Case Report

Corticosteroid-dependent immune checkpoint inhibitor-induced enterocolitis treated with vedolizumab: a case report

Da-Long Wan[1]^, Chen Hu[1], Qing-Hong Ke[1], Tanyang Zhou[2], Linxiang Ruan[3], Hui Tang[4], Yan Shen[1]

[1]Department of Hepatobiliary and Pancreatic Surgery, The First Affiliated Hospital, Zhejiang University School of Medicine, Hangzhou, China; [2]Hepatobiliary and Pancreatic Interventional Treatment Center, Division of Hepatobiliary and Pancreatic Surgery, The First Affiliated Hospital, Zhejiang University School of Medicine, Hangzhou, China; [3]Department of Radiology, The First Affiliated Hospital, Zhejiang University School of Medicine, Hangzhou, China; [4]Department of Pathology, The First Affiliated Hospital, Zhejiang University School of Medicine, Hangzhou, China

Contributions: (I) Conception and design: DL Wan, C Hu, QH Ke; (II) Administrative support: Y Shen; (III) Provision of study materials or patients: DL Wan, T Zhou; (IV) Collection and assembly of data: DL Wan; (V) Data analysis and interpretation: DL Wan, L Ruan, H Tang; (VI) Manuscript writing: All authors; (VII) Final approval of manuscript: All authors.

Correspondence to: Yan Shen, PhD. Department of Hepatobiliary and Pancreatic Surgery, The First Affiliated Hospital, Zhejiang University School of Medicine, 79 Qingchun Road, Hangzhou 310003, China. Email: Shenyan1965@163.com.

Background: Immune checkpoint inhibitors (ICIs) have greatly improved the survival in several cancers. Immune-related adverse events (irAEs) are common in patients on ICI therapy, as inhibition of cytotoxic T-lymphocyte antigen 4 (CTLA-4) or programmed cell death protein 1 (PD-1) leads to non-selective activation of the immune system. ICI-induced enterocolitis is highly prevalent and corticosteroid administration is the first-line treatment. Selective immunosuppressive therapy was employed for steroid-refractory patients. The monoclonal antibody vedolizumab exhibits gut-specific immunosuppressive effects by targeting the α4β7 integrin.

Case Description: We report a case of corticosteroid-dependent camrelizumab-induced enterocolitis in a 58-year-old man with hepatocellular carcinoma (HCC) who was treated with vedolizumab. The patient's diarrhea resolved following the administration of two doses of vedolizumab (300 mg), and he was able to stop using corticosteroids. He later underwent surgery and HCC treatment, including appropriate management of ICI-induced enterocolitis, and achieved a complete pathological response.

Conclusions: This report illustrates the valuable role of vedolizumab in treating ICI-induced enterocolitis that is refractory to corticosteroid treatment.

Keywords: Immune checkpoint inhibitor-induced enterocolitis (ICI-induced enterocolitis); hepatocellular carcinoma (HCC); vedolizumab; corticosteroid therapy; case report

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Introduction

Immune checkpoint inhibitors (ICIs) have greatly improved the survival of patients with cancer. The cytotoxic T-lymphocyte antigen 4 (CTLA-4) and programmed cell death protein 1 (PD-1), which are mainly expressed on T cells, play a major role in regulating T-cell activation and preventing excessive immune response or autoimmunity (1). Antibody therapy targeting CTLA-4 (ipilimumab) or PD-1 (nivolumab and pembrolizumab) has been shown to enhance the immune response against tumors, leading to significant survival benefits in patients with advanced melanoma, non-

^ ORCID: 0000-0001-5771-1653.

small cell lung cancer, hepatocellular carcinoma (HCC), renal cancer, and squamous head and neck cancer, and other malignancies (2-6). However, the use of ICIs is accompanied with various immune-related adverse events (irAEs). The activation of ICIs not only stimulates tumor-specific T cells but also elicits responses from various other T cell clones, potentially disrupting peripheral self-tolerance and giving rise to irAEs, such as enterocolitis, hepatitis, nephritis, myocarditis, and rashes (7,8).

Inflammation of the colon, with or without inflammation of the small intestine, is the most common gastrointestinal irAE (9). The severity of gastrointestinal irAEs ranges from manageable lifestyle changes to life-threatening complications, such as perforation. The most common symptom of ICI-induced enterocolitis is diarrhea. In clinical trials and most retrospective studies, diarrhea severity is scored using the Common Terminology Criteria for Adverse Events (CTCAE) on a scale of grade 1 (mild) to grade 5 (death) (10,11). Currently, ICI-induced enterocolitis is treated differently depending on its severity. Mild (grade 1) enterocolitis can be treated symptomatically, but moderate and severe (grade 2–3) enterocolitis generally requires treatment with corticosteroids. If corticosteroids are insufficient and patients do not respond to a high dose of corticosteroids (2 mg/kg methylprednisolone) after a few days, more effective immunosuppressants such as infliximab and vedolizumab can be used (12,13). Vedolizumab, a humanized monoclonal immunoglobulin G1 (IgG1) antibody against α4β7 integrin, has been approved for the treatment of Crohn's disease and ulcerative colitis (14). Moreover, α4β7 integrin is mainly expressed in a subpopulation of $CD4^+$ T cells and primarily facilitates their migration to the gastrointestinal tract (15). Therefore, blocking α4β7 integrin leads to gut-specific immunosuppression, while preserving immune responses in extraintestinal tissues (16).

Herein, we report a case of ICI-induced enterocolitis in a patient with HCC who was treated with a PD-1 inhibitor and developed grade 3 ICI-induced enterocolitis, which did not respond adequately to corticosteroid therapy. The patient was successfully treated with vedolizumab and later achieved a pathological complete response (pCR) to therapy for HCC. We present this case in accordance with the CARE reporting checklist (available at https://jgo.amegroups.com/article/view/10.21037/jgo-24-222/rc).

Highlight box

Key findings

- Vedolizumab is an effective and safe treatment for steroid-refractory/dependent enterocolitis in a patient with hepatocellular carcinoma.

What is known and what is new?

- Vedolizumab also has enteric specificity, with a good safety profile and is unlikely to reverse the therapeutic benefits of immune checkpoint inhibitors (ICIs).
- Vedolizumab may exhibit a superior safety profile in the treatment of ICIs induced enterocolitis among tumor patients compared to infliximab.

What is the implication, and what should change now?

- Striking a balance between the duration of steroid use and the risk of tumor progression poses a significant challenge in managing ICIs-induced enterocolitis among cancer patients. Vedolizumab may exhibit a safety profile in the treatment of ICIs induced steroid-refractory/dependent enterocolitis.

Case presentation

A 58-year-old man with chronic hepatitis B virus (HBV) infection was referred to our hospital in May 2021 for further treatment because of upper abdominal pain and a large liver tumor in the right lobe of the liver. He had no family history of HCC and no history of alcohol or nicotine abuse. No abnormalities were detected on physical examination.

During hospitalization, his HBV-DNA level was 2.97×10^5 IU/mL. The serum alpha-fetoprotein (AFP) level increased to 96.1 ng/mL (normal: <20 ng/mL). Contrast-enhanced computed tomography (CT) revealed a hypodense mass in the right lobe, with a diameter of 14 cm, and invasion of the middle hepatic vein (*Figure 1A*,*1B*). The case of this patient was discussed by our multidisciplinary team, and was diagnosed with HCC (T4N0M0, stage IIIB). He underwent two transarterial chemoembolization (TACE) procedures to downstage the tumor and right portal venous embolization to enlarge the residual liver volume, while systemic therapies, including combination of targeted and immunological therapies, were also advised. During the admission, his HBV infection was treated with oral entecavir (0.5 mg) once daily.

TACE was successively performed on May 19, 2021, and he was treated with camrelizumab 200 mg once every 3 weeks, and oral lenvatinib 8 mg daily from May 25, 2021. His AFP levels returned to normal in June 2021. Then, he underwent TACE again on July 2, 2021 and right portal venous embolization 2 weeks later. CT findings showed no

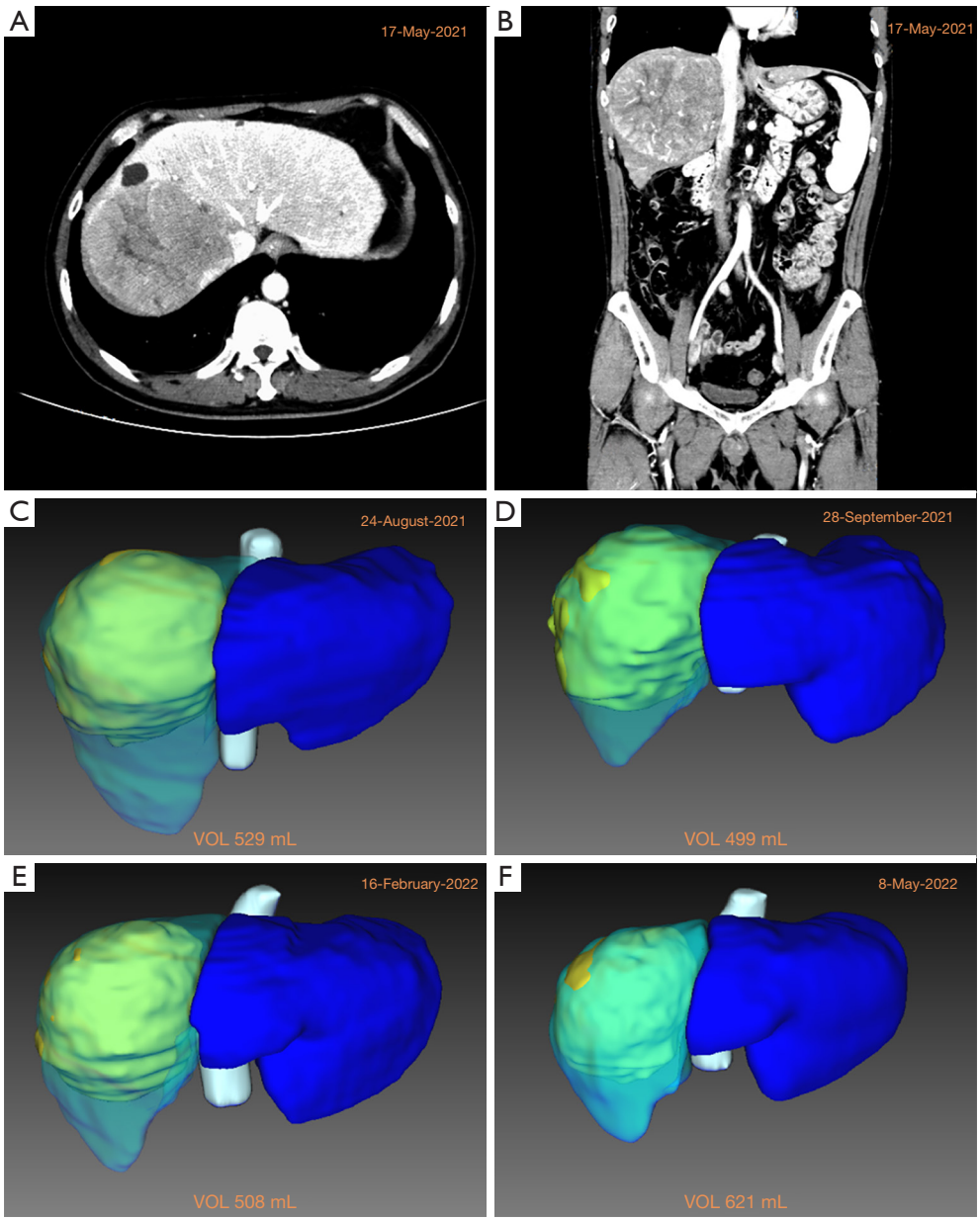

Figure 1 Changes in the patient's liver volume over the course of treatment. (A,B) Contrast-enhanced abdominal computed tomography showing a hypodense mass in the right lobe of the liver with a diameter of 14 cm, and invasion of the middle hepatic vein and inferior vena cava. (C-F) Changes in the volume of the left lobe of the liver during treatment. VOL, volume of the left liver.

significant reduction in the size of the primary lesion, and the volume of the left liver was approximately 529±37.2 mL in August 2021 (*Figure 1C*). The temporal changes in the volume of the left liver were illustrated (*Figure 1D-1F*).

At 3 months after initiating camrelizumab treatment (August 2021), the patient developed severe non-bloody diarrhea (7–8 extra bowel movements a day) (grade 3, CTCAE 5.0), accompanied by fatigue. Consequently, the immunotherapy and targeted therapy were suspended. Stool studies, including stool polymerase chain reaction for *Clostridioides difficile*, were negative. The fecal occult blood test was positive and the fecal calprotectin level was elevated to 61.4 μg/g (normal, <50 μg/g). The serum C reactive protein was 17 mg/L (normal, <8 mg/L). The

patient was treated with methylprednisolone 80 mg/day, administered by intravenous drip, for 1 week, and the dose decreased to 40 mg/day intravenous drip 1 week later. Then, he was treated with oral methylprednisolone 24 mg/day, and the diarrhea was gradually alleviated. The dose of methylprednisolone was reduced stepwise every 5 days to 16, 12, and 8 mg/day. However, at 4 days after methylprednisolone withdrawal, the diarrhea recurred (more than seven times per day). Contrast-enhanced CT showed complete necrosis of the liver tumor, moderate perihepatic effusion, and mucosal edema of the transverse colon (*Figure 2A*,*2B*). The volume of the left lobe of the liver was reduced to 499 mL (*Figure 1D*). Colonoscopy revealed congestion, edema, erosions and fused shallow ulcers with surface pus, throughout the colon, especially in the descending colon and transverse colon, (*Figure 2C*,*2D*). Histology confirmed active colitis with a mixed inflammatory infiltrate (*Figure 2E*,*2F*). Immunohistological examinations showed cytomegalovirus 2 immunonegative (*Figure 2G*,*2H*).

Considering the rapid withdrawal of the methylprednisolone and the patient's methylprednisolone dependence, methylprednisolone administration was re-started on September 1, 2021. He was treated with methylprednisolone 80 mg/day by intravenous drip for 1 week, but this did not alleviate diarrhea. Therefore, the patient was considered steroid-dependent as the enterocolitis recurred upon tapering in accordance with international recommendations. Vedolizumab was administered intravenously in two doses, with a 2-week interval at the fifth week following the onset of initial colitis. After the first vedolizumab treatment, the diarrhea was markedly alleviated. The dose of methylprednisolone was slowly reduced by 10% per week without causing any discomfort to the patient. The ICI-induced enterocolitis eventually resolved and the corticosteroids were successfully withdrawn on December 5, 2021 (*Figure 3*).

After 2 months (February 2022), contrast-enhanced CT of the abdomen showed no obvious activity of the HCC and the left hepatic volume was 508 mL (*Figure 1E*). The patient was advised to resume oral lenvatinib administration of 4 mg/day. In May 2022, contrast-enhanced CT of the abdomen revealed that the left hepatic volume had increased to 621 mL (*Figure 1F*). The patient underwent right hepatectomy and recovered well postoperatively (*Figure 4A*). The postoperative diagnosis was pCR of HCC after receiving a series of combination therapies (*Figure 4B*). At the most recent follow-up examination in December 2023, the patient's diarrhea had completely resolved with no rebound symptoms, and contrast-enhanced magnetic resonance imaging showed no evidence of HCC recurrence.

All procedures performed in this study were in accordance with the ethical standards of the institutional and/or national research committee(s) and with the Helsinki Declaration (as revised in 2013). Written informed consent was obtained from the patient for the publication of this case report and accompanying images. A copy of the written consent is available for review by the editorial office of this journal.

Discussion

We present our experience with vedolizumab as an efficacious and well-tolerated therapy for the management of ICI-induced enterocolitis in a patient who was dependent and refractory to steroids. This result aligns with a recent report where vedolizumab demonstrated successful treatment outcomes in a single patient with ipilimumab-induced colitis (17).

ICIs increase the activation of $CD4^+$ and $CD8^+$ T cells, which can lead to autoimmune enterocolitis. Camrelizumab, a humanized IgG4 monoclonal antibody with high affinity for PD-1, has demonstrated both efficacy and safety in the treatment of advanced HCC. Consequently, it has received approval as a monotherapy option in the second-line setting within China (18). The incidence of immune-related colitis following camrelizumab treatment in advanced HCC was observed to be 0.91% (19). However, when combined with rivoceranib, grade 1–2 diarrhea occurred in 28% of cases, while grade 3 diarrhea occurred in only 2% of cases (20). Intravenous corticosteroids are recommended as the initial standard treatment for ICI-induced enterocolitis. Historically, failure to achieve clinical remission with intravenous corticosteroids has consistently resulted in colectomy. The emergence of effective immunosuppressants such as infliximab and vedolizumab has provided an alternative therapeutic option for cases refractory to steroids (21). However, their utilization also carries an increased risk of opportunistic infections and secondary malignancies (22). Vedolizumab is a humanized monoclonal IgG1 antibody against α4β7 integrin on the surface of $CD4^+$ T cells. The α4β7 integrin binds to its ligand, mucosal vascular addressin cell adhesion molecule (MAdCAM)-1, which is expressed on the endothelial surface of intestinal venules and related lymphatic tissues, and promotes T-cell transport to intestinal mucosa. By blocking the interaction between α4β7 integrin and its ligand MAdCAM-1, vedolizumab prevents

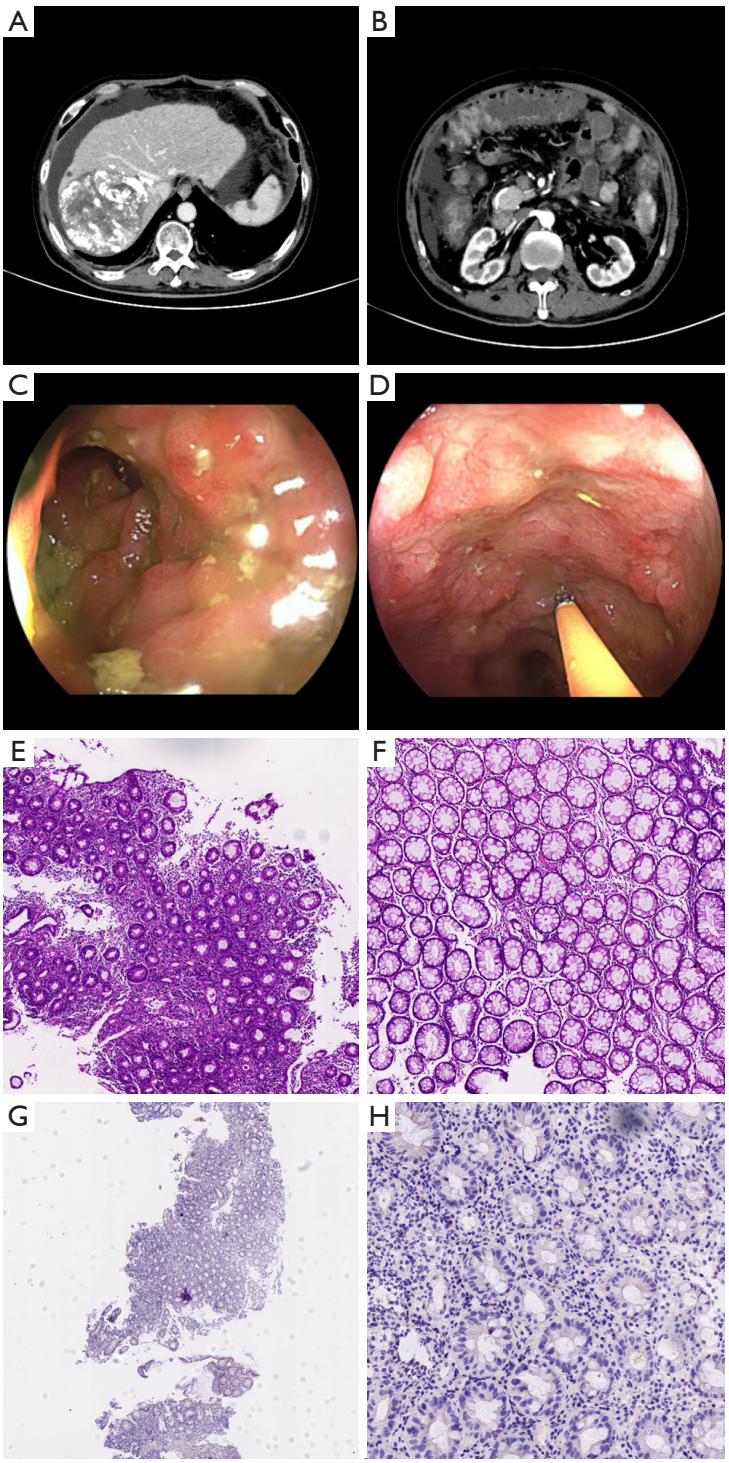

Figure 2 Imaging and pathological features of immune checkpoint inhibitor-induced enterocolitis. (A,B) Contrast-enhanced abdominal computed tomography showing complete necrosis of the hepatocellular carcinoma, moderate perihepatic effusion and mucosal edema of the transverse colon. (C,D) Colonoscopy revealed that the total colon mucosa, especially the descending colon and transverse colon, was congested, edematous, erosive, and covered by fused shallow ulcers, which had partially swollen surface pus. (E,F) Histology confirmed active colitis with a mixed inflammatory infiltrate in the descending colon and sigmoid colon (hematoxylin-eosin staining, ×100, ×200). (G,H) Immunohistological examinations showed cytomegalovirus 2 immunonegative (immunohistochemistry, ×40, ×400).

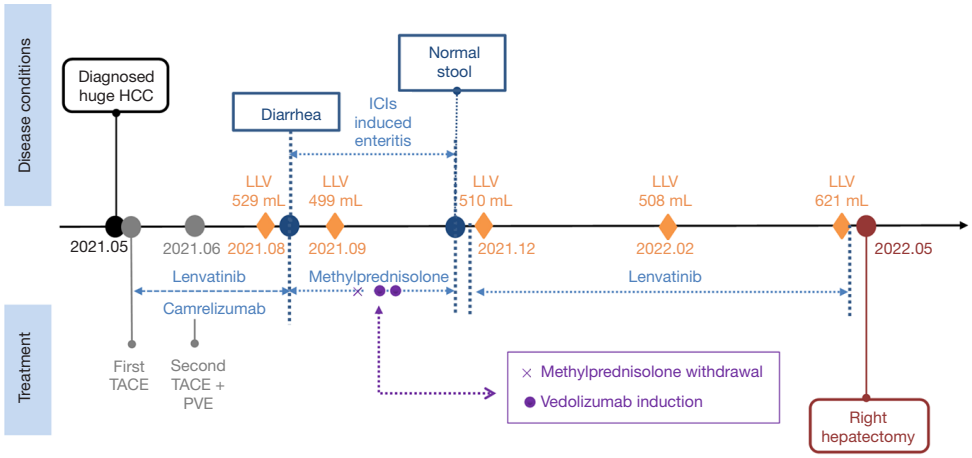

Figure 3 Timeline of the patient's clinical progress showing major treatment modalities and disease status. HCC, hepatocellular carcinoma; TACE, transarterial chemoembolization; PVE, portal vein embolism; LLV, left liver volume; ICI, immune checkpoint inhibitor.

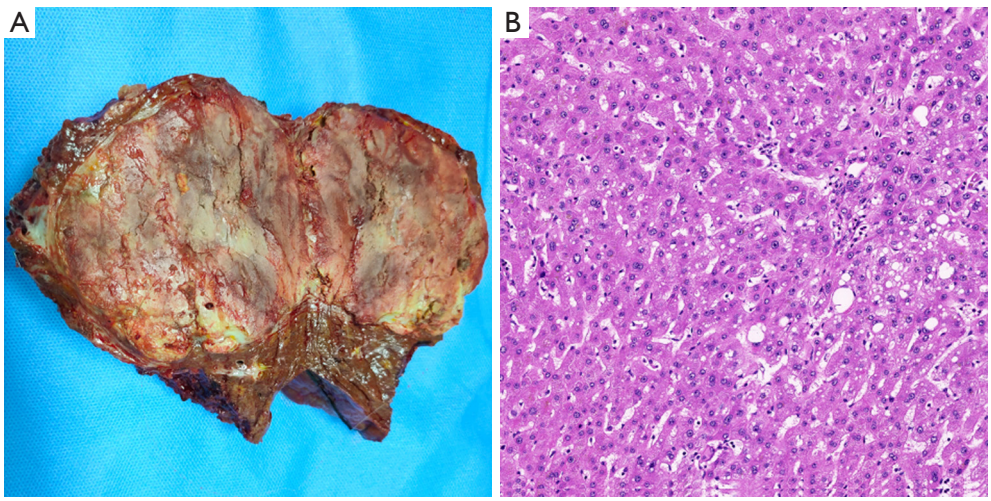

Figure 4 Postoperative gross pathology of the hepatocellular carcinoma. (A) Surgical specimens and (B) pathology showed complete necrosis of the hepatocellular carcinoma (hematoxylin-eosin staining, ×40).

T-cell binding and migration to the inflammatory intestinal mucosa, which may explain its effectiveness in treating ICI-induced enterocolitis (23). Vedolizumab also has enteric specificity, with a good safety profile and is unlikely to reverse the therapeutic benefits of ICIs (16). In fact, some oncology guidelines recommended vedolizumab administration only for patients with contraindications or non-response to infliximab (24). Zou *et al.* (25) considered two reasons. The first was that vedolizumab was not approved for clinical use in inflammatory bowel disease cases until 2014 as there was no sufficient evidence. Second, infliximab biosimilars with favorable costs had comparable efficacy with the originator. However, the majority of studies on infliximab have provided data on the safety of long-term TNF-α blockade in a non-cancer population (26). Given its enteric specificity, vedolizumab treatment has a more favorable safety profile, with lower incidence of serious infections and malignancies than infliximab (14). Kaneoka *et al.* (27) summarized some case reports and found that two or three doses of vedolizumab improved colitis without any side effects, regardless of staging and cycles of ICIs in patients with cancer. They further confirmed the efficiency of vedolizumab in treating severe colitis that failed to resolve with corticosteroid and infliximab. Therefore,

vedolizumab should be considered as a potential first-line agent for the treatment of ICI-induced enterocolitis in steroid-dependent patients. In 2020, a meta-analysis found only two articles and one abstract reporting on the role of vedolizumab in treating ICI-induced enterocolitis (28). In 2021, eight studies in a systematic review were documented for the use of vedolizumab for prevention and treatment of ICI-induced enterocolitis (29). Nielsen *et al.* (30) identified four studies with vedolizumab to assess the efficacy and safety of biologics in the latest meta-analysis after a series of strict inclusion and exclusion criteria. Bergqvist *et al.* (31) reported the cases of seven patients with endoscopically confirmed corticosteroid refractory ICI-induced enterocolitis who were treated with 2–4 doses of vedolizumab (300 mg). Prednisolone was successfully reduced in six out of seven patients. In a larger study by Abu-Sbeih *et al.* (32), 34 patients with corticosteroid refractory enterocolitis (including two patients with prior exposure to infliximab) received a median of 3 doses (range, 1–6 doses) of vedolizumab, and 32 of the 34 patients (94%) responded to vedolizumab therapy. A comparative retrospective study of infliximab versus vedolizumab that included 184 patients revealed that the vedolizumab group had a shorter steroid exposure, fewer hospitalizations, and a shorter hospital stay, but demonstrated a longer time to clinical response than the infliximab group (25). In conclusion, vedolizumab was chosen as a more favorable alternative to conventional infliximab in our case due to its gut-selective mechanism of action, which minimized the potential risk of HCC progression. The use of vedolizumab in this context was deemed less likely to attenuate the antitumor effect of camrelizumab and offered the additional advantage of not increasing susceptibility to opportunistic infections or secondary malignancies, as observed with infliximab administration (14,25). Interestingly, d'Apolito *et al.* (33) reported a case of corticosteroid-refractory immune-mediated colitis treated with vedolizumab and stated that HLA B-35 allele could be a potential biomarker for predicting the genetic basis of susceptibility of patients with high-risk autoimmune disease treated with immunotherapy. To our knowledge, our case is the first report of vedolizumab being used successfully to treat of corticosteroid-dependent ICI-induced enterocolitis in a patient with HCC. The successful management of ICI-induced enterocolitis enabled this patient to undergo surgical treatment and to achieve a pCR.

In addition to enabling corticosteroid tapering, vedolizumab treatment led to a notable reduction in the levels of C-reactive protein, reflecting the attenuated inflammation in the gut. Adverse events related to vedolizumab are rare, owing to its highly gut-selective mode of action. However, following initiation of vedolizumab among patients with ulcerative colitis, a tolerable side effect profile with upper respiratory tract infection, mild nasopharyngitis, headache, arthralgia, nausea, and fatigue has been reported (14,34). In our case, the patient did not experience any vedolizumab-related adverse events. Among the various immunosuppressants, corticosteroids have the highest risk of enabling serious infections, and the risk increases when corticosteroids are combined with systemic immunosuppressants. A retrospective study of 740 patients with melanoma who were treated with ICIs found that 7.3% of them experienced serious infections, and corticosteroids and/or infliximab were considered to be the main risk factors (35). Therefore, vedolizumab is a safe alternative for treating of ICI-induced enterocolitis.

Our case management has some limitations. Generally, the decision to initiate secondary immunosuppression was made for two reasons: an inadequate initial response to corticosteroid therapy or recurrence of enterocolitis symptoms during corticosteroid tapering. The rapid withdrawal of corticosteroids in this patient led to dependence to corticosteroids and recurrence of enterocolitis. Corticosteroids should be gradually reduced by 10% per week (2). A comprehensive approach involving an oncologist, a gastroenterologist, and a pathologist with expertise in the disease process and the treatment of ICI-induced enterocolitis is imperative for optimal management of these patients.

Conclusions

In conclusion, our patient successfully underwent right hemihepatectomy and had satisfactory outcomes. Undoubtedly, this was a result that was well-worth waiting for our patient. Our case report can provide insights for clinicians when they encounter a similar situation. To our knowledge, this is the first case report to suggest that vedolizumab is an effective and safe treatment for steroid-refractory/dependent enterocolitis in a patient with HCC. The appropriate management of the ICI-induced enterocolitis enabled patient to undergo surgical treatment and to achieve a pCR. With the increasing use of ICI-therapy, the prevalence of ICI-induced enterocolitis is likely to increase considerably in the near future. Therefore, there is a growing need to develop evidence-based interventions, taking the long-

term cancer-treatment strategy into account, to prolong the survival of patients with cancer.

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Footnote

Reporting Checklist: The authors have completed the CARE reporting checklist. Available at https://jgo.amegroups.com/article/view/10.21037/jgo-24-222/rc

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Conflicts of Interest: All authors have completed the ICMJE uniform disclosure form (available at https://jgo.amegroups.com/article/view/10.21037/jgo-24-222/coif). The authors have no conflicts of interest to declare.

Ethical Statement: The authors are accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved. All procedures performed in this study were in accordance with the ethical standards of the institutional and/or national research committee(s) and with the Helsinki Declaration (as revised in 2013). Written informed consent was obtained from the patient for the publication of this case report and accompanying images. A copy of the written consent is available for review by the editorial office of this journal.

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