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肠系膜脂膜炎合并卡氏肺孢子菌肺炎1例报告

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摘 要

目的：探讨肠系膜脂膜炎合并卡氏肺孢子菌肺炎的临床表现、诊断方法及治疗策略，加强对肠系膜脂膜炎合并卡氏肺孢子菌肺炎的理解。方法：介绍1例肠系膜脂膜炎合并卡氏肺孢子菌肺炎的临床资料，并进行相关文献复习。结果：该患者在服用激素治疗肠系膜脂膜炎减量过程中出现胸闷、憋气、发热，胸部CT出现双肺多发磨玻璃影，确诊肠系膜脂膜炎合并卡氏肺孢子菌肺炎，应用复方新诺明及卡波芬净及时治疗后好转出院。结论：肠系膜脂膜炎合并卡氏肺孢子菌肺炎如能早期确诊，治疗效果较好。

关键词

卡氏肺孢子菌肺炎，肠系膜脂膜炎，诊治

Reports of a Case of Mesenteric Panniculitis Complicated with Pneumocystis Carinii Pneumonia

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Abstract

Objective: To investigate the clinical features, diagnosis and treatment strategies of patients with mesenteric panniculitis (MP) complicated with pneumocystis carinii pneumonia (PCP), and to

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strengthen the understanding of mesenteric panniculitis complicated with Pneumocystis carinii pneumonia. Methods: Introduce the clinical data of 1 case of mesenteric panniculitis complicated with pneumocystis carinii pneumonia, and review the relevant literature. Results: The patient diagnosed with mesenteric panniculitis combined with Pneumocystis carinii pneumonia is treated promptly, and is discharged from the hospital. The outpatient return visit did not have obvious respiratory symptoms. Conclusion: If patients with mesenteric panniculitis complicated with Pneumocystis carinii pneumonia can be diagnosed early, the treatment effect is better.

Keywords

Mesenteric Panniculitis, Pneumocystis Carinii Pneumonia (PCP), Diagnosis and Treatment

1. 引言

卡氏肺孢子菌肺炎是一种严重的呼吸系统机会性感染，误诊率及病死率均较高，临床更多见于人类免疫缺陷病毒(Human immunodeficiency virus, HIV)感染患者，但非 HIV 感染合并卡氏肺孢子菌肺炎发病率逐年增加。目前仍未有肠系膜脂膜炎合并卡氏肺孢子菌肺炎的病例报告。为提高临床认识，实现早期诊断，减少误诊、漏诊，经患者本人知情同意，现将我院收治的 1 例肠系膜脂膜炎合并卡氏肺孢子菌肺炎病例介绍如下。

2. 病例资料

患者青年女性，主因“胸闷、憋气 3 天，发热 1 天”于 2020 年 9 月 12 日入院。患者 3 天前着凉后出现胸闷、憋气，多于侧卧位时明显，未予重视。1 天前患者出现发热，体温最高达 39.6℃，胸闷、憋气较前加重，无咳嗽、咳痰，自服“对乙酰氨基酚”后体温降低，数小时后体温再次上升至 38℃以上来诊。给予患者左氧氟沙星 0.5 g 静滴后体温逐渐降至正常，胸闷、憋气等不适较前减轻，以“社区获得性肺炎”收入我院急诊内科。既往：外院确诊“肠系膜脂膜炎”4 月，予甲泼尼龙片 24 mg bid、环磷酰胺片 0.1 g qod，后因胃、后背及脊柱疼痛，停用环磷酰胺，换用吗替麦考酚酯。40 余天前我院风湿免疫科住院期间更换治疗方案为甲泼尼龙片 40 mg qd，吗替麦考酚酯分散片 0.75 g bid。其后规律门诊复诊激素减量，1 月前激素减量至 36 mg qd，环磷酰胺 0.1g qd 隔周服用。体检：T：36.3℃ P：87 次/分 R：20 次/分 BP：152/73 mmHg。双肺呼吸音清，未闻及干湿性啰音，右下腹压痛，双下肢指凹性水肿。余查体阴性。入院前查血常规：白细胞计数 12.21 * 10^9/L；中性粒细胞计数 10.15 * 10^9/L；胸部 CT 提示：双肺炎症可能性大。入院诊断：社区获得性肺炎、肠系膜脂膜炎。

入院后辅助检查：血常规 + CRP：白细胞计数 7.81 * 10^9/L (4.0~10.0 * 10^9/L)，中性粒细胞计数 6.69 * 10^9/L (2.0~7.0 * 10^9/L)，血红蛋白 108 g/L (110~150 g/L)，中性粒细胞百分率 85.60% (50%~75%)，全血 C 反应蛋白 16.43 mg/L (<5 mg/L)；肝功：乳酸脱氢酶 288.00 U/L (135~215 U/L)；血糖分析：葡萄糖 7.05 mmol/L (3.9~7.0 mmol/L)；血脂分析：总胆固醇 6.09 mmol/L (3~5.2 mmol/L)，低密度脂蛋白 3.67 mmol/L (<3.12 mmol/L)；电解质、肾功、血磷、血钙、血镁、CK + CKMB、降钙素原、传染性标志物 4 项均未见明显异常。入院后经验性给予左氧氟沙星抗感染，甲泼尼龙片抗炎等治疗，胸闷、憋气无明显缓解，抗生素升级为莫西沙星后因恶心、头晕等不良反应，换用哌拉西林纳他唑巴坦纳继续抗感染治疗。期间

患者仍有反复高热，体温最高达 39.3℃，更换口服甲泼尼龙片为甲强龙 20 mg qd 静滴，并予对乙酰氨基酚对症降温，发热无明显好转。完善 CD4 绝对计数 435 cells/UL，EB 病毒衣壳抗原 IgG、核抗原 IgG 阳性，巨细胞病毒抗体及 DNA，血培养、痰培养、抗酸菌检测、真菌 G 试验、曲霉菌 GM 试验等均为阴性。复查胸部 CT：双肺多发磨玻璃样影(如图 1)。呼吸科会诊，考虑不除外特殊病原体感染。于 09 月 22 日完善气管镜检查，病理回示：(双肺多部位盲检)少许肺及呼吸性粘膜组织呈慢性炎，肺泡上皮较增生，肺泡腔内见灶性泡沫细胞聚集及渗出物机化反应，肺泡间隔增宽伴间质纤维组织增生；肺泡灌洗液病原学检测阴性。NGS 检查结果回示考虑为卡氏肺孢子菌肺炎。转入我院呼吸内科，给予卡泊芬净及复方新诺明抗感染治疗。患者胸闷、憋气及胸痛缓解，复查 CT 示肺内病变较前明显好转(如图 2)，于 10 月 1 日出院。出院后患者门诊复诊，截至目前为止未再出现明显胸闷、胸痛、发热等症状。

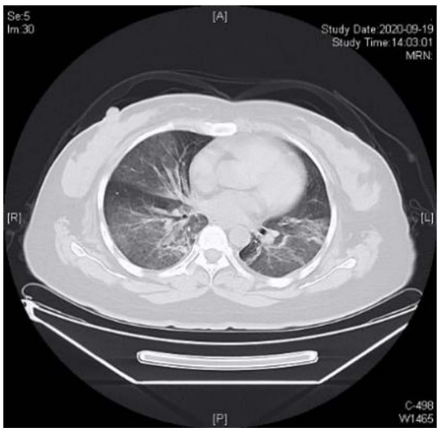

Figure 1. On September 19, chest CT showed extensive ground glass opacity in both lungs
图 1. 9 月 19 日胸部 CT 示双肺广泛磨玻璃影

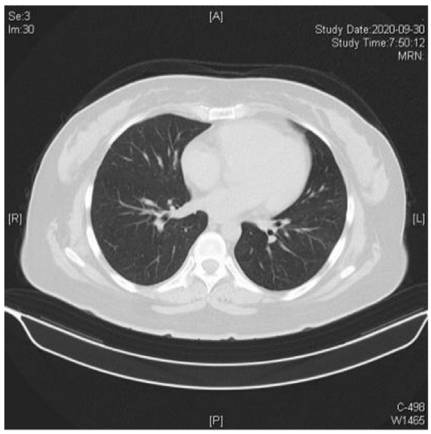

Figure 2. On September 30, chest CT showed that bilateral lung lesions were significantly improved
图 2. 9 月 30 日胸部 CT 示双肺病变较前明显好转

3. 讨论

卡氏肺孢子菌肺炎(Pneumocystis carinii pneumonia, PCP)，是由寄生于人体肺泡上皮细胞表面的卡氏肺囊虫大量繁殖造成上皮细胞损害，阻碍气体交换的一种机会性感染[1]。该病是由 T 细胞和体液免疫反应(在较小程度上)介导的。在免疫抑制的患者中，感染会导致由单核细胞、CD8 淋巴细胞和激活的巨噬细胞介导的免疫反应功能失调，从而导致弥漫性肺损伤[2]。此外，吸入的肺孢子虫滋养体抑制肺泡内的

上皮修复过程，导致严重的肺损伤[3]。PCP 感染后可以表现为轻微的炎症至重症肺炎，严重者导致呼吸衰竭甚至死亡[4]，临床特征是干咳、发热和呼吸困难。胸部 X 线片上，最常见弥漫性双肺间质浸润，高分辨率计算机断层扫描可能显示出广泛的磨玻璃影及囊性病变[5] [6]，是一种临床病死率极高的疾病。

本病多发生在免疫功能缺陷及长期应用免疫抑制剂的患者中。HIV 阴性的患者 PCP 发病率较低，约为 0.01%~0.10% [1]。但值得注意的是，随着免疫抑制剂的逐步使用，非 HIV 感染的免疫抑制人群的发病率也随之增加[5] [7] [8]。在 HIV 阴性患者中，炎症性疾病占 PCP 的 20% [9]。对于接受免疫抑制药物治疗的自身免疫性疾病患者，合并 PCP 目前还没有具体的共识指南[10] [11] [12]。已经明确的是，炎症性疾病及其并发症和免疫抑制治疗方案增加了这类患者患 PCP 的风险[13]。

肠系膜脂膜炎(Mesenteric panniculitis，MP)是一种临床上十分罕见的慢性肠系膜炎症性疾病。MP 的病因可能是腹部手术、外伤、缺血、药物、过敏或自身免疫性疾病[14] [15] [16] [17]。临床上根据 MP 患者的具体情况，通常选择保守治疗，如抗感染和/或免疫抑制治疗、糖皮质激素、非甾体类抗炎药物、秋水仙碱、黄体酮、环磷酰胺药物治疗等，但疗效报道不一致[15] [16] [17] [18]。该患者炎症性疾病本身及糖皮质激素、环磷酰胺、吗替麦考酚酯等免疫抑制药物的使用，可能都是促进 PCP 的因素。

非 HIV 感染患者合并 PCP 的死亡率(27%~62%)高于 HIV 感染者(4%~15%) [6] [10] [19]-[24]。成人数据显示，与 HIV 感染者可能缓慢且隐匿的表现不同，非 HIV 感染的免疫功能受损者可能会出现快速进展性的病程，通常在几天内出现呼吸衰竭[1] [5] [21] [25]。此外，非 HIV 感染患者合并 PCP 的临床表现也取决于所使用的免疫抑制剂，其中最常提到的有：长时间应用中到高剂量糖皮质激素、环磷酰胺、利妥昔单抗、阿林单抗和肿瘤坏死因子拮抗剂[26] [27]。糖皮质激素可以减少 CD4 淋巴细胞，进而可能导致 PCP 更易发生。在非 HIV 感染免疫抑制人群中，使用糖皮质激素已成为 PCP 的主要致病因素，当强的松龙达到或超过 16 mg 时，风险尤其增加[28]。有趣的是，在一些研究中，相当比例的 PCP 只在皮质类固醇逐渐减量过程中出现[29]。本例病人在肠系膜脂膜炎治疗过程中先后应用环磷酰胺、吗替麦考酚酯等免疫抑制剂及糖皮质激素，且正是在糖皮质激素减量过程中出现 PCP 感染。值得注意的是，并非所有免疫抑制患者都会出现淋巴细胞减少症[29]。与 HIV 感染者一样，监测 CD4+ T 淋巴细胞似乎不足以预测 PCP 发生风险[30]。支气管镜、支气管灌洗或经支气管活检诊断率高于痰液诊断的诊断率，但不易获得[31]。非 HIV 感染的 PCP 患者少见，缺乏特异有效且易行的诊断手段，是导致该病诊断困难乃至误诊、漏诊的重要原因。该病例 CD4 + T 淋巴细胞未见明显下降，支气管镜检测及肺泡灌洗液均未检见卡氏肺孢子球菌，但不能据此排除 PCP 感染。PCP 患者临床病程凶险，病死率极高，尤其非 HIV 感染的 PCP 患者病情进展迅速，提示我们临床诊治过程中，辅助检查不十分支持的情况下仍不应忽视该病可能。另外，肠系膜脂膜炎罕见，对肠系膜脂膜炎的病因及治疗认识不足，导致未对患者免疫抑制药物及糖皮质激素用药史引起足够重视也是 PCP 诊断延误的原因之一。

PCP 的一线治疗是甲氧苄啶－磺胺异甲噁唑静脉注射，因其具有良好的组织渗透性、快速反应和低成本等优点[5]。不良反应包括白细胞减少、血小板减少和包括史蒂文斯－约翰逊综合征在内的皮疹[32]。然而，这些反应在儿童中似乎不太常见[33]。也有研究支持卡泊芬净用于 PCP 的治疗。目前已有 TMP-SMZ 治疗 PCP 失败换用卡泊芬净治疗成功的报道[34]，亦有卡泊芬净联合 TMP-SMZ 成功治疗 PCP 且减少 TMP-SMZ 不良反应发生率的报道[35]。PCP 早期确诊治疗反应较好，多数可以得到恢复，更严重的病程往往认为继发于更具播散性的肺部炎症反应和诊断延迟[5] [36]。所以，临床工作中，应充分强调早期诊断的重要性，提高对非 HIV 感染合并 PCP 患者，乃至肠系膜脂膜炎合并 PCP 患者的认识。同时，提倡对于临床上疑诊 PCP 的患者，在第一时间，而不必须等待病原学证据确诊，果断、及时地给予经验性治疗。

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