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PERSPECTIVE ARTICLE OPEN ACCESS

Anti-cancer immunotherapy-related cardiotoxicity: preventable and treatable

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Abstract – Anti-cancer immunotherapies, particularly immune checkpoint inhibitors, have significantly advanced oncology treatments but are associated with rare, potentially severe cardiotoxicities. Despite their success, these therapies are associated with immune-related adverse events including rare but severe immunotherapy-related cardiotoxicities. This review examines various immunotherapy types, such as immune checkpoint inhibitors, adoptive T-cell therapies, and cancer vaccines, highlighting their cardiovascular risks. Cardiotoxicities can lead to life-threatening complications, especially in high-risk patients. Early detection and prevention are crucial, with comprehensive cardiovascular assessments and routine biomarker monitoring playing a central role. With immunotherapies becoming more prevalent, this review calls for stronger evidence-based guidelines to manage and prevent these cardiovascular complications, ensuring that patients can benefit from life-saving immunotherapies without jeopardizing cardiovascular health.

Key words: Immunotherapy, Cardiotoxicity, Immune checkpoint inhibitors, Cancer immunotherapy, Melanoma.

Introduction

Anti-cancer immunotherapy has revolutionized oncology by leveraging the immune system to target cancer cells, offering hope for controlling advanced diseases. Immune checkpoint inhibitors (ICIs) and other immunotherapeutic agents are increasingly common, but can cause immune-related adverse effects, including toxicities in the gastrointestinal, endocrine, hepatic, and dermatologic systems [1]. Among the most concerning, is immunotherapy-related cardiotoxicity, a rare but potentially life-threatening complication, encompassing myocarditis, pericardial disease, arrhythmias, cardiomyopathy, congestive heart failure, and others [1–3]. Current treatments for immune-related adverse events (irAEs) are outlined in the Common Terminology Criteria for Adverse Events and American Society of Clinical Oncology guidelines. Steroids remain the primary treatment for cardiotoxicity or irAEs of any other organ system [1–4]. Fortunately, immunotherapy-related cardiotoxicity can be preventable and treatable with early detection and prevention. This article reviews types of immune therapies, signs of cardiovascular toxicities, and interventions to mitigate these risks, ensuring patients can benefit from these advances without compromising cardiovascular health.

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Categories of immunotherapy in oncology

Table 1 summarizes types of immune therapies, their mechanisms, and approved indications. Cardiotoxicity from ICIs is rare (approximately 1%), but severe events such as refractory arrhythmia or cardiogenic shock can result in devastating clinical consequences with high mortality rates [2, 5]. The true incidence has not been fully characterized and is likely underreported, varying by rarity, immunotherapy type, cancer type, and patient factors. A contemporary comparative meta-analysis study showed treatment with ICIs was associated with a higher risk of myocarditis, pericardial disease, and MI compared with chemotherapy or placebo, though this was not reflected in observational studies [6]. Combination ICI therapy carries a higher risk and severity of cardiotoxicity than monotherapy [5, 7]. Cardiotoxicity in adoptive T-cell therapies is commonly related to cytokine release syndrome (CRS) when activation of the immune system results in a surge of cytokines and chemokines and subsequent T-cell therapy mediated tumor-cell lysis [8, 9]. Cohort studies on CAR T-cell therapy revealed 10% – 20% of patients experienced major adverse cardiovascular events, such as arrhythmias, cardiomyopathy, myocardial infarction, and heart failure [8].

T-VEC, the oncolytic virus therapy for melanoma, has a low toxicity profile consisting of flu-like symptoms, such as

Table 1. Summary of categories of immunotherapy, including subtypes, agents, mechanism of action, and approved indications for advanced solid tumors.

Type	Subtype	Agents	Mechanism	Approved indications
Immune Checkpoint Inhibitors (ICI)	PD-1 inhibitors	Pembrolizumab (Keytruda)	Binds to PD-1 to block activation by ligands PD-L1 and PD-L2	CSCC, MCC, melanoma, head/neck cancer, NSCLC, mesothelioma, esophageal cancer, gastric cancer, colorectal cancer, liver cancer, biliary tract cancer, renal cancer, urothelial carcinoma, cervical cancer, endometrial cancer, TNBC, lymphoma
		Nivolumab (Opdivo)	Binds to PD-1 to block activation by PD-L1 and PD-L2	Melanoma, head/neck cancer, NSCLC, mesothelioma, esophageal cancer, gastric cancer, colorectal cancer, liver cancer, renal cancer, urothelial carcinoma, lymphoma
		Cemiplimab (Libtayo)	Binds to PD-1 to block ligand PD-L1	CSCC, BCC, NSCLC
	PD-L1 inhibitors	Atezolizumab (Tecentriq)	Binds to PD-L1 to inhibit activation with receptor PD-1	Melanoma, alveolar soft part sarcoma, NSCLC, liver cancer, urothelial carcinoma, TNBC
		Avelumab (Bavencio)	Binds to PD-L1 to inhibit activation with receptor PD-1	MCC, renal cancer, urothelial carcinoma
		Durvalumab (Imfinzi)	Binds to PD-L1 to inhibit activation with receptor PD-1	MCC, NSCLC, liver cancer, biliary tract cancer, endometrial cancer
		Retifanlimab (Zynyz)	Binds to PD-1 to block activation by ligands PD-L1 and PD-L2	MCC
	CTLA-4 inhibitor	Ipilimumab (Yervoy)	Binds to CTLA4 to inhibit downregulation of T-cell activation	Melanoma, NSCLC, mesothelioma, esophageal cancer, liver cancer, renal cancer, colorectal cancer
	LAG-3 inhibitor	Relatlimab/Nivolumab (Opdualag)	Nivo (see above). Relatlimab binds to LAG-3 and blocks its interaction with MHC class II on TILs	Melanoma
	Tumor-infiltrating lymphocyte (TIL) therapy	Lifileucel (Amtagvi)	TILs are isolated, expanded in vitro with lL-2, and selected to recognize TAAs to initiate tumor lysis	Melanoma

(Continued on next page)

Table 1. (Continued)

Type	Subtype	Agents	Mechanism	Approved indications
Adoptive Cell Therapies (ACT)	Engineered T-cell receptor (TCR) therapy	Afamitresgene autoleucel (Tecelra)	Engineered T lymphocytes target MAGE-A4	Synovial sarcoma
		Tisagenlecleucel (Kymriah)	Engineered T lymphocytes target CD19 antigen	Lymphoma, leukemia
	CAR T-cell therapy	Axicabtagene ciloleucel (Yescarta)	Engineered peripheral blood T lymphocytes (PBTL) target CD19 antigen	Lymphoma
		Lisocabtagene maraleucel (Breyanzi)	Engineered CD4+ and CD8+ T lymphocytes target CD19 antigen	Lymphoma, leukemia
		Ciltacabtagene autoleucel (Carvykti)	Engineered T lymphocytes target BCMA	Multiple myeloma
		Idecabtagene vicleucel (Abecma)	Engineered PBTLs target BCMA	Multiple myeloma
		Brexucabtagene autoleucel (Tecartus)	Engineered PBTLs target CD19 antigen	Lymphoma, leukemia
Oncolytic Virus Therapy (OVT)		Talimogene laherparepvec (T-VEC, Imlygic)	Selectively replicates in tumors cells to induce lysis, GM-CSF attracts immune cells	Melanoma
	Dendritic cell vaccine	Sipuleucel-T (Provenge)	Generates immune response against PAP	Prostate cancer
Cancer Vaccines	Whole cell vaccine	Bacillus Calmette-Guérin	Mechanism unclear, likely activates a Th1 cytokine response	Bladder cancer
	Personalized cancer vaccine	mRNA-4157	Targets tumor-associated antigens with personalized mRNA	Melanoma

Abbreviations: BCC: Basal Cell Carcinoma; BCMA: B-Cell Maturation Antigen; CD: Cluster of Differentiation; CSCC: Cutaneous Squamous Cell Carcinoma; CTLA4: Cytotoxic T-Lymphocyte Antigen 4; CTL: Cytotoxic T Lymphocyte; GM-CSF: Granulocyte-Macrophage Colony-Stimulating Factor; LAG-3: Lymphocyte Activation Gene-3; MAGE-A4: Melanoma-Associated Antigen A4; MCC: Merkel Cell Carcinoma; NSCLC: Non-Small Cell Lung Cancer; PAP: Prostatic Acid Phosphatase; PD-1: Programmed Death-1; PD-L1: Programmed Death-Ligand 1; PD-L2: Programmed Death-Ligand 2; TAAs: Tumor-Associated Antigens; TNBC: Triple Negative Breast Cancer.

fever, chills, and myalgia, or injection site reactions of redness or swelling. In one study assessing T-VEC in a real-life clinical setting, there was only one cardiac adverse event reported [10]. Studies on the cardiotoxic effects of anticancer vaccines are limited, but the side effects have been reported as flu-like symptoms similar to T-VEC, including fever, chills, headache, and myalgia [11].

Recent advancements in immune therapies include the development of cancer vaccines. One example is the personalized cancer vaccine developed by Moderna and Merck using V940 (mRNA-4157), a personalized neoantigen therapy using mRNA that can encode patient-specific neoantigens, to prevent melanoma recurrence. The open-label phase 2 trial revealed combining mRNA-4157 with Pembrolizumab reduced risk of recurrence or death compared to Pembrolizumab alone. The phase 3 trial of Adjuvant V940 (mRNA-4157) Plus Pembrolizumab Versus Adjuvant Placebo Plus Pembrolizumab in High-Risk Stage II–IV Melanoma Patients is expected to be complete in 2029 [12, 13]. Additionally, the SCIB1 and iSCIB1+ DNA vaccines, in phase 2 of clinical trials, have shown promise in treating melanoma. A preliminary study with SCIB1 has demonstrated a markedly enhanced T cell response, contributing to the eradication of tumors in melanoma patients [14].

Further examples of cancer vaccines include dendritic and whole cell, which employ immunotherapy cancer vaccines sipuleucel-T and Bacillus Calmette-Guérin. Usage of sipuleucel-T has resulted in significant overall survival outcomes for men with metastatic castration-resistant prostate cancer. As an active cellular immunotherapy, the vaccine generates immune responses against PAP. The bacillus Calmette-Guérin immunotherapy is effective in preventing and treating superficial bladder cancer and mechanism behind the vaccine is the potentially activation of TH1 cytokine responses [14, 15].

Symptoms, signs, and laboratory findings

Cardiotoxicity associated with immunotherapy exhibits a spectrum of clinical presentations. Symptoms are contingent upon the specific type of cardiovascular toxicity and may range from asymptomatic presentations to non-specific symptoms like fatigue and palpitations, to more severe clinical manifestations like chest pain and dyspnea and even sudden cardiac death. The median onset of symptoms varies based on the employed immunotherapeutic agents, typically occurring between three months and one year post-treatment, influenced by cancer type and cardiotoxicity nature [2, 16]. Thus, rigorous cardiovascular monitoring is crucial for early detection and management. Table 2 outlines the types of cardiotoxicity, symptoms, diagnostic tests, and potential treatments.

Immunotherapy-associated cardiotoxicities vary by agent, timing, and clinical presentation. Myocarditis induced by ICIs often occurs within the first three months of beginning ICI treatments. Other cardiac complications, such as myocarditis, pericarditis, and cardiomyopathy, have been discovered within 2–17 weeks following ICI treatment initiation [28]. By contrast, cardiotoxicities associated with adoptive T-cell therapies, such as CAR T-cell therapy, occur much sooner at a median of 5–21 days [29]. These differences are due to mechanistic reasons: ICI-related myocarditis may be driven by T-cell infiltration of the myocardium but CAR T-cell cardiotoxicities related to systemic inflammatory surges.

Management strategies for immunotherapy-related cardiotoxicities are generally tailored due to severity. Mild cases may respond well to temporarily ceasing immunotherapy and initiation of high-dose corticosteroids [30]. Moderate or severe cases may require more aggressive treatments or combination immunosuppression. Overall, graded approaches are essential as therapies may be elevated or depressed based on clinical response, biomarkers, and imaging findings.

Future clinical efforts are informed by advances in preclinical research. Currently, to reduce or control immunotoxicities, oncology and cardiology societies are developing new or improving upon current consensus guidelines to optimize screening, identify predictive biomarkers, and study novel therapies for mitigating risks [30]. These efforts may result in standardized clinical protocols and novel therapies that will improve patient outcomes without compromising oncologic benefits of immunotherapy.

Prevention approaches

As immunotherapy becomes more prevalent in cancer treatment, immunotherapy-related cardiotoxicity is a growing concern. Despite widespread use of these therapies, evidence-based cardiovascular screenings or preventative strategies are lacking. Understanding and mitigating this risk is crucial for optimizing patient outcomes while maximizing the therapeutic benefits of immunotherapies.

Preventing cardiotoxicity begins with risk stratification at cancer diagnosis, involving a thorough cardiovascular assessment, including obtaining cardiac history, physical examinations, and other baseline testing. Clinicians should consider factors such as pre-existing cardiovascular diseases, advanced age, and other risks before starting treatments, following guidelines outlined by the Heart Failure Association and International Cardio-Oncology Society [31]. For instance, a study by Drobni et al. shows ICI use was associated with a three-fold increase in the progression of atherosclerotic plaque [32]. Patients receiving corticosteroids during checkpoint therapy showed slower plaque progression (3.5% per year) compared to those not on corticosteroids (6.9% per year).

Many studies also call for baseline cardiac testing with ECG, cardiac stress test, echocardiogram, and troponin measurements to evaluate cardiac function, ischemic burden, and for any cardiac abnormalities prior to starting treatment [2, 6, 16, 31]. Patients at higher risk for cardiovascular toxicities are recommended to be referred to cardiologists or specialized cardio-oncologists for further evaluation and to optimize supportive strategies, such as controlling hypertension or dyslipidemia.

Ongoing monitoring of cardiac function during treatment is critical for early detection of cardiotoxicity. In high-risk patients, regular measurements of cardiac biomarkers (e.g., troponin, BNP), electrocardiograms (ECGs), and echocardiograms may help detect cardiovascular toxicities. A retrospective single center study on ICI-associated myocarditis found a strong association between an increase of cardiac biomarkers and disease

Table 2. Overview of common immune therapy related cardiovascular toxicities [2, 4, 17–27].

Cardiotoxicity Type	Common symptoms	Workup and evaluation	Treatment
Myocarditis	Shortness of breath, palpitations, edema, fatigue, myalgias, low blood pressure, chest pain, asymptomatic	Elevated BNP, NT-proBNP, troponins ECG, TTE, CMR endomyocardial biopsy	High-dose corticosteroids (methylprednisolone, prednisone) Immunosuppressive therapies in steroid refractory cases (alemtuzumab, abatacept, intravenous immunoglobin, mycophenolate)
Pericarditis	Shortness of breath (most common) chest pain, hypotension, fever, light-headedness	Troponin typically not elevated ECG, TTE, CT, CMR	High-dose corticosteroids Colchicine NSAIDs Pericardiocentesis, pericardial window
Arrhythmias	Palpitations, shortness of breath, chest pain/ discomfort, dizziness, fainting, fatigue	ECG	High-dose corticosteroids Antiarrhythmics (beta-blockers, amiodarone, calcium channel blockers) Pacemaker as indicated
Cardiomyopathy	Shortness of breath, edema, fluid overload, palpitations, chest pain	Elevated troponin and NT-proBNP TTE, ECG, PET/CT	Corticosteroids Diuretics Anticoagulants
Myocardial infarction	Chest pain, shortness of breath, diaphoresis, dizziness/lightheadedness, edema, nausea, vomiting, fatigue, tooth pain, jaw pain, neck pain, left arm pain	Elevated troponin ECG	Cardiac catheterization Supportive measures (i.e.: nitroglycerin, anticoagulants, statins)

severity, such as elevations in creatinine kinase, myoglobin, and troponin, occurring before symptom onset, such as diagnosis of arrhythmia or myocarditis. These cardiac biomarkers decreased after the glucocorticoid treatment with methylprednisolone, supporting the benefit of routine monitoring for early detection of immunotherapy-related cardiovascular toxicities [33].

Early referral to a cardiologist for co-management in high-risk populations, such as those with underlying heart conditions or EKGs, is crucial for preventing and managing cardiotoxicities. A multidisciplinary team of interventional cardiologists, cardiac surgeons, cardiologists, and other specialists evaluate patients' comorbidities, clinical stability, etc. to develop a patient-specific treatment plan. The "Heart Team" (HT) approach assesses cardiovascular conditions including peripheral vascular disease, pulmonary embolism, valvular heart disease, and cardiogenic shock to gather clinical data, classifies risk factors, and potentially perform noninvasive testing before the HT meets to create a formal management recommendation [34].

Conclusion

Immunotherapy-associated cardiovascular toxicities, though rare, carry high mortality risks. Their true incidence is understudied, underreported, and expected to increase with increased immunotherapy use. Therefore, developing strategies for risk stratification, prevention, and monitoring is essential. As understanding of these cardiotoxicities evolve, new prevention and management approaches are likely.

Early recognition of symptoms, comprehensive cardiac assessments with baseline diagnostics, and a multidisciplinary approach can help minimize the risk of adverse cardiovascular events.

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Ethics approval

Ethics approval was not required.

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