

非感染性肉芽肿性全葡萄膜炎1例

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摘要

目的: 本文旨在分析非感染性肉芽肿性全葡萄膜炎的临床表现、诊断思路及治疗效果。方法: 收治我院1例双眼非感染性肉芽肿性全葡萄膜炎患者的临床资料, 包括眼部表现、辅助检查、病原学筛查、治疗方案及随访结果, 并结合相关文献进行讨论。患者经静脉激素冲击治疗后葡萄膜炎症状缓解, 病情好转后出院。随访3个月, 患者病情稳定, 视力恢复良好。结论: 非感染性肉芽肿性全葡萄膜炎可表现为多种眼部炎症体征, 诊断需在充分排除感染因素基础上结合典型眼部体征和辅助检查综合判断。对炎症活动明显者, 早期、足量糖皮质激素治疗有助于迅速控制炎症、改善预后; UBM在识别炎症相关瞳孔阻滞及动态评估前节结构变化方面具有重要参考价值。

关键词

非感染性葡萄膜炎, 肉芽肿性葡萄膜炎, 宏基因组检测, 超声生物显微镜, 病例报告

A Case of Non-Infectious Granulomatous Panuveitis

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Abstract

Objective: To analyze the clinical manifestations, diagnostic approach, and therapeutic outcomes of non-infectious granulomatous panuveitis. **Methods:** The clinical data of one patient with bilateral non-infectious granulomatous panuveitis admitted to our hospital were collected, including ocular manifestations, auxiliary examinations, pathogen screening, treatment regimen, and follow-up outcomes, and were discussed in conjunction with the relevant literature. After intravenous corticosteroid

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pulse therapy, the uveitic manifestations and related symptoms were rapidly relieved, and the patient was discharged after clinical improvement. During the 3-month follow-up, the condition remained stable and visual acuity recovered well. Conclusion: Non-infectious granulomatous panuveitis may present with various ocular inflammatory signs. Its diagnosis requires a comprehensive assessment based on typical ocular signs and auxiliary examinations after infectious causes have been fully excluded. In patients with marked inflammatory activity, early and adequate corticosteroid therapy can help rapidly control inflammation and improve prognosis. Ultrasound biomicroscopy is of important value in identifying inflammation-related pupillary block and dynamically evaluating anterior segment structural changes.

Keywords

Non-Infectious Uveitis, Granulomatous Panuveitis, Metagenomic Testing, Ultrasound Biomicroscopy, Case Report

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1. 研究背景

葡萄膜炎是一类发病机制复杂且严重威胁视力的眼内炎症性疾病，在青壮年人群中尤为高发，发病率呈逐年上升趋势[1]-[3]。其发病机制复杂，涉及感染、免疫失衡及遗传易感性等多因素交互作用[4]。根据病因，葡萄膜炎可分为非感染性葡萄膜炎和感染性葡萄膜炎，而根据临床病理特征，则可分为肉芽肿性和非肉芽肿性炎症[5][6]。非感染性肉芽肿性葡萄膜炎是一组无明确病原体感染证据，其核心病理特征为葡萄膜组织内出现由单核吞噬细胞、上皮样细胞及多核巨细胞聚集形成的结节样浸润(即肉芽肿性炎症)[7][8]。非感染性病因可根据主要炎症来源的解剖位置进一步细分为前部、中部、后部和全葡萄膜炎[5]。本院收治 1 例 25 岁青年男性双眼非感染性肉芽肿性全葡萄膜炎患者，经规范化个性化治疗后痊愈出院，现报道如下。

2. 临床资料

患者，男，25 岁，因“双眼眼红伴视力下降半月余”入院。患者于入院前半月余无明显诱因出现双眼眼红，随后逐渐出现视力下降，无明显眼痛、畏光、流泪，无复视、虹视及闪光感。发病前 1 周有上呼吸道感染病史，未予特殊处理。既往无眼外伤史、眼内手术史及其他特殊病史。1) 眼科专科检查：右眼裸眼视力 0.2，非接触性眼压 6.4 mmHg；左眼裸眼视力 0.2，非接触性眼压 5.9 mmHg。双眼结膜充血(+)，角膜后均可见大量羊脂状 KP，前房闪辉(++)，均存在虹膜后粘连。双眼虹膜纹理尚清，瞳孔缘均可见散在灰白色半透明结节；右眼瞳孔欠圆，直径约 4.0 mm，左眼瞳孔欠圆，直径约 4.5 mm，对光反射均消失。双眼晶状体表面可见色素粘连，晶状体尚透明。玻璃体混浊均较明显(++++)，眼底窥视不清(图 1)。2) 辅助检查：眼部 B 超提示双眼玻璃体明显浑浊(图 2)。眼底荧光素血管造影(FFA)示左眼视网膜成像尚清，随造影时间延长可见广泛散在多灶性点状高荧光灶，以颞侧、下方及鼻侧象限较为明显，晚期呈弥散性强荧光，视盘于中晚期出现弥漫性荧光增强，边界欠清；右眼亦可见广泛散在多灶性点状高荧光灶，以后极部、颞侧及鼻侧象限较明显，视盘于中晚期出现显著弥漫性荧光增强，边界欠清(图 3)。UBM 示双眼多个象限虹膜明显前膨隆，前房变浅，房角狭窄，并存在瞳孔阻滞表现，前房内可见散在点状高回声(图 4)。3) 入院诊断：双眼肉芽肿性全葡萄膜炎。4) 为进一步明确病因并鉴别感染性与非感染性葡萄膜炎，

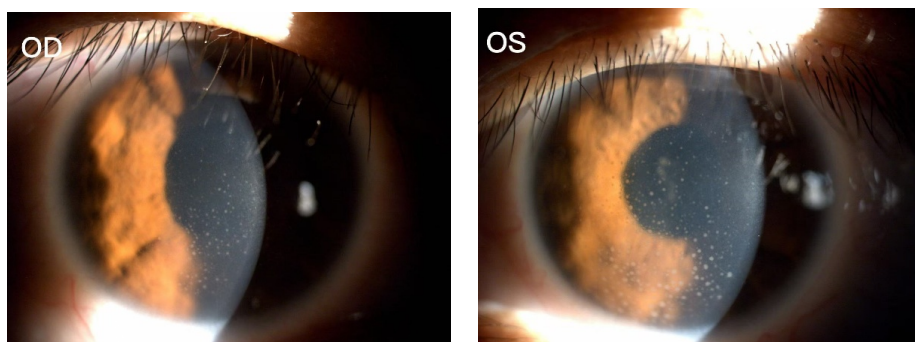


Figure 1. Photograph of the anterior segment of both eyes (Before treatment)

图 1. 双眼前节照片(治疗前)

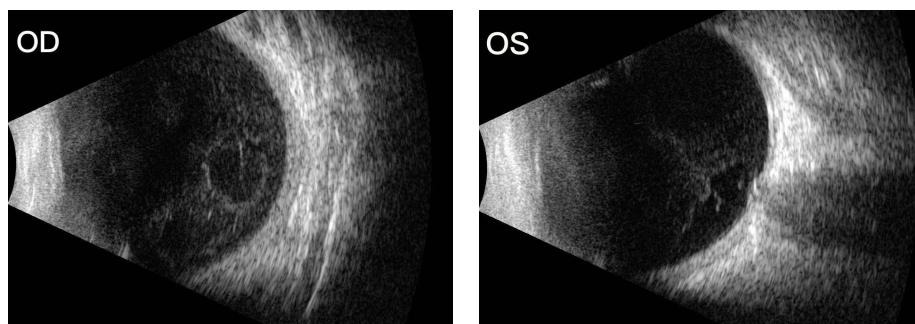


Figure 2. B-scan image of both eyes (Before treatment)

图 2. 双眼 B 超(治疗前)

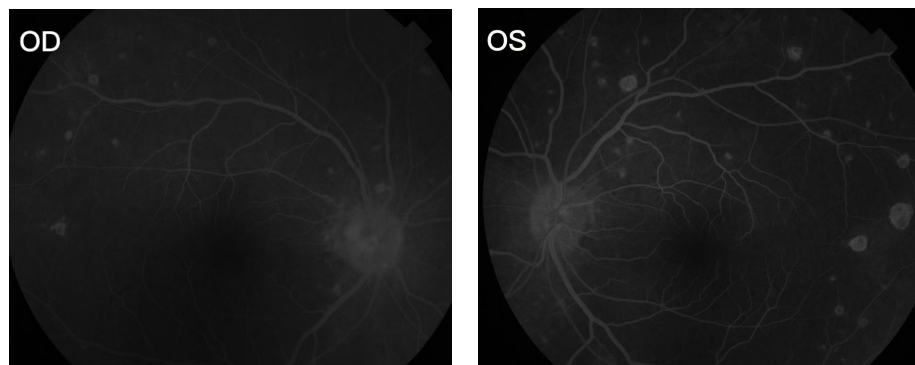


Figure 3. Fundus fluorescein angiography in both eyes (Before treatment)

图 3. 双眼荧光素眼底血管造影(治疗前)

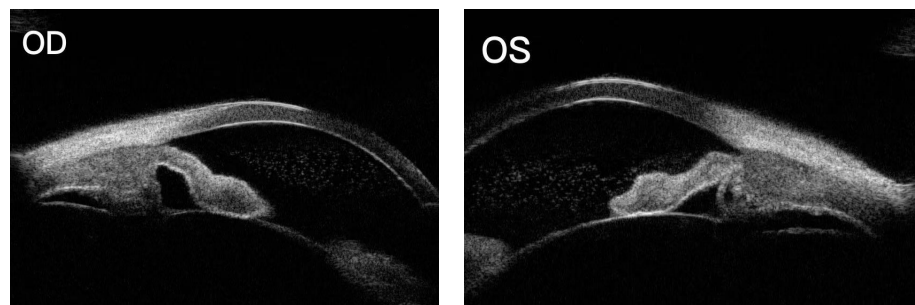


Figure 4. Ultrasound biomicroscopy in both eyes (Before treatment)

图 4. 双眼超声生物显微镜(治疗前)

入院后完善相关检查：实验室检查示：巨细胞病毒 IgG 抗体 86.2 IU/ml，单纯疱疹病毒(I+II)型 IgG 抗体 > 30.0 Index，风疹病毒 IgG 抗体 49.4 IU/ml，补体 C4 为 408.81 mg/L，HLA-B27 阴性，其余检查未见明显异常。进一步行房水病原微生物宏基因组检测(mNGS)：采集房水样本送检，经核酸提取、文库构建及高通量测序后，先去除人源序列，再与涵盖细菌、真菌、病毒及寄生虫等病原体的微生物参考数据库进行比对分析，以完成广谱病原学筛查。结果未检出明确病原微生物。结合临床表现、辅助检查及病原学筛查结果，最终诊断为双眼非感染性肉芽肿性全葡萄膜炎。在排除激素使用相关禁忌症后，给予甲泼尼龙静脉冲击治疗，联合局部散瞳、抗炎及对症支持治疗，并予补钾、补钙及护胃等支持治疗，治疗后患者眼部症状逐渐改善，瞳孔散大，双眼玻璃体混浊减轻，视力提高至 0.5。随后改为口服甲泼尼龙维持治疗后出院。出院后 1 周门诊复查：右眼裸眼视力 0.4，左眼裸眼视力 0.6-；右眼眼压 14.7 mmHg，左眼眼压 12.4 mmHg。双眼结膜充血(+)，角膜后仅见少量陈旧性 KP，前房中深，前房闪辉(++)，瞳孔呈药物性散大，形态欠圆，仍可见陈旧性瞳孔粘连，对光反射迟钝，晶状体透明(图 5)。B 超示双眼玻璃体轻度浑浊(图 6)；UBM 示部分象限仍可见虹膜轻度前膨隆及房角狭窄，但前房内点状高回声几乎消失，较前明显改善(图 7)。

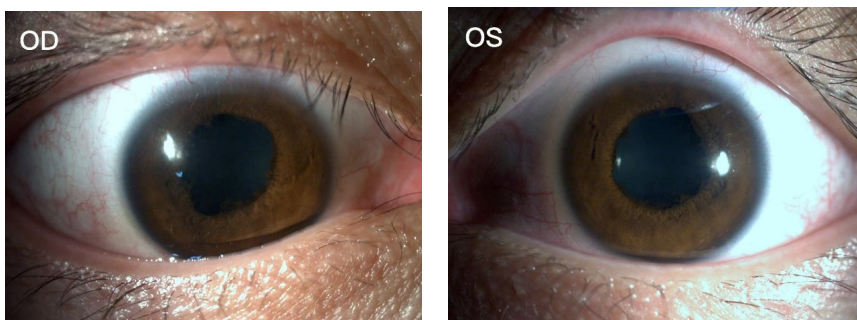


Figure 5. Photograph of the anterior segment of both eyes (After treatment)

图 5. 双眼前节照片(治疗后)

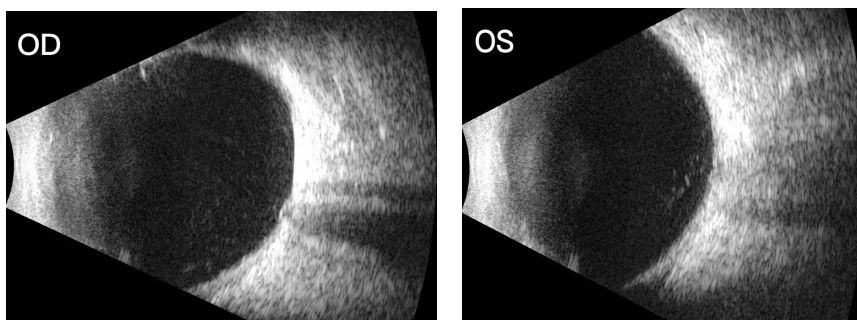


Figure 6. B-scan image of both eyes (After treatment)

图 6. 双眼 B 超(治疗后)

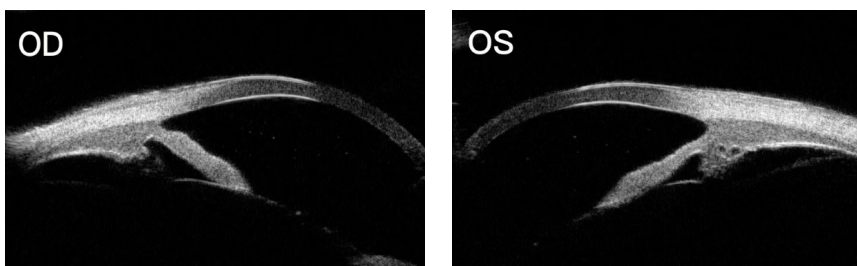


Figure 7. Ultrasound biomicroscopy in both eyes (After treatment)

图 7. 双眼超声生物显微镜(治疗后)

3. 讨论

本例患者为青年男性，双眼急性起病，入院时主要表现为大量羊脂状角膜后沉着物、瞳孔缘灰白色半透明结节、明显玻璃体混浊以及眼底荧光血管造影所示广泛视网膜血管渗漏和视盘荧光增强，符合双眼肉芽肿性全葡萄膜炎的临床特征[8][9]。肉芽肿性葡萄膜炎并非某一单独疾病，而是一组具有相似炎症表型的综合征，其背后可对应感染性与非感染性多种病因，因此讨论的重点应是追问其潜在病因及全身关联疾病[9]。本例无眼外伤及眼内手术史，不支持交感性眼炎[10]；HLA-B27 阴性且临床表现不符合 HLA-B27 相关急性前葡萄膜炎[11]，结合双眼同时受累、前后节均有明显炎症活动及对糖皮质激素冲击治疗反应迅速等特点，最终更倾向于非感染性肉芽肿性全葡萄膜炎。

非感染性肉芽肿性葡萄膜炎的诊断本质上仍属于排除性诊断。对于具有羊脂状 KP、虹膜结节、玻璃体炎及血管炎表现的患者，感染性病因必须首先被充分排除，尤其是结核、梅毒、疱疹病毒家族感染及弓形虫感染等[8][12]。房水或玻璃体液 mNGS 为感染性葡萄膜炎的病原学筛查提供了更广谱的技术手段，对有限样本条件下的病原识别具有明显优势；其诊断效能受样本类型、病原负荷、污染背景及判读阈值影响，因而阴性结果不能脱离临床背景孤立解读[13]。因此，本例结合病史、眼部表现、常规实验室检查、房水 mNGS 结果以及激素治疗反应，综合判断为非感染性肉芽肿性葡萄膜炎。

本例另一值得关注的特点是患者入院时双眼眼压偏低，但 UBM 提示多个象限虹膜前膨隆、前房变浅、房角狭窄并存在瞳孔阻滞。这一现象提示活动性葡萄膜炎中的“低眼压”与“房角狭窄”并不矛盾[14]。一方面，急性炎症期睫状体受累可导致房水分泌减少，同时炎症介导的前列腺素增多又可增加葡萄膜巩膜外流，从而出现低眼压[14][15]；另一方面，广泛虹膜后粘连可造成瞳孔阻滞，引起虹膜膨隆和继发性房角关闭[16]。因此，低眼压并不能排除机械性房角关闭，反而提示应结合 UBM 动态评估前节结构改变[17]。本例治疗后 UBM 显示虹膜膨隆减轻、前房内高回声减少，与临床症状和 B 超结果同步改善，说明 UBM 在识别炎症相关瞳孔阻滞、判断病理机制及监测治疗反应方面具有较高价值[17]。

在治疗方面，对于双眼受累、炎症活动重、玻璃体混浊明显并已影响视功能的非感染性葡萄膜炎，系统性糖皮质激素仍是快速控制炎症的一线方案[5]。本例在排除激素禁忌证及感染因素后予以甲泼尼龙静脉冲击联合局部散瞳、抗炎及降眼压治疗，数日内视力、前节炎症体征、B 超及 UBM 表现均明显改善，提示早期足量激素治疗对阻断炎症瀑布反应、恢复眼内屏障稳定性具有重要意义。但糖皮质激素并不适合长期单药维持，对于复发性、慢性或减量后反跳者，也应尽早考虑甲氨蝶呤、吗替麦考酚酯、硫唑嘌呤等免疫调节药物，必要时可升级至阿达木单抗等生物制剂，以实现激素减量和长期炎症控制[18][19]。

此外，本例短期疗效良好，但肉芽肿性葡萄膜炎的长期监测同样关键[5][18]。持续或反复炎症以及糖皮质激素暴露均可增加并发性白内障、继发性青光眼、持续性低眼压、黄斑水肿、角膜内皮损伤和永久性瞳孔粘连等风险[11][20]。尤其本例已有明显虹膜后粘连、瞳孔变形及前节解剖结构异常，提示即使急性期控制良好，后续仍需要在规律随访中持续监测视力、眼压、前房炎症情况、黄斑 OCT 及眼底血管炎等活动情况。

4. 结论

本病例提示，对青壮年双眼急性肉芽肿性全葡萄膜炎患者，应重视感染性病因排查。在炎症活动期，即使眼压偏低，也不能忽视瞳孔阻滞及继发性房角关闭的可能，必要时应结合 UBM 评估前节结构变化。对感染因素基本排除且炎症较重者，及早应用足量糖皮质激素有助于尽快控制炎症并改善视功能，后续仍需长期随访以监测复发及并发症。

声 明

该病例报道已获得病人的知情同意。

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