Refractory fulminant colitis requiring surgical intervention in a patient with ulcerative colitis on atezolizumab therapy for small cell lung cancer: An atypical case

P168

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Introduction

Atezolizumab is a programmed death-ligand 1 (PD-L1) targeted antibody that prevents binding of PD-L1 to specific T-cell receptors, thereby increasing anti-cancer immunity. It has been regarded as a useful first-line treatment in patients with small cell lung cancer with a more tolerable side effect profile than chemotherapeutic agents. However, few studies focusing on the severity of adverse effects from immune checkpoint inhibitors (ICPI) have been previously reported, particularly, acute fulminant colitis requiring surgical invention.

Case Presentation

We report a case of fulminant colitis that was refractory to high dose corticosteroid treatment in a patient with known ulcerative colitis (UC) undergoing treatment for small cell lung cancer (SCLC) with atezolizumab. We performed an exploratory laparotomy, pancolectomy, and creation of an ileostomy. The patient had an uncomplicated postoperative course and was discharged from the hospital on postoperative day 12.

Discussion

The upregulation of PD-L1 expression in patients with ulcerative colitis may play a significant role in an imbalanced T-helper cell response, creating a pro-inflammatory state. The use of ICPIs to treat SCLC has been reported to increase the risk of developing inflammatory colitis. The use of atezolizumab in a patient with known inflammatory bowel disease (IBD) may predispose this population to a higher risk of developing severe inflammatory colitis.

Coronal View Moderate Pancolitis



Figure 1: Significant bowel wall thickening to cecum, ascending, and descending colon

Ulcerative Colitis-Associated Low-Grade Dysplasia

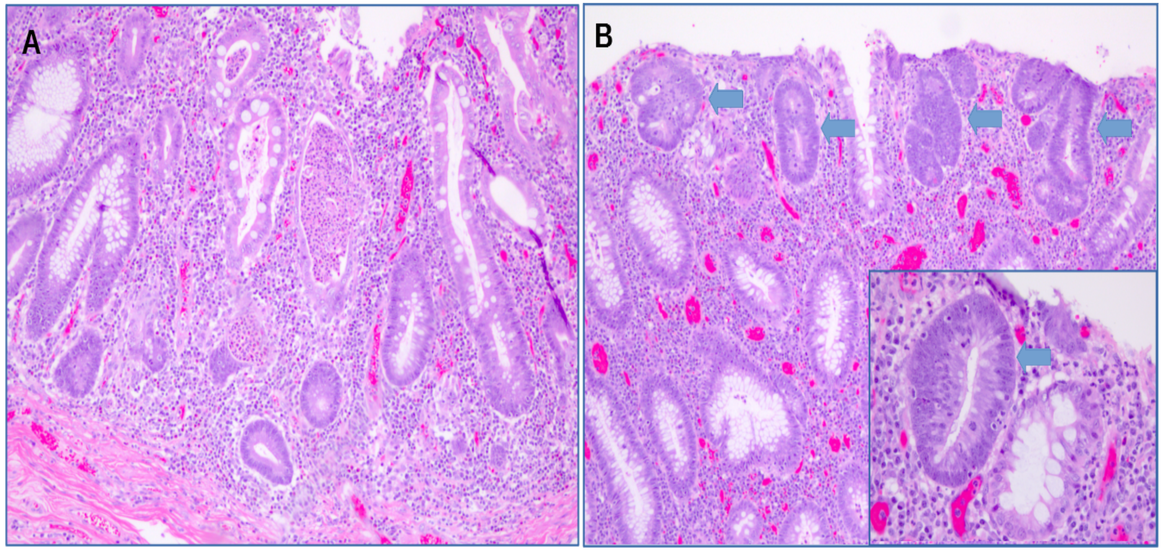


Figure 2: (A) Severe colitis with acute inflammatory activity, crypt distortion, reactive crypt epithelial change. **(B)** Low-grade dysplasia (arrows) on surface, indicating lack of surface maturation. Dysplastic gland (arrow in the insert) epithelial cells showing pseudostratified, crowded, elongated and hyperchromatic nuclei, compared to adjacent non-dysplastic gland. Preserved nuclear polarity.

Conclusion

We present an unusual complication associated with medical intervention in an immunocompromised patient without an established pathophysiology. The suspicion of using ICPIs in patients with IBD as a potential cause for the development of fulminant colitis is relevant and essential in the diagnostic workup for this patient population complaining of significant gastro-intestinal symptoms.

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