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Crohn`s disease, vedolizumab and autoinmmune hemolytic anemia: coincidence or causality?

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Abstract:

Autoimmune hemolytic anemia is an autoimmune destruction of red blood cells, usually mediated by immunoglobulin G and/or immunoglobulin M. We present the case of a 75-year-old woman diagnosed with Crohn's disease who developed autoimmune hemolytic anemia while receiving treatment with vedolizumab. Was the anemia an immune-mediated manifestation of her Crohn's disease or an unusual adverse effect of the treatment? In our case, the patient was in remission and the anemia disappeared after discontinuation of vedolizumab, which reinforces the suspicion that vedolizumab has contributed to the onset of autoimmune hemolytic anemia. This association has been rarely described in the literature, we think that it is important to report this case to raise awareness and contribute to a better understanding of this potential association.

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Dear Editor,

Autoimmune hemolytic anemia (AIHA) is an autoimmune destruction of red blood cells, usually mediated by immunoglobulin G (IgG) and/or immunoglobulin M (IgM).

We present the case of a 75-year-old woman diagnosed with Crohn's disease (CD) who developed AIHA while receiving treatment with vedolizumab.

The patient had a history of follicular lymphoma, in complete remission since 2013. She was diagnosed with CD with extensive ileal involvement in 2020, and vedolizumab was selected due to the effectives and its favourable safety profile in patients with oncological backgrounds (1). She achieved an adequate clinical and radiological response during maintenance with vedolizumab 300 mg each 4 weeks.

In June 2024, she was admitted with acute gastroenteritis. A normocytic normochromic anemia (hemoglobin 8.7 g/dL) was detected, and a single intravenous dose of ferric carboxymaltose (1000 mg) was administered nevertheless normal ferritin levels. However, follow-up revealed recurrent anemia with significantly elevated ferritin levels (>1000 mg/dL). Other laboratory test suggested hemolysis (increased of reticulocytes (8.9%), total bilirubin 2.0 mg/dl, LDH 251 U/L and haptoglobin <8 mg/dL) with Coombs direct test (IgG 3+, C3d 3+), so she was diagnosed with warm AIHA. Fecal calprotectin levels were normal (45 mg/kg). Additionally, follicular lymphoma recurrence and IBD activity were ruled out through thoracoabdominopelvic CT imaging and upper and lower endoscopic evaluation. To our knowledge, only three cases of AIHA had been potentially linked to vedolizumab (2-4), but we decided to discontinue the drug in February 2025. In May 2025 she has recovered completely of anemia and started treatment with ustekinumab.

Discussion

Extraintestinal manifestations are common in patients with inflammatory bowel disease (IBD), but the association between IBD and AIHA has been scarcely reported, with most cases occurring during active disease (5).

In our case, the patient developed AIHA after three years of vedolizumab treatment while she was in remission. Was the anemia an immune-mediated manifestation of

her CD or an unusual adverse effect of the treatment? The differential diagnosis between immunomodulated manifestations and vedolizumab-induced hemolytic anemia is complex and almost impossible to establish. However, in our case, the resolution of the anemia after discontinuation of vedolizumab would support the involvement of vedolizumab in the etiology of AIHA.

We think that this report is important to add evidence of this possible association and most of all, underscores the importance of maintaining a high index of suspicion in patients with severe anemia and IBD undergoing treatment with novel therapies.

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