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CASE REPORT | COLON

Conundrum of a Large Bowel Neoplasm: Collision Tumor

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ABSTRACT

A 79-year-old Hispanic man was admitted to the intensive care unit with symptomatic iron-deficiency anemia and watery diarrhea. Radiological images revealed diffuse colonic wall thickening, a soft-tissue fullness in the ascending colon, and multiple mesenteric lymphadenopathies. Colonoscopy showed multiple aphthous ulcers throughout the colon and a large deep ulcer with irregular raised borders in the rectosigmoid area. Histological exam of the ulcers showed severe ulcerative colitis, while biopsy of the deep ulcer revealed a well-differentiated adenocarcinoma. Colectomy specimen was consistent with colliding diffuse large B-cell lymphoma and adenocarcinoma.

INTRODUCTION

A collision tumor is characterized by 2 histologically distinct malignancies coexisting in the same location. Collision of malignancies occurs between primary synchronous tumors originating in the same organ or between metastases from other sites. Each malignancy has a distinct boundary and is separated by non-neoplastic stroma without histologic admixture or transitional area between them. Collision tumors involving synchronous colorectal cancer (CRC) and lymphomas are extremely rare.[1] While CRC is the fourth most common malignancy, primary gastrointestinal (GI) lymphomas are infrequent, accounting for 2% of all GI cancers.[2,3] The most common sites of primary GI lymphomas are the stomach and the proximal small bowel, with less frequent involvement of the colon (5%).[4] Different predisposing factors for the development of GI lymphomas include environmental and infectious agents, immune status, and inflammatory bowel disease.[3]

CASE REPORT

A 79-year-old Hispanic man with arterial hypertension was admitted to the intensive care unit with a 2-week history of weakness, fatigue, and non-bloody watery diarrhea. The physical examination revealed an acutely ill man with sinus tachycardia and hypotension. Abdominal and digital rectal exams were normal. Laboratories were remarkable for iron-deficiency anemia (hemoglobin 5 g/dL). An abdominal computed tomography showed diffuse pancolonic wall thickening, a soft-tissue fullness in the ascending colon, and multiple intra-abdominal lymphadenopathies. Colonoscopy revealed multiple aphthous ulcers throughout the colon and a large, deep, ulcerated lesion at the rectosigmoid region. Biopsies of the rectosigmoid ulcer were compatible with a moderately differentiated adenocarcinoma, while those of the aphtous ulcers were consistent with severe ulcerative colitis (Figure 1).

Subsequent colonoscopy found a rapidly growing rectosigmoid carcinoma almost occluding the lumen in the background of a severe pancolitis. A total proctocolectomy with end ileostomy and partial omentectomy was performed. Histological examination demonstrated invasion of the rectosigmoid adenocarcinoma into perirectal

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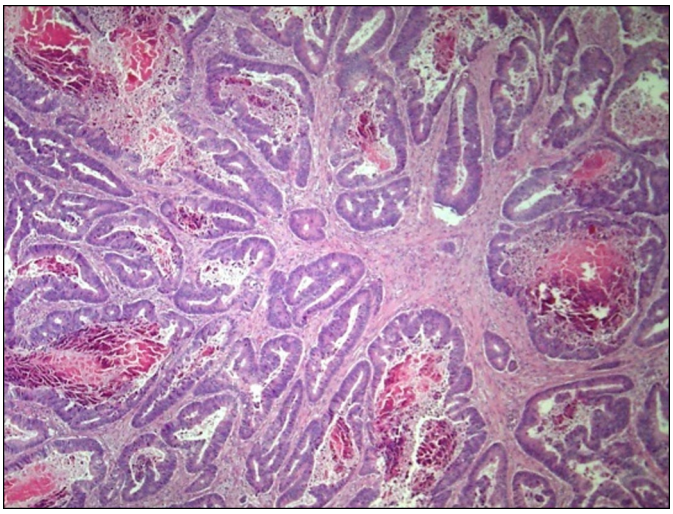

Figure 1. Neoplastic infiltrating glands consistent with adenocarcinoma.

tissue and clean resection margins. Four of 24 lymph nodes were positive for a metastatic stage III CRC. Adjacent to the carcinoma, a diffuse mononuclear large cell infiltrate was positive for bcl-2 and CD20 immunoperoxiadases, which is consistent with a diffuse large B-cell lymphoma (Figures 2 and 3). Evaluation for high-grade B cell lymphoma included bone-marrow aspiration, flow cytometry, serum levels of β2-microglobulin, uric acid, lactate dehydrogenase, and HIV, all of which were negative.

The patient received chemotherapy and local radiotherapy as a result of aggressive tumor behavior. The treatment consisted of 4 cycles of cyclophosphamide, hydroxydaunomycin, vincristine, prednisone, and rituximab, followed by radiotherapy. The remaining chemotherapy cycles were given at the end of radiotherapy. After completing treatment for lymphoma, adjuvant chemotherapy with 5-fluorouracil, leucovorin, and oxaplatin (FOLFOX) for CRC was also administered. Following aggressive medical and surgical management, the patient survived 30 months after diagnosis.

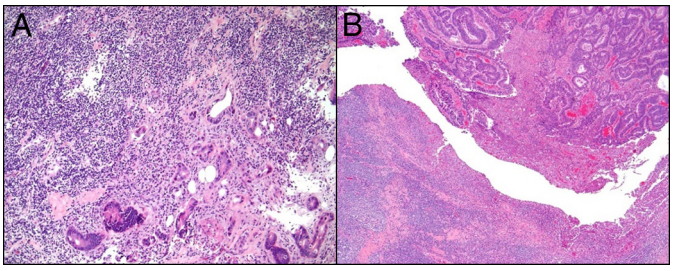

Figure 2. (A) Hematoxylin and eosin stain showing small, round lymphocytic infiltrate next to and admixed with a moderately differentiated adenocarcinoma. (B) Evidence of colliding lymphoma (upper) and adenocarcinoma (lower).

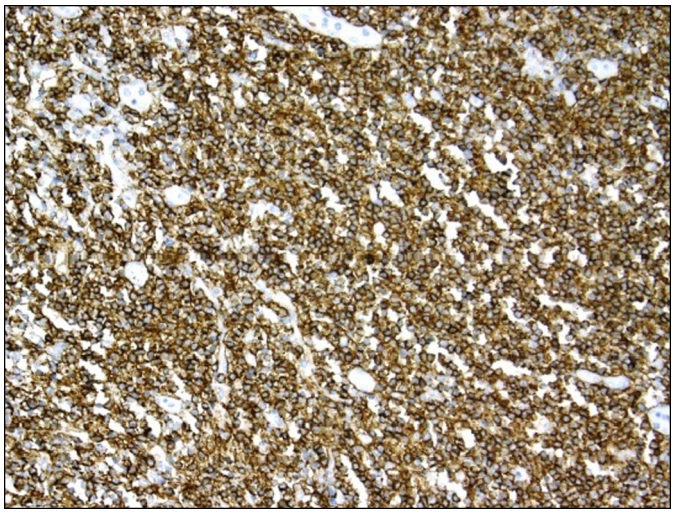

Figure 3. Immunostaining showing a section of the neoplastic lymphoid cells, which show membranous staining with CD20, consistent with a B-cell lymphoma.

DISCUSSION

Colliding lesions of colonic lymphoma and CRC are rare. Adenocarcinoma is the most common colonic malignancy but only presents with synchronous or metachronous tumors in 5% of cases.[5,6] In contrast, colorectal lymphoma is extremely infrequent, representing <0.5% of all primary CRC.[4] The clinical presentation of collision tumors is not specific and depends primarily on the affected organ. Our patient presented with symptomatic anemia without evidence of bleeding, which led us to perform a diagnostic colonoscopy.

The evolution of collision tumors is intriguing in terms of carcinogenesis of malignant lymphoma and progression to carcinoma.[5] One hypothesis suggests that tumors arise in continuity through an accidental event and that the presence of one tumor precipitates the adjacent tumor by altering the microenvironment.[4,6] A lymphomatous process may be the initial event, compromising the patient's immune system.[4] Nonetheless, there is no evidence that immunodeficiency induces activation of oncogenes or inactivation of tumor-suppressor genes.[5] In our case, ulcerative colitis could have been the precipitating factor that led to dysplastic changes evolving into malignancy.

Optimal management of GI lymphomas is the subject of debate and has not been studied in randomized trials.[4] In cases of collision tumors, the influence of one malignancy on the behavior of the other greatly increases the complexity of treatment.[1] Our approach consisted of surgical resection followed by directed chemotherapy and radiotherapy, and finally chemotherapy for CRC. This case accentuates how the choice of treatment invariably depends on the patient's age and performance status, clinical scenario, histological subtype, and extent of disease.

Little is known regarding colorectal lymphomas in minority groups or its association with CRC.[7] Studies have demonstrated that, in Hispanics, CRC is diagnosed at an earlier age and with a more advanced stage, and it tends to have a more aggressive course.[7] Studies regarding incidence, prevalence, and epidemiology of collision tumors and their clinical presentation and response to treatment are needed.

DISCLOSURES

Author contributions: All authors wrote and edited the manuscript. DH Toro is the article guarantor.

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Informed consent was obtained for this case report.

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REFERENCES

1. Genovese F, Becchina G, Nagar C, et al. Primary diffuse large b-cell lymphoma developing within a rectal tubular adenoma with low-grade dysplasia: A case report. *J Med Case Rep*. 2014;8:103.
2. Jemal A, Siegel R, Ward E, et al. Cancer statistics, 2008. *CA Cancer J Clin*. 2008;58:71.
3. Lin HH, Jiang JK, Lin JK. Collision tumor of low-grade B-cell lymphoma and adenocarcinoma with tuberculosis in the colon: A case report and literature review. *World J Surg Oncol*. 2014;12:147.
4. Redman RA, Chesney J. Interesting collision between an indolent B-cell lymphoma and a microsatellite unstable adenocarcinoma of the colon. *J Hematol Thromb Dis*. 2013;1:110.
5. Sasaki S, Hatanaka K, Sahara N, et al. Collision tumor of primary malignant lymphoma and adenocarcinoma in the colon: Report of a case. *Surg Today*. 2010;40:975–81.
6. Bhattacharya A, Saha R, Biswas J, et al. Collision tumors in the gastrointestinal tract: A rare case series. *Int Med Case Rep J*. 2012;5:73–7.
7. Cruz-Correa M. Increasing colorectal cancer burden among young US Hispanics: Is it time to change current screening guidelines? *Dig Dis Sci*. 2013;58(7):1816–8.