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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*

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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,未检测到 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*

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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 retrospectively reviewed 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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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5kg。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2cm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4.5cm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3.10mg/L;乳酸脱氢酶195IU/L;铁蛋白473 μ g/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、髌骨、胫骨病变,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,未检测到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 $\times$ 10.6 $\times$ 10.2cm包块,确认病变性质后行盆腔局部放疗(61Gy/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CD138、CD38、MUM-1);但很少表达或弱表达B细胞标记物(如CD20、PAX-5)^[11];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(CD138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,CD10 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^[23]。

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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