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人类免疫缺陷病毒阴性的阴道残端浆母细胞淋巴瘤一例并文献复习 *

文 禄[1] 郭 英[2] 谢云霞[1] 范国庆[2] 陈协群[1] 杨 岚[1△]

(1 第四军医大学西京医院血液内科 陕西 西安 710032;2 第四军医大学西京医院病理科 陕西 西安 710032)

摘要 目的:浆母细胞淋巴瘤(PBL)为罕见的、侵袭性极强的 B 细胞淋巴瘤,好发于 HIV 阳性患者的口腔,本文报道了目前国内外首例 HIV 阴性的原发于阴道残端的 PBL,并通过文献复习总结了 PBL 的临床病理学特征、诊断及鉴别诊断、治疗及预后。**方法:**回顾分析该病例的临床病理学资料,并结合国内外相关文献进行讨论。**结果:**该患者诊断明确,HIV 阴性,以阴道残端起病,免疫组化示 MUM-1 阳性,不表达 CD20、CD79a 和 PAX-5,Ki-67 阳性率 73 %。EBER 原位杂交阴性。分子学诊断结果示:样品免疫球蛋白基因发生克隆性重排,未检到 TCR 基因克隆性重排。CHOP 样方案疗效不佳,部分缓解后疾病快速进展。该患者从确诊至死亡时间为 10.3 个月。**结论:**PBL 罕见,病情进展迅速,预后差,生存期短,在进行阴道残端肿瘤的诊断及鉴别诊断时,阴道残端 PBL 应纳入鉴别范围。

关键词: 浆母细胞淋巴瘤;人类免疫缺陷病毒;阴道残端

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A Case of Plasmablastic Lymphoma of the Vagina Stump without Human Immunodeficiency Virus Infection and Literature Review*

WEN Lu[1], GUO Ying[2], XIE Yun-xia[1], FAN Guo-qing[2], CHEN Xie-qun[1], YANG Lan[1△]

(1 Department of Hematology, Xijing Hospital, Xi'an, Shaanxi, 710032, China;

2 Department of pathology, Xijing Hospital, Xi'an, Shaanxi, 710032, China)

ABSTRACT Objective: Plasmablastic lymphoma (PBL) was a rare, aggressive variant of B-cell lymphoma that frequently occurred in the oral cavity of patients with human immunodeficiency virus (HIV) infection. We reported the first case of HIV-negative PBL with initial presentation of the vagina stump to investigate the clinicopathological characteristics, diagnosis, differential diagnosis, treatment and prognosis of PBL. **Methods:** The relevant clinicopathological materials of PBL patient was retrospected and a literature review was conducted. **Results:** The patient was explicitly diagnosed to be HIV negative with a vaginal stump mass as the main lesion. Immunohistochemical examination revealed that the tumor cells were positive for MUM-1, negative for CD20, CD79a and PAX-5. The Ki-67 proliferative index was 73%. The tumor cells were negative for EBV virus-encoded RNA in situ hybridization (EBER-ISH). Molecular diagnostic results showed immunoglobulin gene rearrangement in the sample analyzed. TCR gene rearrangement was not observed. CHOP-like regime showed no satisfactory effect and the disease progressed soon after a partial response. The patient died 10.3 months after the diagnosis. **Conclusions:** As PBL was a rare distinct variant with high malignancy, poor prognosis and shorter overall survival, it should be included as a differential diagnosis in cases of suspected vaginal stump tumor.

Key words: Plasmablastic lymphoma; Human immunodeficiency virus; Vagina stump

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前言

浆母细胞淋巴瘤(plasmablastic lymphoma,PBL)是一类罕见的、具有高度侵袭性的 B 细胞淋巴瘤,肿瘤细胞表现为类似于 B 免疫母细胞的大细胞弥漫性增殖,同时表达浆细胞相关抗原[1]。1997 年,Delecluse 等首次报道了 16 例原发于口腔的伴有特殊免疫表型的侵袭性非霍奇金淋巴瘤,其中 15 例患者人类免疫缺陷病毒(HIV)阳性,这类肿瘤被称为浆母细胞淋巴瘤[2]。由于其特殊性,2008 版的 WHO 淋巴及造血系统肿瘤分类将其划归为弥漫大 B 细胞淋巴瘤 (diffuse large B-cell lymphoma,DLBCL)的一种罕见亚型[1]。PBL 常发生于 HIV 阳性患者的口腔,近年来,原发于口腔外且 HIV 阴性的 PBL 在国内外多有报道。本文报道了首例原发于阴道残端且 HIV 阴性的 PBL,并结合文献探讨 PBL 的临床病理学特征、诊断及鉴别诊断、治疗及预后。

1 病例资料

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作者简介:文禄(1989-),女,硕士研究生,主要研究方向:造血调控,E-mail:wenlu2011d@sina.cn

△通讯作者:杨岚,电话:029-84775202,E-mail:yanglan@fmmu.edu.cn

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患者,女,53 岁。因“子宫全切术后 1 年半,左髋部疼痛 5 月,咳嗽 1 周入院”。患者于 2008 年 5 月因经期延长伴周期缩短 4 年于当地医院行全子宫切除 + 左侧阔韧带肌瘤剥除 + 阴道前后壁膨出、陈旧性会阴裂伤修补术。术后病理结果回报示子宫及阔韧带多发性平滑肌瘤。于 2009 年 8 月始无明显诱因出现左侧腰骶部、髋部及下肢疼痛,为持续性隐痛,伴左下肢凹陷性水肿,伴阴道排液及肛门下坠感,无明显阴道出血,1 月内体重减轻 5 kg。于 2009 年 12 月就诊于西京医院。妇科查体:外阴:已婚已产式,发育正常;阴道:通畅,黏膜光滑,残端可见直径约 2 cm 块状新生物,表面可见坏死物;宫颈、宫体缺如。三合诊:阴道残端可及 4.5 cm 包块,双侧未达盆壁;直肠黏膜光滑,指套无血染。实验室检查:HIV 阴性;乙肝五项全阴性;β-2 微球蛋白 3.10 mg/L;乳酸脱氢 195 IU/L;铁蛋白 473 μg/L;血清蛋白电泳、免疫球蛋白定量、轻链定量未见明显异常。CT 示:肝脏及右肾多发占位,左侧肾上腺结节符合转移瘤,胰头部转移瘤可能。MR 示:阴道后壁腔内异常改变,结合病史,考虑阴道残端癌改变,盆腔内及双侧股骨、髂骨、骶骨病变,考虑转移可能。免疫组化结果(阴道残端,2009-12-25)回报示:MUM-1(+),CD20 (-),CD79a (-),PAX-5 (-),Ki-67 阳性率 73%,CD10(-),CD30(-),CD45(+/-),CD56(-),CD138(-),CD38(-),ALK(-),S-100(-),HMB45(-),AFP(-),EBER 原位杂交(-);分子学诊断结果示:样品免疫球蛋白基因发生克隆性重排,未检到 TCR 基因克隆性重排。骨髓活检提示淋巴瘤累及骨髓。结合形态学、免疫组化、基因重排结果及实验室检查,诊断为(2009-12-25):浆母细胞淋巴瘤,临床分期为ⅣB。患者于 1 月初就诊于西京医院血液科,行 CHOPE 方案化疗 4 疗程后达部分缓解 (PR),再次行 CHOPE 方案化疗 2 疗程后病情进展 (PD),盆腔 MR 可见 10.4× 10.6× 10.2 cm 包块,确认病变性质后行盆腔局部放疗(61 Gy/23 次),复查盆腔 MR 提示包块缩小。后患者病情再次进展,于确诊本病后 10.3 个月死亡。

2 讨论

PBL 是一类罕见的,好发于 HIV 阳性患者口腔的具有特殊免疫表型的 B 细胞淋巴瘤。近年来,文献有报道发生于皮肤、皮下组织、胃、肛周、肺、淋巴结等部位的 HIV 阴性 PBL[3]。本例是首例发生于阴道残端的 HIV 阴性 PBL。

2.1 流行病学

根据文献报道,81 %的 PBL 发生于 HIV 阳性患者,占所有 HIV 相关淋巴瘤的 2.6 %[4]。在 HIV 阴性患者中,29 %的患者有其它方面的医源性免疫抑制,如因器官移植、自身免疫性疾病、恶性肿瘤等应用免疫抑制剂的患者[5]。其余 HIV 阴性患者未发现处于免疫抑制的状态。PBL 好发于成年男性,HIV 阳性患者的中位发病年龄为 38 岁 (男 / 女:7/1)[6],HIV 阴性患者的中位发病年龄为 57 岁(男 / 女:1.9/1)[5]。

2.2 发病机制

PBL 的发病机制仍未阐明,目前认为 PBL 起源于后生发中心的终末分化的活化 B 细胞,处于免疫母细胞转化成浆细胞的阶段,这些细胞已经历过类别转换和体细胞的高频突变[9]。此过程中的染色体和细胞内分子信号通路异常可能导致了细胞恶变。

MYC 基因重排是 PBL 中首个被发现的细胞遗传学异常,也是 PBL 中最常见的染色体结构异常 [7, 8],在某种意义上和 Burkitt 淋巴瘤相似,不同的是后者起源于生发中心 B 淋巴细胞[9]。Valera 等采用荧光原位杂交(FISH)技术测得 41 例 PBL 中有 20 例发生了 MYC 基因重排,占 49 %,其中 IgH 基因是 MYC 基因重排的主要伙伴;31-41 %的病例检测到了 BCL2、BCL6、MALT1 或 PAX5 基因扩增[7]。MYC 基因重排与 PBL 的浆母细胞形态及侵袭性的临床过程有关,在 PBL 的发病中起着重要的作用[10]。其具体机制还需进一步研究。

EB 病毒被认为与 PBL 的发病密切相关[11]。在 HIV 相关的 PBL 病例中,74 %的病例为 EB 病毒阳性;在 HIV 阴性的 PBL 病例中,这一百分比为 54 %[5,6,12,13]。EB 病毒作为最早被发现的与人类肿瘤相关的病毒,可在 B 淋巴细胞中形成潜伏感染,在机体免疫力下降时,激活且增殖[14]。EB 病毒感染导致 PBL 的原因,可能是由于 CD4 阳性 T 细胞持续减少,破坏了 CD8 阳性细胞毒性 T 细胞介导的抗病毒免疫,从而不能有效控制 EB 病毒感染的 B 细胞增生,在此过程中某个克隆增生形成优势,最终形成 PBL[15]。此外,少数病例并不伴有 EB 病毒感染,也没有免疫异常病史[16]。

Notch1 在淋巴祖细胞发育过程中的 T 系、B 系选择中是重要的调节信号,可抑制 B 系淋巴细胞的特殊转录因子的表达,Seegmiller 等发现,在 9 例 PBL 中免疫组化检测到 Notch1 均为阳性。因此,PBL 的发生还可能与 Notch1 信号途径的活化有关[17]。此外,由于不同文献得出了相互冲突的数据结果,PBL 与人类疱疹病毒 8 型(HHV-8)的相关性还有待商榷[18,19]。

2.3 临床特点

对于 HIV 阳性的 PBL,口腔为典型的发病部位,口腔外发病部位最常见的是胃肠道、淋巴结和皮肤 [6]。在 HIV 阴性的 PBL 中,淋巴结外发病占 82 %,口腔和胃肠道也是最常见的原发部位[5],但其在口腔的发病率(16 %)明显低于 HIV 阳性病例(58 %)[20]。据文献报道,还有少数病例原发于中枢神经系统、副鼻窦,纵膈,肺,肝,睾丸等。本文报道的病例为首例发生于阴道残端的 HIV 阴性 PBL。所有的 PBL 中,60 %的病例发病时表现为临床Ⅲ期或Ⅳ期;对于 HIV 阳性患者,临床分期呈双峰分布,超过 80 %的患者表现为Ⅰ期或Ⅳ期[9]。

2.4 诊断与鉴别诊断

浆母细胞淋巴瘤形态学上类似于 B 免疫母细胞或浆细胞的大型异型淋巴样细胞弥漫性增生,可见星空现象及凋亡小体,肿瘤细胞圆形或者椭圆形。免疫表型往往具有极典型的浆细胞表型 (如 CDl38、CD38、MUM-1);但很少表达或弱表达 B 细胞标记物(如 CD20、PAX-5)[1];具有较高增殖活性(Ki67 超过 60 %)[21]。Colomo 等认为 PBL 免疫表型差别大,MUM-1 是唯一恒定的阳性指标[12],本例 PBL 的免疫表型(CDl38 阴性、CD38 阴性、MUM-1 阳性)与上述结论一致。

PBL 的肿瘤细胞的形态学特点及免疫表型容易与某些恶性疾病混淆,如除 PBL 以外的具有浆母细胞分化的大 B 细胞淋巴瘤,CD20 及 PAX-5 阴性有助于排除其他类型的侵袭性 B 细胞淋巴瘤,CD30 及 ALK 阴性则可与间变性大细胞淋巴瘤鉴别。PBL 与浆母细胞骨髓瘤(plasmablastic plasma cell myeloma,PBPCM)在形态学上极为相似,并且两者有很多相同的免疫组

化特点,但治疗方法却不同,故其鉴别诊断十分重要。M 蛋白、骨质破坏的出现以及 CD56、cyclin D1 的表达常发生在 PBPCM 患者中;此外,CDl0 在 69 %的 PBL 患者中表达,而仅在 29 %的 PBPCM 患者中表达[18];EB 病毒编码的 RNA(EBER)在 PBPCM 患者中几乎均为阴性[18]。有研究提出,PAX5、CD20 阴性或弱阳性,同时 Blimp-1、XBP-1 阳性可作为诊断 PBL 的可靠证据[22]。

2.5 治疗及预后

有关 PBL 的治疗主要以早期化疗为主。目前,CHOP 或 CHOP 类似方案被广泛应用,可获得良好的总有效率,但伴随而来的是高复发率及低生存率 [23]。据文献报道,当采用比 CHOP 方案强度更大的治疗方案 (如 EPOCH,HyperCVAD,或 CODOX-M/IVAC 方案)时,患者并未获得生存优势[23]。在 HIV 阳性患者,辅以高活性抗逆转录病毒治疗 (highly active antiretroviral therapy,HAART),可改善预后[23]。有临床研究显示,应用硼替佐米单药或联合地塞米松、吉西他滨、奥沙利铂等治疗有效[24],但尚无大宗病例报道,且均为暂时性缓解。在接受用化疗的病人中总有效率达到 77 %,其中完全缓解占 46 %,部分缓解占 31 %,未接受化疗的病人中位生存期仅为 3 个月[9]。PBL 总体预后差,HIV 阳性患者中位生存期为 15 个月,而 HIV 阴性患者的仅为 9 个月[5,6]。据文献报道,年龄大于 60 岁、临床分期为Ⅲ期或Ⅳ期、骨髓浸润、未接受治疗、免疫抑制均是预后不良的因素[5,9]。

综上所述,PBL 是非霍奇金淋巴瘤的一种特殊类型,好发于 HIV 阳性患者的口腔,随着国内外多例原发于口腔外 HIV 阴性 PBL 的报道,PBL 的定义将越来越宽泛。PBL 具有高度侵袭的生物学行为,因其临床特征不够典型,所以要结合临床特征、免疫表型和分子生物学特征等综合分析。本文报道了首例发生于阴道残端的 HIV 阴性 PBL 患者,发病时已为Ⅳ期,病情进展迅速,预后差,生存期短,因此,在进行阴道残端肿瘤的诊断及鉴别诊断时,PBL 应列入考虑,这对其诊断、预后评估及合理治疗均具有重要意义。

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