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Shining Damaged Hearts: Immunotherapy-related Cardiotoxicity in the Spotlight of Nuclear Cardiology

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Introduction

Immunotherapy has revolutionized cancer treatment, offering promising outcomes for patients with various malignancies. However, alongside its benefits, immunotherapy can lead to immune-related adverse events, including cardiotoxicity. This article delves into the mechanisms, clinical manifestations, and diagnostic approaches to Immunotherapy-Related Cardiotoxicity (IRC), emphasizing the pivotal role of nuclear cardiology in its detection and management. By shedding light on IRC, we aim to enhance awareness among healthcare providers, ultimately improving patient outcomes in the era of cancer immunotherapy [1].

Immunotherapy has emerged as a cornerstone in the management of cancer, harnessing the immune system's power to target malignant cells selectively. However, this approach is not without its drawbacks. Immunotherapy-related adverse events, including cardiotoxicity, have garnered attention due to their potential severity and impact on patient outcomes. Cardiotoxicity associated with immunotherapy poses unique challenges in diagnosis and management, necessitating a multidisciplinary approach. Nuclear cardiology, with its advanced imaging modalities, plays a crucial role in identifying and monitoring immunotherapy-related cardiotoxicity. This article explores the mechanisms underlying IRC, its clinical manifestations, diagnostic strategies, and the role of nuclear cardiology in its detection and management [2].

Description

Immunotherapy encompasses various modalities, including Immune Checkpoint Inhibitors (ICIs), monoclonal antibodies, and adoptive cell therapies. While these treatments offer remarkable efficacy against cancer, they can also disrupt immune homeostasis, leading to autoimmune reactions against normal tissues, including the heart. The precise mechanisms of IRC are complex and multifactorial, involving immune dysregulation, inflammation, and direct myocardial damage. ICIs, such as anti-programmed cell death protein 1 and anti-Cytotoxic T-lymphocyte-associated protein 4 antibodies, are particularly implicated in IRC. These agents enhance T-cell activation and proliferation, inadvertently targeting antigens expressed by cardiac myocytes or vascular endothelial cells, resulting in myocarditis, vasculitis, or pericarditis. Furthermore, cytokine release syndrome, a systemic inflammatory response triggered by immunotherapy, can exacerbate myocardial injury, leading to heart failure, arrhythmias, or cardiogenic shock [3].

IRC can manifest across a spectrum of clinical presentations, ranging from asymptomatic cardiac biomarker elevation to life-threatening cardiac events. Common symptoms include dyspnoea, chest pain, palpitations, and fatigue. However, IRC may be insidious, with some patients presenting with nonspecific symptoms or even asymptomatic myocardial injury detected incidentally on routine surveillance. Given the diversity of clinical presentations and the potential for severe complications, early recognition and prompt intervention are paramount in managing IRC [4].

The diagnosis of IRC relies on a combination of clinical evaluation, cardiac biomarkers, Electrocardiography (ECG), echocardiography, and advanced imaging techniques. Cardiac biomarkers, such as troponin and Brain Natriuretic Peptide (BNP), serve as sensitive indicators of myocardial injury and cardiac dysfunction. ECG findings may range from nonspecific ST-T wave changes to conduction abnormalities or life-threatening arrhythmias. Transthoracic echocardiography provides valuable information regarding ventricular function, wall motion abnormalities, and pericardial effusion. However, given the limitations of these modalities in detecting subclinical myocardial injury, nuclear cardiology emerges as a cornerstone in the evaluation of IRC.

Nuclear cardiology techniques, including Myocardial Perfusion Imaging (MPI) and Positron Emission Tomography (PET), offer unparalleled sensitivity in detecting myocardial inflammation, ischemia, and fibrosis. MPI utilizing Single-photon Emission Computed Tomography (SPECT) or PET radiotracers, such as technetium-99m or rubidium-82, enables the assessment of myocardial blood flow and viability. In the context of IRC, MPI can detect perfusion defects indicative of myocarditis or microvascular dysfunction, guiding therapeutic decisions and risk stratification. Additionally, PET imaging with radiotracers targeting inflammation provides valuable insights into the extent and distribution of myocardial inflammation, facilitating early diagnosis and monitoring of therapeutic response. Furthermore, advanced techniques, such as cardiac magnetic resonance imaging with late gadolinium enhancement, offer complementary information regarding myocardial edema, fibrosis, and tissue characterization.

The management of IRC necessitates a multidisciplinary approach involving cardiologists, oncologists, and other specialists. Treatment strategies may include immune suppression with corticosteroids, immunomodulatory agents, or biologic therapies. However, the optimal management approach remains elusive, given the limited data and heterogeneity of IRC presentations. Close monitoring with serial cardiac biomarkers and imaging studies is crucial to guide therapy and assess treatment response. In severe cases, temporary or permanent discontinuation of immunotherapy may be warranted, balancing the risks of cancer progression against cardiac complications [5].

Conclusion

Immunotherapy-related cardiotoxicity poses significant challenges in the management of cancer patients, necessitating heightened awareness, early recognition, and multidisciplinary collaboration. Nuclear cardiology emerges as a cornerstone in the detection and monitoring of IRC, offering advanced imaging modalities for assessing myocardial inflammation, ischemia, and fibrosis. By leveraging these techniques, clinicians can optimize patient care, mitigate cardiac risks, and improve outcomes in the era of cancer immunotherapy.

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Conflict of Interest

None.

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