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Case Report

Successful Treatment of Steroid-Refractory Immune Checkpoint Inhibitor–Related Colitis With Vedolizumab: A Case Report and Literature Review

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Abstract

A 72-year-old man with metastatic lung adenocarcinoma developed severe immune checkpoint inhibitor–related colitis after nivolumab therapy. The patient presented with frequent diarrhea and dehydration. Infectious causes were excluded, and colonoscopic findings were consistent with active colitis. Despite high-dose intravenous corticosteroid treatment, no clinical improvement was observed, and the condition was considered steroid-refractory. Vedolizumab was initiated as salvage therapy, resulting in a rapid clinical response, with a marked reduction in stool frequency and improvement in symptoms. Corticosteroids were successfully tapered, and no recurrence was observed during early follow-up.

Keywords: Immune checkpoint inhibitor, immune-related colitis, nivolumab, vedolizumab.

INTRODUCTION

Immune checkpoint inhibitors (ICIs) have significantly improved outcomes in advanced malignancies, including non–small cell lung cancer, by enhancing T-cell–mediated antitumor activity. However, immune activation may lead to immune-related adverse events (irAEs), among which colitis is one of the most common gastrointestinal toxicities.[1,2] Although most cases respond to corticosteroids, a subset of patients develop steroid-refractory disease requiring additional immunosuppressive therapy. Vedolizumab, a gut-selective anti-integrin agent, has emerged as a potential treatment option in such cases.[3,4] Here, we present the case of a patient with steroid-refractory nivolumab-associated colitis who was successfully treated with vedolizumab.

CASE PRESENTATION

A 72-year-old man was evaluated in August 2025 for dyspnea and cough. Thoracic computed tomography revealed a lobulated, centrally necrotic mass measuring 7 cm at its longest diameter in the middle lobe of the right lung. For staging, positron emission tomography-computed tomography demonstrated intense fluorodeoxyglucose uptake in the primary lung mass, as well as findings consistent with bone metastases in the right humerus and eighth rib. Core needle biopsy of the lung mass showed focal positivity for thyroid transcription factor-1 and negativity for p63 and p40, consistent with lung adenocarcinoma. The patient received first-line chemotherapy with carboplatin and paclitaxel at another institution. After three cycles of chemotherapy, radiological progression was observed, and second-line treatment with nivolumab was initiated in November 2025.

After the third cycle of nivolumab, the patient presented to the outpatient clinic with a 1-week history of diarrhea occurring approximately 20 times per day, containing blood and mucus, accompanied by deterioration in general condition and reduced oral intake. He was admitted to the oncology ward with a preliminary diagnosis of immune checkpoint inhibitor–related colitis. In 2003, he had undergone total laryngectomy for laryngeal carcinoma followed by adjuvant radiotherapy; he had not received chemotherapy and had remained in remission. The patient also had a history of coronary artery disease. Five months before the current admission, he had been hospitalized for non-ST-elevation myocardial infarction (NSTEMI), underwent coronary angiography, and received coronary stent implantation. His current medications included acetylsalicylic acid, clopidogrel, metoprolol, atorvastatin, levothyroxine, inhaled ipratropium bromide and salbutamol, and granisetron. On admission, he denied fever, chills, or abdominal pain. The patient had a tracheostomy. Physical examination revealed dry oral mucosa and decreased skin turgor, consistent with dehydration. Repeated stool studies performed over 3 consecutive days were negative for infectious pathogens, including Giardia antigen,

stool parasite cyst and trophozoite examination, Clostridioides difficile antigen, and stool culture. Stool microscopy revealed leukocytes and erythrocytes. The fecal calprotectin level was greater than 840 µg/g. Colonoscopy was performed up to the mid-descending colon; further advancement was not possible because of solid fecal material. In the examined segments, the colonic mucosa appeared hyperemic, edematous, and friable, with diffuse superficial ulcers covered by confluent white exudates. The normal mucosal and submucosal vascular pattern was absent (Figure 1A-B). Histopathological examination of biopsy specimens demonstrated marked mixed inflammatory cell infiltration, moderate cryptitis, surface epithelial injury, moderate crypt atrophy, moderate crypt architectural distortion, and lymphoid hyperplasia, consistent with active colitis. Cytomegalovirus immunohistochemistry was negative.

Based on these findings, the patient was diagnosed with nivolumab-related colitis, and treatment with intravenous methylprednisolone at a dose of 1 mg/kg/day was initiated. Despite 1 week of corticosteroid therapy, stool frequency did not decrease. After gastroenterology consultation, vedolizumab (300 mg IV) was initiated at weeks 0, 2, and 6, with maintenance dosing every 8 weeks. The patient received the first induction dose on February 6, 2026, and the second induction dose on February 20, 2026. After vedolizumab therapy, his clinical condition improved significantly, with stool frequency decreasing to 4 times per day and stool consistency normalizing. Nivolumab therapy was permanently discontinued, and treatment with pemetrexed was planned. The patient's clinical course, including oncologic treatment and the development of immune-related colitis, is summarized in Figure 2.

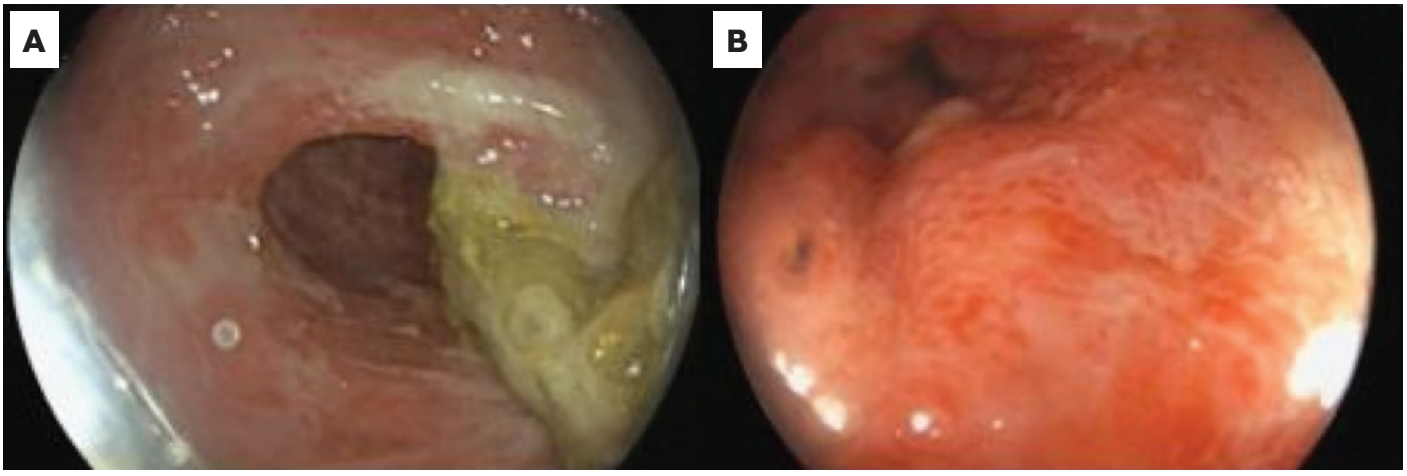

Figure 1. Colonoscopic findings of immune checkpoint inhibitor–associated colitis. **(a)** Diffuse hyperemia, mucosal edema, and friability with confluent superficial ulcerations covered by white exudates. The normal vascular pattern is absent. **(b)** Marked erythema and edema with loss of mucosal and submucosal vascular architecture, consistent with severe inflammatory activity.

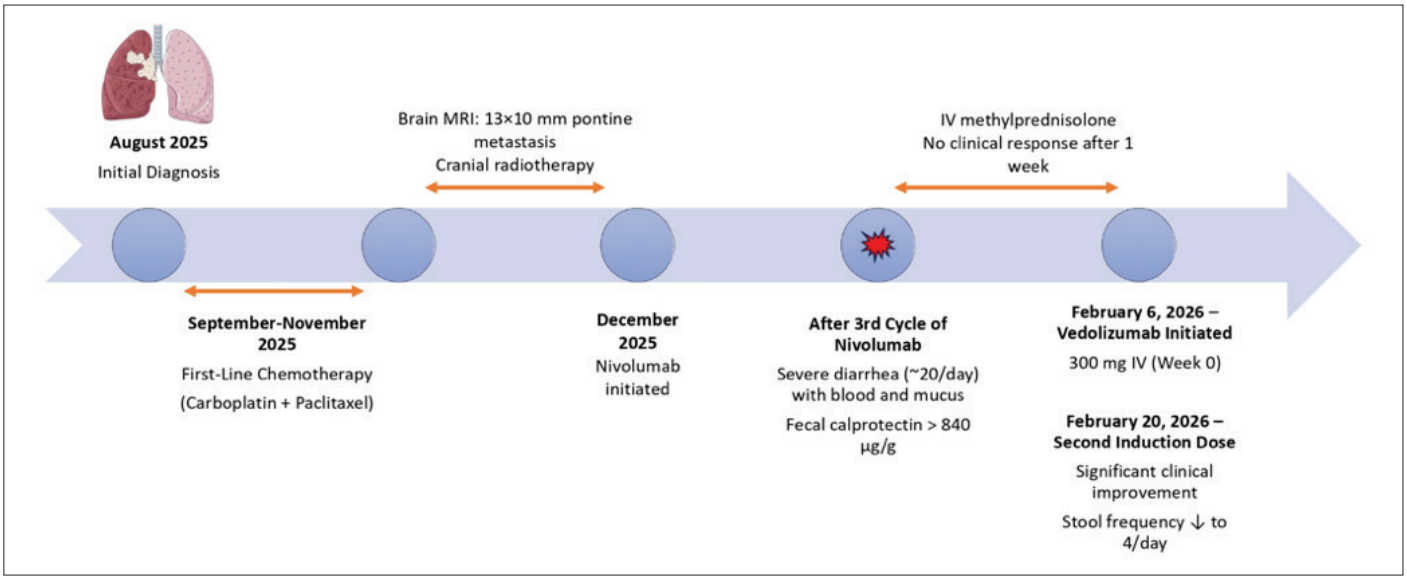

Figure 2. Clinical timeline of metastatic lung adenocarcinoma and the development of steroid-refractory nivolumab-associated colitis successfully treated with vedolizumab.

DISCUSSION

Immune checkpoint inhibitor–associated colitis is one of the most common and clinically significant immune-related adverse events in patients receiving PD-1 blockade. In the present case, severe steroid-refractory colitis developed after second-line nivolumab therapy and was successfully managed with vedolizumab.

Vedolizumab is a gut-selective monoclonal antibody directed against α4β7 integrin. It blocks lymphocyte trafficking to the gastrointestinal mucosa while minimizing systemic immunosuppression. In our case, vedolizumab was preferred for steroid-refractory immune checkpoint inhibitor–associated colitis, particularly because anti–tumor necrosis factor (anti-TNF) therapy was considered less suitable because of the patient's comorbidities.

To date, no randomized controlled trials have evaluated vedolizumab specifically for immune checkpoint inhibitor–associated colitis. Available evidence is largely derived from retrospective and multicenter observational studies, the largest of which demonstrated comparable clinical remission rates between vedolizumab and infliximab.5 In observational studies evaluating biologic therapies for immune-mediated colitis, vedolizumab has demonstrated efficacy comparable to that of infliximab. In a 2-center retrospective study of 184 patients conducted by Zou et al.,[6] clinical remission rates were similar between vedolizumab and infliximab (89% vs. 88%, p=0.79). Although the time to clinical response was longer in the vedolizumab group, patients treated with vedolizumab had shorter durations of corticosteroid exposure and reduced lengths of hospital stay. These findings suggest that vedolizumab is an effective alternative in steroid-refractory cases, particularly when minimizing systemic immunosuppression is clinically desirable. The gut-selective anti-integrin mechanism of vedolizumab provides a theoretical safety advantage over systemic tumor necrosis factor blockade. Compared with anti-TNF agents, vedolizumab has a lower impact on systemic immune function, which may translate into a reduced risk of systemic infectious complications.[7] In oncology patients, this consideration is particularly relevant because preserving systemic antitumor immunity remains a critical concern.

In addition, anti-TNF agents have been associated with adverse cardiac outcomes in patients with moderate-to-severe heart failure, with some studies reporting worsening cardiac function and increased hospitalization rates.[8] Consequently, multiple guidelines and regulatory product labels caution against the use of anti-TNF therapy in patients with advanced heart failure. In contrast, long-term safety analyses of vedolizumab in inflammatory bowel disease populations have not demonstrated an increased incidence of cardiovascular adverse events.[9,10] In our patient, who had a recent history of coronary artery disease, vedolizumab was therefore considered a more targeted and potentially safer therapeutic option than systemic tumor necrosis factor blockade. The rapid clinical improvement observed after induction therapy further supports its effectiveness in this setting.

Compared with classic inflammatory bowel disease, ICI-associated colitis may show a relatively rapid response to biologic therapies such as infliximab and vedolizumab. In many patients, clinical remission can be achieved after induction therapy alone, and prolonged maintenance biologic treatment may not always be necessary once immune checkpoint inhibitor therapy is discontinued. However, the optimal duration of biologic therapy in ICI-associated colitis remains uncertain, and long-term prospective data are still lacking.[3,4]

ICI-associated colitis is not only a significant acute toxicity but is also characterized by a substantial risk of recurrence. Reported relapse rates range up to 30% to 40%, particularly among patients with steroid-refractory disease, those requiring prolonged corticosteroid tapering, and those undergoing immunotherapy rechallenge. In a large cohort study by Abu-Sbeih et al.,[11] recurrence rates were notably higher in patients requiring biologic therapy or presenting with severe colitis. Similarly, in the multicenter retrospective analysis by Zou et al.,[6] although clinical remission rates were comparable between vedolizumab and infliximab, the importance of minimizing systemic immunosuppression for long-term disease control was emphasized. These findings highlight that therapeutic goals in ICI-associated colitis should extend beyond short-term symptomatic improvement and aim to achieve sustained control of mucosal inflammation. In steroid-refractory cases, gut-selective agents such as vedolizumab may provide a rational strategy by preserving systemic immune function while potentially reducing the risk of recurrent inflammatory activity.

In conclusion, vedolizumab is an effective salvage therapy for steroid-refractory immune checkpoint inhibitor–associated colitis, particularly in patients for whom minimizing systemic immunosuppression is desirable.

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