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Managing Cardiovascular Toxicities in Cancer Therapy

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Abstract: Cancer therapy advancements have significantly increased the survival rates and concurrently led to the rise of the subspeciality of cardio-oncology, due to the cardiovascular side effects associated with cancer therapy. A broad spectrum of cardiotoxic effects arises due to cancer therapy, such as structural damage, cardiac arrhythmias, hypercoagulability, thrombosis, and bleeding risks, ultimately resulting in acute or chronic heart failure. Timely interventions, including early identification of cardiac dysfunction, are adopted to improve clinical outcomes for oncology patients. This review explores the spectrum of cardiotoxic effects due to cancer therapy, as well as screening strategies for cardiovascular dysfunction. It also discusses the underlying mechanisms and risk factors that led to cardiac damage due to oncologic treatment. Prevention and management strategies include the use of modified chemotherapeutic agents, cardio-protectants such as dexrazoxane, medical cardio-protection using ACE inhibitors and ARBs, along with lifestyle modifications. The review also highlights future directions in biomarker-based early detection, monitoring, and targeted interventions of cardiovascular conditions in cancer patients.

Keywords: "*Cardiovascular Toxicity" "Cancer Therapy" "Radiation Therapy" "Arrhythmia" "Left Ventricular Dysfunction" "Anthracyclines" "Biomarkers".*

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I. INTRODUCTION

Over the past decades, major progress in cancer treatment has led to a significant rise in the number of people surviving cancer. About half of cancer patients are expected to live for ten years or more. However, the associated cardiotoxic effects raise serious concerns about the potential medium to long-term cardiovascular disease (CVD) risks with cancer treatment [1]. In addressing these issues, cardiologists and oncologists need to provide treatment according to the prognosis of such patients, which has led to the development of the cardio-oncology specialty [2]. There is an increased risk of cardiovascular conditions, such as including venous thromboembolism, heart failure, cardiomyopathy, arrhythmias, pericarditis, coronary artery disease, stroke, and valvular heart disease, and may last for years after diagnosis in patients receiving cancer treatments [1]. These side effects can impact the continuation of successful cancer therapy and increase morbidity due to the potentially fatal danger and mortality of such conditions. [3]. Common cancer treatments, like radiation therapy and immunotherapy, damage cardiac tissue, and drugs like anthracyclines, Imatinib, used in cancer therapy, cause cardiomyocyte apoptosis[4,5]. Cancer treatment frequently requires management of cardiovascular dysfunction, in which risk-potentiating anti-neoplastic drugs must be temporarily or permanently stopped [6].

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Thus, it is of vital importance to evaluate a patient's baseline cardiovascular risk before initiating cancer therapy, identify and manage cardiovascular issues as soon as they develop during treatment, and establish long-term monitoring strategies into effect for cancer survivors [7].

II. SPECTRUM OF CARDIOTOXIC EFFECTS

Chemotherapeutic drug administration during cancer treatments carries a significant cardiovascular risk [7]. It includes various degrees of arrhythmia, hypertension, thrombosis, myocardial ischemia, and cardiomyopathy, which can lead to heart failure (HF) and left ventricular dysfunction (LVD) [3]. These conditions are associated with high morbidity and disability [5].

- *Radiation Therapy-Induced Cardiac Arrhythmias and Structural Damage*

Most of the cardiotoxicity symptoms are related to adjuvant radiotherapy (RT) induced blood vessel damage [8]. Mediastinal radiation exposure leads to the formation of fibrotic blood vessels, altered conduction system and cardiomyocytes, which can result in cardiac arrhythmias, as well as conditions such as pericarditis, pleural effusions, and atherosclerotic disease, due to mechanisms related to cardiac apoptosis and endothelial damage [7]. Additionally, recognised events are thrombosis, wall rupture, cytoplasmic swelling, and irregularities in the endothelial cell membrane—the reduction in capillary to myocyte ratio results in myocardial death, ischemia, and fibrosis. The normal adipose tissue in the outer layer of the heart is replaced by dense collagen and fibrin, giving rise to pericardial fibrosis, effusion, and tamponade. Coronary artery disease is caused by RT-induced damage to the intima of the coronary artery. Myocardial fibrosis due to RT may impair cardiac compliance, resulting in HF and diastolic function. Cell fibrosis may also predispose the conduction system to arrhythmias [8].

- *Targeted Therapy Effects on Cardiovascular Health*

Targeted cancer therapy, such as epidermal growth factor receptor (EGFR) inhibitors, can cause cardiotoxic effects, including heart failure, prolonged QTc interval, tachycardia, and decreased ejection fraction through mechanisms involving ion channel blocking, oxidative stress, inflammation, and apoptosis. Inhibitors of anaplastic lymphoma kinase (ALK) and c-ros oncogene 1 (ROS1) disrupt the autophagy process, leading to cardiomyocyte death and cardiac injury, resulting in bradycardia, myocarditis, and cardiomyopathy. Bevacizumab and other vascular endothelial growth factor (VEGF) inhibitors cause hypertension, which raises the risk of myocardial ischemia and heart failure [9].

- *Myocardial Damage from Standard Chemotherapeutics*

Standard chemotherapeutics, like anthracyclines, antimetabolites, and cyclophosphamide, can cause irreversible myocardial damage, resulting in LVD and both diastolic and systolic dysfunction, ultimately leading to acute or chronic HF [5]. Improved screening and imaging strategies have helped prevent cardiac dysfunction in cancer treatment, and are aided by administering cardioprotective therapy to regulate cancer therapeutics-related cardiac dysfunction [10].

- *Immunotherapy-Related Cardiovascular Risks*

Another set of drugs that promote T-cell activation and anti-tumour immune response amplification by interfering with immune checkpoint molecules are called immune checkpoint inhibitors (ICIs). Success with ICIs has made it an upcoming treatment method in oncology, but their increased use has also led to an accompanying augmentation of cardiovascular risks. They mainly include myocarditis, pericarditis, LVD, takotsubo-like syndrome, arrhythmias, and ischemia. Myocarditis is the most prevalent pathology and may result in death [11].

- *Thrombosis and Bleeding Risks*

One of the primary factors driving thrombosis is malignancy-induced hypercoagulability, because cancer treatments damage the vascular endothelium and interfere with normal coagulation, they hasten thrombotic events [3]. The most common of these are venous thrombosis, like deep vein thrombosis and pulmonary embolism, and arterial thrombosis, which causes cardiac ischemia and stroke. Venous thrombosis is the second most common factor of morbidity in patients receiving active anti-cancer treatments, like VEGF inhibitors, immunomodulatory drugs, and proteasome inhibitors. There is also a corresponding increase in bleeding risks due to thrombocytopenia induced by cancer therapy [1].

III. MECHANISM AND RISK FACTORS:

Cardiovascular toxicities arise through different molecular and cellular mechanisms specific to each cancer therapy[12]. These mechanisms influence the severity and timing of cardiac injury, shaped further by individual patient risk factors[13]. Doxorubicin and other anthracyclines induce apoptosis and combat cancer by intercalating DNA and inhibiting topoisomerase II. On the contrary, redox cycling produces free radicals and reactive oxygen species (ROS), which cause oxidative stress and mitochondrial dysfunction in cardiomyocytes. This leads to cardiomyopathy, fibrosis, and irreversible myocardial damage[14, 12]. Trastuzumab inhibits HER2-mediated survival pathways in cardiomyocytes. This leads to reversible, non-dose-dependent left ventricular dysfunction by further disrupting the PI3K/Akt pathway, which damages cell metabolism and resilience [14]. VEGF inhibitors cause endothelial dysfunction, oxidative stress, vasoconstriction, systemic hypertension, thrombotic events, and microvascular rarefaction by limiting endothelial survival and nitric oxide production. If untreated, this can lead to heart failure and left ventricular hypertrophy [14, 15, 16].

Radiation therapy-generated ROS are linked to myocardial damage, oxidative stress, inflammation, and cardiac fibrosis [12, 17]. Direct myocardial cell death and

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microvascular damage lead to long-term effects such as restrictive cardiomyopathy and coronary artery disease [15]. Severity depends on exposure to key cardiac structures, treatment type, and dosage [16]. Immune Checkpoint inhibitors (ICI) increase T-cell activity by blocking PD-1, PD-L1, and CTLA-4; however, they can also cause immune-related myocarditis, pericarditis, arrhythmias, and heart failure [12, 18]. Moreover, myocardial inflammation, edema, and fibrosis are brought on by T-cell infiltration, cytokines (IL-6, TNF-α), and inflammasome activation[19]. The risk of cardiotoxicity across cancer therapies is influenced by shared factors such as age, pre-existing cardiovascular disease, cumulative exposure, concurrent cardiotoxic treatments, and metabolic comorbidities [12,13,17,18]. Chronic obesity indices a cardiomyopathy which is characterized by diastolic dysfunction through epicardial fat–driven inflammation and myocardial hypertrophy, potentially compounding cancer therapy–related heart failure[20]. Additionally, oncologic treatment-related immunosuppression may predispose patients to secondary complications like fungal endocarditis, further worsening cardiovascular outcomes [21]. Each therapy also presents specific vulnerabilities: anthracyclines (e.g., doxorubicin) carry a dose-dependent risk exacerbated by genetic predisposition [13]; trastuzumab is associated with a higher risk in patients with prior anthracycline exposure, baseline cardiac disease, or chemotherapy-linked metabolic syndrome [13].

Recognizing heart complications early is key across all cancer treatments. Regular use of tools like troponin tests, echocardiograms, ECGs, and imaging helps catch problems before they become serious [16,17,22]. Integrating multi-speciality care helps tackle these varied risks effectively and also supports safer delivery of therapy [14].Table 1 outlines common cancer therapies, their associated cardiovascular toxicities and mechanisms of injury.

Table 1. Major Cancer Therapies and Associated Cardiotoxicities

Cancer Therapy	Cardiotoxic Effects	Mechanism	References
Anthracyclines (e.g., DOX)	Cardiomyopathy, Heart Failure	ROS generation, DNA intercalation, and topoisomerase II inhibition	**[12,14-16]**
Trastuzumab	Reversible LV dysfunction, HF	HER2 inhibition, PI3K/Akt pathway disruption	**[12,14]**
VEGF Inhibitors	Hypertension, LV dysfunction, Myocardial Ischemia, HF	Endothelial dysfunction, ↓ NO, oxidative stress	**[9,14-16]**
Immune Checkpoint Inhibitors	Myocarditis, Arrhythmias, HF, Pericarditis, Ischemia	T-cell activation, cytokine release, myocardial inflammation	**[11,18,19]**
Radiation Therapy	Fibrosis, Cardiomyopathy, Pericarditis, CAD, Arrhythmias,	ROS generation, fibrosis, endothelial, and myocardial damage	**[8,15-17]**

Abbreviations: DOX - doxorubicin; ROS - reactive oxygen species; LV - left ventricular; NO -nitric oxide; HF - heart failure; CAD - coronary artery disease. ↓ denotes decrease.

IV. SCREENING AND MONITORING

In cancer therapy, screening for cardiotoxicity involves assessing the likelihood of cardiovascular problems as a result of cancer treatments. This is important because more people are surviving cancer owing to advanced treatments, and those undergoing cancer treatment have a higher risk of cardiovascular disease than the general population [23].

Screening is performed in three stages — before, during, and after treatment.

Before treatment, baseline echocardiography assesses heart function to help guide treatment decisions. If heart function is borderline low, such as a left ventricular ejection fraction (LVEF) between 50–55% or if LVEF is below 50% there is a higher risk of cardiotoxicity. Similarly, global longitudinal strain (GLS) is assessed, and a baseline below 16% may indicate early cardiac dysfunction and an increased risk of damage from cancer therapy. In both cases, reduced LVEF or impaired GLS, cardioprotective treatment with ACE inhibitors or ARBs and beta-blockers is usually initiated to help preserve heart function [23].

Due to limitations of echocardiography, such as poor acoustic windows or borderline LVEF results, cardiovascular magnetic resonance imaging (CMR) serves as a valuable secondary test. CMR is used for its better non-radiative tissue and myocardial characterization and to clarify uncertain findings [24]. While transthoracic echocardiography (TTE) remains the primary imaging tool throughout treatment, the frequency of its use is limited by cost and resource availability [25]. This helps in adjusting treatment plans to protect cardiac health[23]

Beyond baseline assessment, monitoring during treatment is important to detect early subclinical myocardial damage and allow timely intervention. While LVEF remains a standard measure, it is insensitive to early cardiac changes. GLS provides a more sensitive marker, with a >13-15%

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reduction from baseline indicating subclinical dysfunction [23]. GLS is also valuable in assessing the risk of cancer therapy–related cardiac dysfunction (CTRCD). An absolute value of <18% during treatment is linked to an elevated risk for CTRCD. However, both baseline and absolute values are mixed or are less predictive with their results, respectively, resulting in the need for GLS monitoring during treatment, offering more useful information than baseline alone [26].

Given the limits in imaging sensitivity and prognostic value, cardiac biomarkers such as natriuretic peptides and troponins have gained in importance as tools for early cardiotoxicity diagnosis during chemotherapy. These biomarkers complement imaging and help detect subclinical dysfunction during cancer therapy [27]. Cardiac troponins (cTn I and T) are important for detecting cardiac injury, especially in patients receiving chemotherapy. High-sensitivity assay allows for diagnosis before irreversible dysfunction develops. While troponin T is less selective, prolonged increases in troponin I suggest an increased risk of LV dysfunction. In addition to showing myocardial strain, NT-proBNP can rise due to non-cardiac issues [28]. Table 2 outlines the screening tools, their purpose and limitations.

Despite stronger cardioprotective therapy in the GLS arm, the SUCCOUR trial (Strain Surveillance of Chemotherapy for Improving Cardiovascular Outcomes) could not find any differences in LVEF changes between the GLS and ejection fraction-guided groups. This suggests that GLS helps with early identification, but further research is needed to see how it affects long-term outcomes [10].

For early diagnosis, risk evaluation, and monitoring for cardiotoxicity, echocardiography, as well as GLS, are vital. Timely interventions, with early identification of subclinical cardiac dysfunction, improve long-term patient outcomes[23].

Table 2. Screening Tools: Uses and Limitations

Screening Tool	Purpose	Limitations	References
Baseline Echocardiography	Assess left ventricular function	Insensitivity of LVEF to early myocardial damage	**[23]**
Global Longitudinal Strain	Detects sub-clinical cardiac dysfunction	Baseline values alone are less predictive	**[23,26]**
Cardiovascular Magnetic Resonance	Myocardial tissue characterization	Costly, less availability	**[24]**
Transthoracic Echocardiography	Imaging modality for cardiac function monitoring	Acoustic window quality, resource availability	**[25]**
Cardiac Biomarkers (cTn I/T, NT-proBNP)	Detects myocardial injury and strain	Less selectivity, arises due to non-cardiac causes	**[27,28]**

Abbreviations: LVEF- Left Ventricular Ejection Fraction, cTn I-Cardiac Troponin I, cTn T-Cardiac Troponin T, NT-pro-BNP-N-terminal pro–B-type Natriuretic Peptide

V. PREVENTION AND MANAGEMENT OF CARDIOTOXICITY

- *Cardiotoxicity Prevention: Liposomal Anthracyclines and Dexrazoxane*

Early prevention and management strategies are necessary to maintain cardiac function during treatment and to improve patient outcomes[29]. Anthracyclines, such as doxorubicin (DOX), are the recommended chemotherapies for malignancies. However, its therapeutic potential is limited by cardiotoxicity. Encapsulating free DOX into a phospholipid bilayer and externally PEGylating (Polyethylene glycol modification) improves the drug's half-life, reduces the volume of distribution, plasma clearance, and lessens the degree of toxicity linked to the use of anthracyclines [30].

Dexrazoxane is a promising agent that works by binding to iron, decreasing the production of harmful free radicals in heart cells, and preventing DNA damage caused by anthracyclines in cancer patients. Adult patients with metastatic breast cancer who had previously received high cumulative doses of anthracyclines made up the majority of randomized trials. Therefore, the Food and Drug Administration indicates that dexrazoxane is only authorised for patients with metastatic breast cancer who might benefit from continued DOX therapy and have received more than 300 mg/m2 of DOX. In a cohort study, it was found that Dexrazoxane decreased the risk of heart failure, and there was no perceptible difference in oncologic outcomes or adverse effects[31].

- *Medical Cardio-Protection*

Angiotensin converting enzyme inhibitors(ACEi) and beta blockers are cardioprotective agents that enable pharmacologic prevention. In the largest clinical trial using β-blockers for cardiotoxicity prevention, the CECCY trial (Carvedilol's Role in Preventing Cardiotoxicity Caused by Chemotherapy) found that the incidence of cardiotoxicity ranged from 13.5% to 14.5%. Carvedilol did not affect the occurrence of early onset LVEF decrease in this case, but diastolic dysfunction and troponin levels were significantly reduced when carvedilol was used [32]. The recommended and authorised strategic management of preventing and treating chemotherapy-induced cardiotoxicity in heart failure

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with left ventricular dysfunction and the event of cardiac damage are beta blockers, angiotensin receptor blockers (ARB), and inhibitors [33].

In more severe cases like New York Heart Association (NYHA) class III or IV heart failure, a QRS length of ≥120–150 ms or left ventricular dysfunction (LVEF<35%) patient may consider cardiac resynchronisation therapy (CRT)[34]. Cancer treatment can also cause secondary hypertension, especially with vascular endothelial growth factor (VEGF) inhibitors and other antineoplastic treatments. Guidelines recommend ACEi and ARBs as first-line treatments in these situations because of their protective effects on the kidneys and endothelium. Calcium channel blockers like nifedipine can also successfully manage hypertension associated with it[35]. QT interval prolongation that arises due to anticancer treatments, which is corrected by cardiac monitoring, caution when using or stopping cancer medications, and correction of electrolyte abnormalities[36]. Cancer patients are also at high risk for developing venous thromboembolism (VTE), especially when using immunomodulatory drugs. This can be prevented by the use of medications like aspirin, low molecular weight heparin, or direct oral anticoagulants for a minimum of 6 months based on notable guidelines[37].

- *Emerging Therapies and Lifestyle Intervention*

Although standard drugs like beta-blockers, ACE inhibitors, and DOX remain fundamental, lifestyle interventions are promising alternatives. Diets high in antioxidants, vitamin E, and vitamin D supplements, and structured exercise regimens may contribute to improved cardiovascular resistance. Additionally, newer agents like sacubitril/valsartan and SGLT2 (sodium-glucose co-transporter 2 inhibitors) are showing healthy promise in this setting[38].

Prevention and management require a multifaceted approach involving the use of liposomal drug formulations, cardioprotective agents, close monitoring, and lifestyle intervention, all without compromising cancer treatment outcomes. Ensuring early risk assessment, patient education, and collaboration between oncology and cardiology teams can further enhance safety and improve the long-term quality of life in cancer patients[39].

VI. SURVIVORSHIP AND SPECIAL POPULATIONS

Long-term cardiovascular risks can persist in cancer survivors despite preventive measures, especially in high-risk populations. Among these, one of the highly vulnerable groups is childhood cancer survivors. Many childhood cancer patients are commonly treated with anthracycline-induced chemotherapy or chest-induced radiotherapy, both of which were found to lead to cardiotoxicity of acute complications such as arrhythmias and heart failure, and chronic conditions such as coronary artery disease (CAD) and cardiomyopathy [40]. Childhood cancer survivors also have a 15-fold greater chance of developing cardiomyopathy than their healthy siblings [41]. A study conducted by the Childhood Cancer Survivor Study (CCSS) on adult survivors of childhood cancers and their siblings compared the mortality of cardiovascular risks. Evidence demonstrated that the risk of heart failure, stroke, and CAD was elevated even 20 years post-treatment. Moreover, these events were shown to happen at a younger age in cancer survivors, and mortality rates were higher than in the non-cancer population. These findings prove that it is important for childhood cancer survivors to continue with lifelong cardiac monitoring and management [42].

The incidence of cardiotoxicity in cancer patients has been shown to rise with age and is more likely in older adults approaching their elderly years. Although there is an increasing number of elderly cancer patients surviving, not enough studies illustrate the cardiotoxicities and their long-term effects. Cardiovascular disease (CVD) risk is more prevalent in elders due to predisposing risk factors such as hypertension, hyperlipidemia, smoking, and obesity, or having diabetes [43]. A secondary analysis of the ASPREE (Aspirin in Reducing Events in the Elderly) trial, a study conducted among healthy older adults to investigate the effects of aspirin on cardiovascular events, was performed on participants who had a prior history of cancer. Results showed an increase in the incidence of CVD, stroke, and myocardial infarction in cancer survivors than those with no cancer history. Higher risks were seen in those treated with cytotoxic chemotherapy [44].

Young pregnant female cancer survivors with prior exposure to anthracycline chemotherapy are shown to have a 40-fold increase in risk of developing left ventricular dysfunction or chronic heart failure. However, studies have found that women with no history of cardiotoxicity due to cancer treatment show a much lower risk in comparison to women with a cardiotoxicity history [45].

Cardiotoxicity can be a major mortality risk, and it is important to monitor and manage this condition while undergoing or after cancer therapy. The emergence of cardio-oncology clinics is crucial for survivorship planning and effective cardiac treatment. The European Society of Cardiology (ESC) guidelines recommend that cardio-oncology clinics should be team-based, involving oncologists, cardiologists, hematologists, and primary risk care staff for surveillance and management [46].

VII. FUTURE DIRECTIONS

Over the next decade, promising developments are expected in cardio-oncology, particularly in the early detection, monitoring, and treatment of cardiovascular conditions in cancer patients. One such development is the use of diagnostic biomarkers, such as microRNA (miRNA), in the potential diagnosis and prognosis of cardiovascular disease. MiRNAs are small, non-coding RNA molecules that regulate

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gene expression and are involved in certain cellular processes in cardiac development. When the heart tissue gets damaged due to factors such as chemotherapy, specific miRNAs are released into the bloodstream. Studies have shown that miRNAs have been used as diagnostic biomarkers in conditions such as acute myocardial infarction (MI) injury, coronary artery disease (CAD), heart failure, and arrhythmias [47]. These conditions may be induced by cancer therapy cardiotoxicity, and miRNAs have shown potential in early detection. Additionally, research is being conducted on new biomarkers for the early diagnosis of cardiotoxicity, particularly in myocardial ischemia, such as fatty acid binding protein (FABP) and glycogen phosphorylase isoenzyme BB (GPBB) [48].

Moreover, genetic and pharmacogenomic testing is now being explored to aid in identifying genetic variants that are more susceptible to cancer therapy-induced cardiotoxicity. Recent studies on childhood cancer survivors have shown specific gene variants that are linked to a higher risk of cardiotoxicity post-chemotherapy. Polymorphisms in genes such as CBR3, which increase anthracycline metabolites, RARG, which affects heart cells' response to anthracyclines, and SLC28A3, which affects how anthracyclines are taken up by heart cells [49]. Understanding these genetic risks can help in identifying vulnerable groups early and help in creating treatment plans accordingly.

Advanced imaging techniques and AI are also shaping the future of cardio-oncology. New non-invasive molecular imaging techniques have emerged, including cardiac magnetic resonance (CMR), myocardial contract echocardiography, and positron emission tomography (PET) scans. These techniques can help to enhance the early detection of cardiac injury [50]. In addition, AI advances have facilitated the analysis of data such as biomarkers detected in blood, ECG results, echocardiography, computed tomography (CT) scan, and CMR in predicting the risk of cardiovascular disease or cardiac injury [51]. Certain AI tools have also shown great performance in echocardiography reading and coronary CT analysis, achieving diagnostic accuracy of over 96%. Moreover, AI can now automatically detect small degrees of heart damage in cardiac MRI with high accuracy, making it helpful in the cardio-oncology field [52].

Large healthcare platforms working with leaders and large-scale societies have initiated the collection of real-world data in hopes of further research and improving the current cardio-oncology care system. Along with data that will provide important information and the use of AI, precision medicine is expected to reduce the future risk of cardiotoxicity due to cancer therapy [53].

VIII. CONCLUSION

As cancer treatment continues to increase the survival rates through its increasing effectiveness, it also contributes to challenges such as cardiotoxicity, which often remain unrecognized. This adversely affects the clinical outcomes of cancer survivors. To effectively address this issue, integration of cardio-oncology services, timely screening, and use of cardio-protective strategies must be taken into account to decrease the cardiovascular burden in cancer patients significantly. Longitudinal studies are essential to assess the long-term cardiovascular outcomes in cancer survivors, especially those exposed to newer targeted and immunotherapeutic agents. Further research is needed to identify reliable early biomarkers and imaging techniques for predicting subclinical cardiac dysfunction. Collaborative effort in cardio-oncology will help deliver effective and safe cancer treatments.

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