

Immune-related adverse events in cancer therapy: Management and best practices.

Henri Gord*

Department of Oncology, University of Alberta, Canada

Introduction

The advent of immunotherapy has transformed the landscape of cancer treatment, offering new hope to patients with previously untreatable malignancies. However, the activation of the immune system, which is central to the efficacy of immunotherapies such as immune checkpoint inhibitors, can also lead to immune-related adverse events (irAEs). These toxicities, resulting from overactivation of the immune response, pose significant challenges for clinicians and patients alike [1].

irAEs are inflammatory side effects that can affect any organ system, reflecting the broad impact of immune activation. Commonly affected organs include the skin, gastrointestinal tract, liver, and endocrine glands. For instance Rash and pruritus are among the most frequently reported irAEs [2].

Colitis, characterized by diarrhea and abdominal pain, is a serious complication. Immune-mediated hepatitis can manifest as elevated liver enzymes and jaundice. Hypothyroidism, hyperthyroidism, and adrenal insufficiency are common endocrine-related irAEs. In rare cases, severe or life-threatening irAEs, such as myocarditis or neurological toxicities, may occur [3].

Effective management of irAEs requires early recognition and prompt intervention to minimize morbidity. Key principles include Patients undergoing immunotherapy should be educated about potential irAEs and encouraged to report symptoms promptly. Regular monitoring through clinical and laboratory assessments is essential [4].

The Common Terminology Criteria for Adverse Events (CTCAE) is used to grade irAEs based on their severity, guiding treatment decisions. Often managed with symptomatic treatment and close monitoring. Require corticosteroids, such as prednisone or methylprednisolone, to suppress the immune response. For steroid-refractory cases, additional immunosuppressive agents like infliximab or mycophenolate mofetil may be needed [5].

Depending on the severity and persistence of irAEs, immunotherapy may need to be paused or permanently discontinued. Collaboration among oncologists, immunologists, and organ-specific specialists ensures comprehensive management of irAEs [6].

Identifying patients at higher risk for irAEs, such as those with preexisting autoimmune conditions, can guide treatment

planning. While not universally recommended, certain patients may benefit from preemptive measures, such as prophylactic corticosteroids, to reduce the risk of severe irAEs [7].

Adherence to established guidelines, such as those from the American Society of Clinical Oncology (ASCO) or the National Comprehensive Cancer Network (NCCN), ensures standardized and evidence-based care. Ongoing research aims to better understand the mechanisms underlying irAEs, paving the way for predictive biomarkers that can identify patients at risk [8].

Additionally, the development of targeted immunosuppressive therapies holds promise for managing irAEs more effectively without compromising anti-tumor immunity [9].

Artificial intelligence (AI) and machine learning are being explored to enhance irAE monitoring and prediction, leveraging data from electronic health records and real-world evidence to improve patient outcomes [10].

Conclusion

Immune-related adverse events represent a double-edged sword in cancer immunotherapy, reflecting both its potency and complexity. By implementing best practices in patient management, fostering interdisciplinary collaboration, and advancing research, the oncology community can optimize the balance between efficacy and safety. As immunotherapy continues to evolve, so too must our strategies for managing its challenges, ensuring that patients derive maximum benefit with minimal harm.

References

1. Navarro SL, Chang JL, Peterson S, et al. Modulation of human serum glutathione S-transferase A1/2 concentration by cruciferous vegetables in a controlled feeding study is influenced by GSTM1 and GSTT1 genotypes. *Cancer Epidemiol Biomarkers Prev.*2009;18:2974-8.
2. Wang LI, Giovannucci EL, Hunter D, et al. Dietary intake of cruciferous vegetables, glutathione S-transferase (GST) polymorphisms and lung cancer risk in a Caucasian population. *Cancer Causes Control.*2004;15:977-85.
3. Chen KL, Jung P, Kulkoyluoglu-Cotul E, et al. Impact of diet and nutrition on cancer hallmarks. *J Cancer Prev Curr Res.*2017;7:240.

*Correspondence to: Henri Gord, Department of Oncology, University of Alberta, Canada. E-mail: henrigord@hotmail.com

Received: 02-Dec-2024, Manuscript No. AAJCIT-24-155297; Editor assigned: 03-Dec-2024, Pre QC No. AAJCIT-24-155297(PQ); Reviewed: 17-Dec-2024, QC No. AAJCIT-24-155297; Revised: 23-Dec-2024, Manuscript No. AAJCIT-24-155297(R); Published: 30-Dec-2024, DOI:10.35841/ajcit-7.6.242

4. Newmark HL, Yang K, Kurihara N, et al. Western-style diet-induced colonic tumors and their modulation by calcium and vitamin D in C57Bl/6 mice: a preclinical model for human sporadic colon cancer. *Carcinogenesis*.2009;30:88–92.
5. Ulitsky A, Ananthakrishnan AN, Naik A, et al. Vitamin D deficiency in patients with inflammatory bowel disease association with disease activity and quality of life.*J Parenter Enter Nutr*.2011;35(3):308–316.
6. Cooper MD, Raymond DA, Peterson RD et al. The functions of the thymus system and the bursa system in the chicken.*J Exp Med*.1966;123:75–102.
7. Miller JF, Mitchell GF. Cell to cell interaction in the immune response, I: hemolysin-forming cells in neonatally thymectomized mice reconstituted with thymus or thoracic duct lymphocytes.*J Exp Med*.1968;128:801–820.
8. Coombs RR, Feinstein A, Wilson AB. Immunoglobulin determinants on the surface of human lymphocytes. *Lancet*.1969;2:1157–1160.
9. Swensen SJ, Jett JR, Hartman TE, et al. CT screening for lung cancer: five-year prospective experience. *Radiol*. 2005;235:259–65.
10. Curtis RE, Rowlings PA, Deeg HJ, et al. Solid cancers after bone marrow transplantation. *N Engl J Med*. 1997;336:897–904.