Managing immune checkpoint inhibitor-associated gastritis: Insights and strategies

Yu LL et al. Deepen our understanding of irAE gastritis

Abstract

Immune checkpoint inhibitors (ICIs) are widely used due to their effectiveness in treating various tumors. Immune-related adverse events (irAEs) are defined as adverse effects resulting from ICI treatment. Gastrointestinal irAEs are a common type of irAEs characterized by intestinal side effects, such as diarrhea and colitis, which may lead to the discontinuation of ICIs.

Key Words: Immunotherapy; Immune checkpoint inhibitor; Immune checkpoint inhibitor-related gastritis; Immune-related adverse events; Autoimmune responses

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Core Tip: Immune checkpoint inhibitor (ICI)-related gastritis is rare but may lead to serious complications such as gastorrhagia. The strategies such as early identification, pathological diagnosis, management interventions, and immunotherapy reactivation are discussed to enable clinicians to better manage ICI-related gastritis and improve the prognosis of these patients.

TO THE EDITOR

I am writing to express my concern regarding the paper titled “Immune checkpoint inhibitor-associated gastritis: Patterns and management” by Lin et al(1), recently published in the World Journal of Gastroenterology. The authors systematically summarize the occurrence patterns and management strategies of immune checkpoint inhibitor
ICI-associated gastritis, providing important evidence for research and practice in this field.

Over the last decade, the emergence of ICI therapy has revolutionized the treatment of a growing number of malignancies. Immune-related adverse events (irAE) are side effects that resemble autoimmune responses in the patients receiving ICIs. ICI-related gastritis, although rare, may lead to serious complications, such as gastrorrhagia. The most common abnormality reported on endoscopy is erythema, followed by erosions. Other findings include granularity, sloughing, exudates, ulcers, atrophy, and rarely, severe hemorrhagic gastritis.

A common mechanism (Figure 1) by which ICIs exert their effects involves activation of effector T cells by inhibition of programmed death 1, programmed death-ligand 1, and cytotoxic T-lymphocyte antigen. It is also proposed that the proliferation of activated T cells and increased cytokine production, caused by a lack of self-tolerance, may result in irAEs. However, the detailed mechanisms underlying irAEs remain unclear. Therefore, the treatment decisions for ICI-related gastritis are based on individual clinical presentations.

However, the article does not adequately address individualized treatment options for diverse patient populations. Considering the patient's immune status, gastritis severity, and other factors, the formulation of an individualized treatment plan is particularly important. For example, before starting an ICI, autoantibody screening may be considered for patients with a personal or familial history of autoimmune disease or those presenting with signs or symptoms suggestive of an underlying autoimmune disease. This precaution is due to their enhanced risk of developing a full-blown autoimmune disease post-treatment. Second, the article neglects to mention the evaluation and monitoring strategies prior to ICI therapy when discussing preventive measures, which are important for reducing the risk of gastritis.

Additionally, the issue of re-provocation after ICI treatment warrants attention, particularly the risk of recurrence of gastritis. Upon complete resolution of irAEs, the
resumption of immunotherapy is crucial for treatment and prognosis despite the risk of irAE relapse.

In the future, we need to continue to deepen our understanding of irAE gastritis, enabling timely and appropriate diagnosis and treatment and providing clinicians with guidance for the treatment of ICI-related gastritis to improve patient prognosis.

3 ACKNOWLEDGEMENTS

We acknowledge all the authors whose publications are used as references in our article.

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Figure 1 A common mechanism by which immune checkpoint inhibitors exert their effects involves activation of effector T cells by inhibition of programmed death 1, programmed death-ligand 1, and cytotoxic T-lymphocyte antigen 4. ICI: Immune checkpoint inhibitor; PD-1: Programmed death 1; PD-L1: Programmed death-ligand 1; CTLA-4: Cytotoxic T-lymphocyte antigen 4; irAEs: Immune-related adverse events.
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