomy, Macular Buckle, Internal Limiting Membrane Peeling

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## 1. 概述

近视是一种全球性的公共卫生流行病，在亚洲国家最为明显。据估计，东亚和东南亚发达国家 80% 至 90% 的年轻人患有近视，随着时间的推移，这种患病率可能会增加，因为生活方式的改变促成了更多的近距离工作[1]。据预测，到 2050 年，世界上近一半的人群将患有近视，其中 10% 为高度近视[2]。国际近视研究院对病理性近视(Pathological Myopia, PM)的定义为与近视相关的眼轴过度伸长，可导致眼睛后段结构变化(包括后巩膜葡萄肿、近视性黄斑病变和高度近视相关的视神经病变)，及最佳矫正视力的丧失[3]。与单纯近视不同，PM 引起的视力损害通常是不可逆的，并且可能引起失明。病理性近视中视力丧失的主要原因与近视性黄斑病变(Myopic Maculopathy, MM)有关，包括脉络膜视网膜萎缩、近视性黄斑新生血管、与漆裂纹相关的炎性外层视网膜病变、近视性牵引性黄斑病和圆顶形黄斑[4]-[7]。

Ruia-Medrano 等人[8]提出 ATN 新分类与分级标准，将 MM 分为 3 大类：近视萎缩性黄斑病变(Myopic Atrophy Maculopathy, MAM)、近视牵引性黄斑病变(Myopic Traction Maculopathy, MTM)以及近视新生血管性黄斑病变(Myopic Neovascular Maculopathy, MNM)。其中，MTM 可分为 T0 到 T5 共 6 个等级：T0：无黄斑劈裂；T1：内层或外层中心凹劈裂；T2：内层和外层中心凹劈裂；T3：中心凹脱离；T4：黄斑裂孔；T5：黄斑裂孔性视网膜脱离。Parolini [9] 等人在 2021 年提出了一种新的以光学相干断层扫描技术(Optical Coherence Tomography, OCT)为基础的近视牵引性黄斑病变分类 MTM 分期系统(Myopic Traction Maculopathy Staging System, MSS)。MSS 把 MTM 描述为一种动态发展的疾病，将 MTM 分为 1~4 四个阶段，描述垂直于视网膜轴的视网膜变化，以及 a~c 阶段，描述与视网膜切向轴的中央凹变化。

- 1a 期: 存在内斑裂或内外斑裂, 中央凹轮廓正常;
- 1b 期: 存在内斑裂或内外斑裂, 中央凹存在内层黄斑裂孔(*Inner Lamellar Macular Hole*, I-LMH);
- 1c 期: 存在内斑裂或内外斑裂, 中央凹存在全层黄斑裂孔(*Full-Thickness Macular Hole*, FTMH);
- 2a 期: 以外侧黄斑裂孔为主, 中央凹轮廓正常;
- 2b 期: 以外侧黄斑裂孔为主, 在中央凹存在 I-LMH;
- 2c 期: 以外侧黄斑裂孔为主, 中央凹存在 FTMH;
- 3a 期: 黄斑分裂脱离, 中央凹轮廓正常;
- 3b 期: 黄斑分裂脱离, 中央凹存在 I-LMH;
- 3c 期: 黄斑分裂脱离, 中央凹存在 FTMH;
- 4a 期: 黄斑脱离, 中央凹轮廓正常;
- 4b 期: 黄斑脱离, 中央凹存在 I-LMH;
- 4c 期: 黄斑脱离, 中央凹存在 FTMH。

每个阶段都可能存在视网膜前异常, 可以添加一个“+”来表明其存在。这个分类方案使命名标准化, 让人们了解疾病的自然进展, 并帮助制定管理计划。

## 2. MTM 的危险因素及发病机制

### 2.1. 危险因素

MTM 的危险因素主要包括年龄、女性、较长的眼轴长度, 近视严重程度以及预先存在的后巩膜葡萄肿有关。年龄越大, 近视程度越重, 较长的眼轴长度是进展的独立危险因素[10]-[14]。

### 2.2. 病理发病机制

MTM 的发病机制很复杂。MTM 被认为是由两组不同的作用于视网膜的力引起的: 视网膜前和视网膜下因素[15] [16]。

#### 2.2.1. 视网膜前因素

视网膜前因素主要源于玻璃体 - 视网膜界面异常, 对黄斑区产生离心切向牵引力, 常见原因包括[17]:

(1) 黄斑前膜(ERM): 增生细胞膜紧密贴合于视网膜表面, 引起切向收缩, 导致中心凹变形及视网膜内层牵拉, 促进 MTM 早期(a-c 期)进展。

(2) 玻璃体黄斑牵拉综合征(VMT): 不完全玻璃体后脱离残留的玻璃体皮质附着于黄斑, 产生持续切向牵引。

(3) 玻璃体黄斑粘连: 局部粘连可加剧视网膜表面张力, 尤其在病理性近视眼中更为显著。

这些因素共同作用, 导致视网膜神经纤维层或内界膜水平的分裂, 形成 MTM 的初始结构改变。

#### 2.2.2. 视网膜下因素

视网膜下因素主要源于病理性近视相关的后极部结构性改变, 尤其是后巩膜葡萄肿(*Posterior Staphyloma*, PS) [18]。PS 导致巩膜、脉络膜和视网膜的不均匀拉伸, 形成垂直向牵引力, 是 MTM 中晚期(1~4 期)进展的核心机制。

(1) 后巩膜葡萄肿形态与 MTM 严重程度的相关性

PS 的形态特征与 MTM 的分期和预后密切相关。根据 Ohno-Matsui 分型, PS 可分为宽基底型(Type I)与局限陡峭型(Type II)等。研究表明, 局限陡峭型 PS (尤其是黄斑中心凹位于葡萄肿斜坡或底部时)产生的垂直牵引力更为集中, 更易导致视网膜层间广泛分离, 与更高阶的 MTM 分期(如 3~4 期)及更差的解剖

复位率相关[19]。OCT 测量显示, PS 深度(定义为巩膜穹窿高度)与视网膜劈裂范围呈正相关。深度 > 800  $\mu\text{m}$  的 PS 常伴随更广泛的视网膜层间分离、脉络膜显著变薄及中心凹脱离, 提示垂直牵引力在 MTM 进展中的关键作用[12] [20]。此外, PS 的范围(涉及后极部的面积)也与 MTM 的复杂程度相关, 广泛性 PS (累及黄斑及血管弓区域)患者更易发展为黄斑裂孔或黄斑裂孔性视网膜脱离[21]。

### (2) 巩膜胶原改变与遗传易感性

近年研究提示, MTM 的发生与巩膜细胞外基质重构及遗传背景密切相关[22] [23]。在病理性近视眼中, 巩膜胶原纤维(尤其是 I 型、III 型胶原)合成减少、降解增加, 胶原交联异常, 导致巩膜生物力学强度下降[24]-[26]。基质金属蛋白酶(MMPs, 如 MMP-2、MMP-9)表达上调与其组织抑制剂(TIMPs)失衡, 加速后极部巩膜重塑, 促进 PS 形成与发展[23] [27]。遗传学研究进一步揭示了 MTM 的易感基因[28]。全基因组关联研究(GWAS)发现, 多个近视相关基因座(如 ZC3H11B、BMP2)与 PS 形成风险相关[29], 尤其是胶原蛋白基因(如 COL1A1、COL5A1)的变异可能影响巩膜胶原的结构完整性, 增加 PM 及其并发症(包括 MTM)的易感性[30]。家族性 MTM 病例的报道也提示常染色体隐性遗传的可能性, 但具体致病基因仍需进一步验证[29]。

MTM 的最终发生是视网膜前与视网膜下因素协同作用的结果[16]。切向牵引力初始诱发视网膜内层分裂, 而垂直牵引力(尤其与 PS 相关)进一步加剧外层分离并扩展劈裂腔, 当合力超过视网膜层间粘附力(主要由 Müller 细胞及细胞外基质介导)的弹性阈值时, 即导致 MTM 的不可逆进展[31]-[33]。

## 3. MTM 的治疗

MTM 的治疗是有争议的, 在管理计划上没有达成共识, 应该根据具体情况量身定制。目前, 治疗 MTM 主要有两种手术方法, 内路手术和外路手术。

### 3.1. 内路手术治疗

内路手术包括平面部玻璃体切除术(Pars Plana Vitrectomy, PPV), 伴有或不伴有内限膜(Internal Limiting Membrane, ILM)剥离, 剥离内限膜又分为保留中央凹的内限膜剥离术(Fovea-Sparing Internal Limiting Membrane Peeling, FSIP)与完全内限膜剥离术(Complete Internal Limiting Membrane Peeling, ILMP)加气体填塞术。内路手术在解决视网膜上切向牵引力相关的病理方面更有效, 而因此, 对于有黄斑裂孔的眼睛, 由于内部限制膜施加切向牵引, 首选的手术是平坦玻璃体切除术和内部限制膜剥离术[34]。

### 3.2. 外路手术治疗

外路手术即黄斑扣带术(Macular Buckle, MB)。MB 的材料一直在演变, 近几年来一些新型材料被应用于 MB 中, 例如被称为 NPB 和 NPB 负载装置的新型黄斑扣带[35]、新型钛黄斑扣带[36] (Titanium Macular Buckle, TMB)等。MB 在缩短眼轴、显著改善中心凹及视网膜解剖结构有效果[37], 在解决垂直牵引力继发的病理方面更有效, 其他有分裂和脱离临床表现的眼睛应进行黄斑扣带以抵消垂直牵引力[38]-[40]。

### 3.3. 手术治疗方法的选择

在 MH 治疗领域, PPV 联合内界膜剥离技术可显著提升病理性黄斑区解剖结构的闭合效率。值得注意的是, 针对未合并黄斑裂孔的高度近视患者(国际分期 3a 或 4a 期), ILM 剥离操作可能增加医源性全层黄斑裂孔(Full-Thickness Macular Hole, FTMH)的发生概率[41]。最新研究数据显示, 相较于传统 ILM 剥离术式, FSIP 在降低术后 MH 发生率及优化最佳矫正视力(Best-Corrected Visual Acuity, BCVA)方面展现出更优的临床效果[42]-[44], 但是亦有临床观察表明两种术式在视力预后及并发症发生率方面无统计学差异。在病理性近视继发黄斑区病变的治疗策略选择上, MB 显示出独特优势。该术式不仅能有效改善患者

视功能,还能显著纠正近视性黄斑脱离导致的视物变形症状,提高患者视功能相关生存质量评分[45]。与PPV相比,MB通过机械性缩短眼轴长度,可针对性改善后巩膜葡萄肿等解剖异常,其术后BCVA提升幅度更为显著,且在复杂性黄斑裂孔及伴随黄斑脱离病例中显示出更高的解剖复位率。但需特别关注的是,MB术后可能出现手术源性内斜视、双眼复视等远期并发症,以及植入物暴露、玻璃体积血等围手术期风险[46]-[49]。在手术操作层面,黄斑扣带术存在一定技术难度:需精准分离眼外肌以获得充分术野,准确识别黄斑区巩膜投影位置,并确保硅胶扣带的正确空间定位。这些操作要点直接影响手术疗效及并发症防控,要求术者具备扎实的解剖知识和精细操作技能。

由于MTM的进程比较缓慢,无症状的近视黄斑裂孔可密切随诊观察,然而,临床医生应在监测期间保持警惕,因为中央凹裂可能发展为更严重的并发症,如黄斑裂孔性视网膜脱离。但在玻璃体明显牵拉引起视力下降时,需手术治疗[50]。因此,也有专家主张早期进行手术治疗。对于晚期MTM,建议手术治疗。

究竟应该选择内路手术还是外路手术,目前仍存在争议。Parolini等人建议手术治疗应根据疾病的MSS分期进行定制[51]。对于1a期和2a期,应每12至18个月进行一次监测,因为这些组的BCVA通常仍然良好,进展到更严重阶段的速度较慢。对于1b期和1c期,应考虑PPV,因为它的成功率更高。对于3a和4a阶段,采用MB可以解决主要垂直于黄斑的牵引力。对于2b、3b和4b阶段,应考虑进行黄斑屈曲以解决裂孔或脱离,必要时进行PPV以恢复中央凹轮廓。Ripa M等人证明当MB作为单一手术应用于MTM的3a、3b、4a和4b阶段时,可显著改善中央凹和视网膜解剖学和功能[37]。对于2c、3c和4c期,可以同时对视网膜和黄斑孔进行MB和PPV治疗[51]。例如在黄斑脱离伴黄斑裂孔这种情况下,首选的手术是PPV伴ILM剥离联合MB[49][52]。

### 3.4. 内路手术与外路手术的对比

为更直观地比较两种主流手术方式的差异,从多个维度总结了PPV与MB的特点,见表1:

**Table 1.** Differences between internal approach and external approach surgery

**表 1.** 内路手术与外路手术的区别

对比维度	内路手术(PPV ± ILM 剥离)	外路手术(黄斑扣带术, MB)
主要原理	移除玻璃体及视网膜前膜,解除切向牵引	机械性顶压后极部,缩短眼轴,缓解垂直牵引
核心优势	直接解除玻璃体视网膜界面牵引;技术相对成熟普及	针对性改善后巩膜葡萄肿解剖结构;对眼内扰动小
主要缺点	可能增加医源性裂孔风险(尤其广泛ILM剥离);对后巩膜葡萄肿形态无直接改善	操作难度高,学习曲线陡峭;存在植入物相关并发症风险
解剖成功率范围	黄斑裂孔闭合率: 70%~92%	视网膜复位率: 80%~95%
功能成功率(BCVA 提升)	术后视力稳定或改善比例: 65%~85%	术后视力稳定或改善比例: 75%~90%
主要并发症	医源性裂孔(5%~15%);白内障进展/发生(≈100%未联合晶切);眼压升高	复视/斜视(5%~20%);植入物暴露/感染(1%~5%);屈光改变
典型适应症(参考MSS分期)	1b, 1c期(内层裂孔为主);合并明确玻璃体黄斑牵引	3a, 4a期(广泛劈裂/脱离,无全层裂孔);陡峭型后巩膜葡萄肿
联合手术	常联合ILM剥离、气体/硅油填充	可联合PPV治疗合并的全层黄斑裂孔(如4c期)

## 4. 讨论

近年来,病理性近视导致的眼睛后段结构变化引起了广泛关注。其中 MTM 是致盲的主要原因。OCT 的发展可以更好地可视化 MTM 的详细改变和早期洞察,这不仅有助于疾病的分类和定义,而且有助于评估自然病程和治疗。目前的研究有很多局限性,大部分研究都只有少数病例,平均随访时间不够长。治疗 MTM 的方法主要有玻璃体切除术伴保留中央凹的内限膜剥离术或完全内限膜剥离术和黄斑扣带术,具体治疗方案应该可以参考 MSS 分期,根据实际情况而定。

未来研究方向应聚焦于:① 基于 OCT 生物力学模型量化视网膜前或视网膜下牵引力,实现 MTM 进展风险预测;② 开展多中心随机对照试验,比较 PPV 与 MB 在特定分期中的长期疗效与安全性;③ 从基因-胶原代谢层面探索 PM 及 MTM 的早期干预靶点。

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