ACG CASE REPORTS JOURNAL

CASE REPORT | COLON

Unexpected Discovery at Resection Site: Plasmablastic Lymphoma After Polypectomy

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ABSTRACT

Plasmablastic lymphoma (PBL) is a rare, aggressive subtype of diffuse large B-cell lymphoma that has usually been associated with human immunodeficiency virus and Ebstein–Barr virus infections. Commonly seen in the oral cavity, it can affect any part of the gastrointestinal tract. This report describes a unique case of PBL that appeared in an 81-year-old immunocompetent patient. During an initial colonoscopy, a 2-cm polyp in the distal transverse colon was removed via full-thickness resection with pathology showing tubular adenoma with foci of high-grade dysplasia. After 7 months, a repeat colonoscopy found a 3-cm sessile polyp at the site of resection, which, after removal, was found to be a PBL. This is the first case described in the literature regarding the growth of PBL after full-thickness resection of a tubular adenoma in such a short interval.

KEYWORDS: plasmablastic lymphoma; full thickness resection; polypectomy; adenoma

INTRODUCTION

Plasmablastic lymphoma (PBL) is a rare subtype of diffuse large B-cell lymphoma, first defined as a separate entity by the World Health Organization classification of lymphoid neoplasms, fourth edition (2008) and reconfirmed in the 2017 revision and fifth edition classification (2022).[1–3] This entity is usually extranodal, mainly in the oral cavity, and is frequently associated with Human Immunodeficiency Virus (HIV) and Epstein Virus infection (EBV).[1] It has been reported in extraoral sites, with the gastrointestinal tract being the most frequent location.[4,5] PBLs are characterized by an immunoblastic or plasmablastic cytomorphology and a plasmocytic immunophenotype with negative B-cell markers.[1,6] In addition, they are EBV-positive with a high Ki-67 (proliferative index) and exhibit an aggressive clinical course with early dissemination.[4]

We describe a case of PBL that appeared 6 months after a polypectomy on a previous full-thickness resection (FTR) site.

CASE REPORT

An 81-year-old White man with a history of colon polyps presented to our outpatient gastroenterology clinic for a follow-up colonoscopy and polypectomy after a recent colonoscopy done in a different clinic found a 2-cm polyp in the distal transverse colon and tattooing with biopsies were done. Repeat colonoscopy showed the polyp with evidence of previous tattooing and fibrosis at the base (Figure 1). A submucosal lifting agent was used to provide a lift for endoscopic mucosal resection (EMR), but the polyp did not lift adequately, so a FTR using a commercially available colonic FTR device was made. Pathology reports showed a tubular adenoma with foci of high-grade dysplasia without evidence of invasive carcinoma and clear surgical margins of adenomatous changes. A surveillance colonoscopy was planned in 6 months.

Seven months later, during the repeat colonoscopy, a 3-cm sessile polyp was seen in the distal transverse colon at the site of the previous FTR site on top of a previously placed tattoo (Figure 1). A mucosal resection was performed with submucosal injection with a lifting agent for adequate lift from the underlying tissue, followed by hot snare polypectomy in piecemeal. Small residual

ACG Case Rep J 2025;12:e01633. doi:10.14309/crj.0000000000001633. Published online: February 27, 2025

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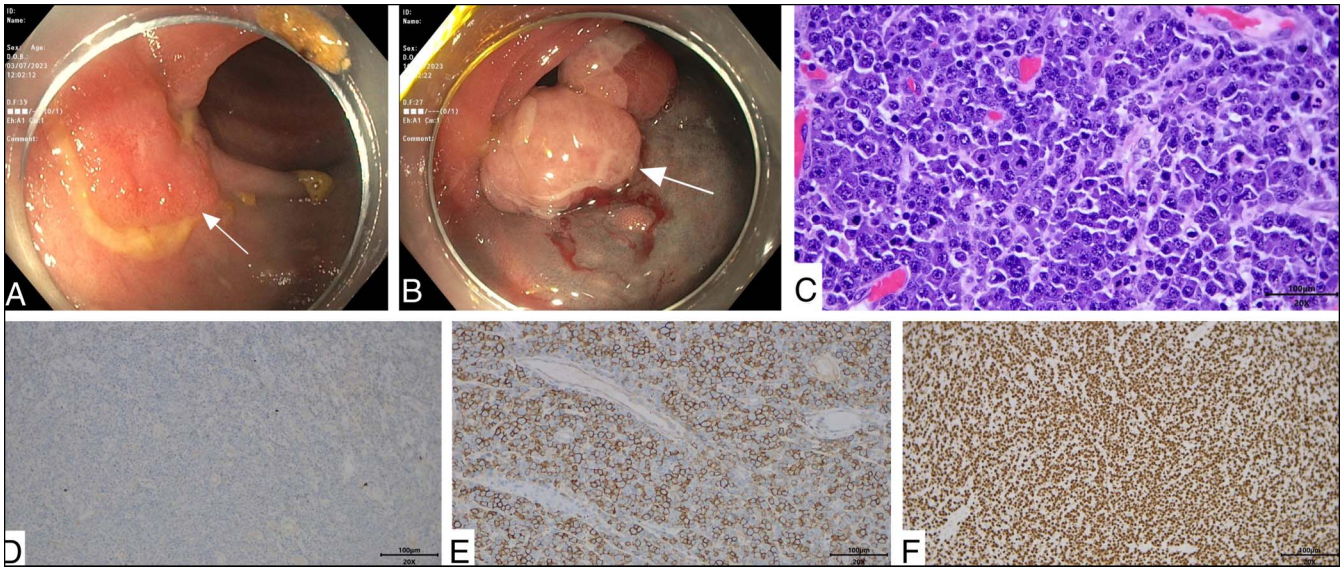

Figure 1. (A) A 2-cm polyp at initial colonoscopy before full-thickness resection. (B) A 3-cm polyp at 6-month interval at previous full-thickness resection site. (C) Hematoxylin and eosin stain showing sheets of plasmablastic cells infiltrating the colonic mucosa (magnification ×20). (D) CD20- cells (magnification ×20). (E) CD138+ cells (magnification ×20). (F) Ki-67>90% positive (magnification ×20).

tissue was removed using avulsion technique utilizing the hot biopsy forceps. The snare tip was used to secure hemostasis, and clips were applied to the mucosal defect to close it. The pathology report showed a colonic mucosa infiltrated by sheets of plasmablastic cells with brisk mitoses. The tumor cells were CD138+, Mum1+, CD56^{+}, c-Myc+, CD20−, Pax5−, CD3−, Bcl-2/6-, HHV8−, EBV ISH +. Ki 67 >90%, exhibiting kappa light chain restriction, wild-type P53 expression, C-Myc, and clonal B-cell gene rearrangement (Figure 1).

A diagnosis of PBL was made. It is the first case described where such a rare malignancy appeared at the site of a previous FTR of a tubular adenoma.

The patient was asymptomatic before diagnosis, denied B symptoms and laboratory testing showed normal white count and lactate dehydrogenase. The positron emission tomography scan did not show any nodal or other organ involvement. HIV testing was negative. He underwent treatment with dose-adjusted etoposide, prednisone, vincristine, cyclophosphamide, and doxorubicin with complete remission of the disease.

DISCUSSION

PBL is an aggressive, rare type of extranodal diffuse large B-cell lymphoma usually associated with HIV and EBV infection.[5] Immunocompetent patients can be affected and are, on average, older at the time of diagnosis (mean age of 55 years compared to 42 years in patients with HIV). Prognosis is usually poor, with median overall survival ranging from 9 to 15 months.[4,6] Nevertheless, a recent study found that patients with PBL treated with multiagent chemotherapy had an improved survival (58.6 months).[7] The International Prognostic Index is a scoring system used to risk-stratify patients with advanced lymphomas such as PBL. However, several retrospective studies showed that prognosis relies mainly on performance status and disease stage.[4,8] Unfortunately, patients with PBL with myc gene rearrangements, like our patient, have shorter overall survival.[4,5,9]

The is the first case described in the literature, regarding the growth of PBL after FTR of a tubular adenoma in such a short interval. This presentation of PBL with a limited involvement to the site of a previous endoscopic resection of an adenomatous polyp, as well as the lack of the known risk factors and associated diseases with this subtype of lymphoma, is unusual and has not been reported in the literature. This case raises a question about the accuracy of the diagnosis of PBL. It is unclear if this is an incidental finding of a true case of PBL limited to the site of a prior adenoma or if there is a possible causative relationship secondary to the manipulation by FTR.

According to the literature, the usual complications after an endoscopic submucosal resection (ESD) or EMR are bleeding (immediate and delayed), perforation, post-ESD coagulation syndrome, and stenosis.[10,11] Moreover, the rate of local recurrence after an ESD for large colorectal neoplasm at 1 and 5 years was 0.2% and 1.6%, respectively.[12] Regarding post-EMR sites, a recent prospective study has identified scarring in 99.7% of patients.[13,14] In addition, a retrospective study found that, after gastric EMR, biopsies showed inflammatory changes (100%), epithelial atypia (60.6%), clear cell degeneration (15.2%), and signet-ring cell-like change (6.1%).[15] The last 2 histological findings could mimic residual adenocarcinoma and, if not properly assessed, could lead to unnecessary therapeutic interventions. Buried atypical glands, which can mimic dysplasia and malignancy, can be seen in post-EMR biopsies as well.[15,16]

Another consideration should be made to the possibility of a non-PBL lymphocytic reaction related to the previous mechanical manipulation and injection of a lifting agent and their subsequent tissue reaction. A case series described a granulomatous reaction with colonic mass formation mimicking invasive adenocarcinoma after the injection of a certain lifting agent.[17] Another case series reported the presence of foreign body giant cells and eosinophils in postlifting inflammatory lesions.[18] However, the appearance of high-grade lymphoma-like changes has not been described in the literature.

Similar cases should be reported in the future to attempt to establish this association should this be indeed a plausible etiology of this rare form of lymphoblastic disorder. Regardless, it should be noted that rapid polyp growth should be concerning for an aggressive malignancy and proper treatment and follow-up could be key to patient survival.

DISCLOSURES

Author contributions: Original concept: R. Njeim, Y. El Douaihy. Data/Pathology collection: Y. Singh. First draft of the manuscript: R. Njeim, M. Abureesh, Y. Singh. R. Njeim and Y. El Douaihy reviewed and edited the manuscript. Y. El Douaihy is the article guarantor.

Financial disclosure: None to report.

Informed consent was obtained for this case report.

Received April 27, 2024; Accepted February 3, 2025

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