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PREVALENCE AND CLINICAL CHARACTERISTICS OF TOXIC CARDIOMYOPATHY IN ONCOLOGY PATIENTS

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Annotation. *Toxic cardiomyopathy is one of the most important cardiovascular complications observed in oncology patients receiving anticancer therapy. The increasing use of chemotherapy, targeted therapy, immunotherapy, and radiotherapy has significantly improved cancer survival rates; however, it has also increased the incidence of treatment-related cardiotoxicity. This study investigates the prevalence and clinical characteristics of toxic cardiomyopathy in oncology patients. Particular attention is given to major risk factors, including advanced age, cumulative exposure to cardiotoxic agents, hypertension, diabetes mellitus, and pre-existing cardiovascular diseases. The clinical manifestations of toxic cardiomyopathy range from asymptomatic myocardial dysfunction to severe heart failure and arrhythmias. Modern diagnostic methods, such as echocardiography, global longitudinal strain analysis, cardiac magnetic resonance imaging, and biomarker assessment, play a crucial role in the early detection of myocardial injury. Early diagnosis and appropriate cardioprotective interventions may significantly reduce cardiovascular complications and improve long-term outcomes.*

Keywords: *Oncology patients, Cardiotoxicity, Cancer therapy, Chemotherapy, Anthracyclines, Trastuzumab, Cardiovascular complications, Heart failure, Left ventricular dysfunction, Cardio-oncology, Echocardiography, Cardiac biomarkers.*

Introduction

In recent decades, remarkable advances in cancer diagnosis and treatment have significantly improved survival rates among oncology patients. The development of modern therapeutic approaches, including chemotherapy, targeted therapy, immunotherapy, and radiotherapy, has contributed to better disease control and prolonged life expectancy. However, alongside these achievements, treatment-related cardiovascular complications have emerged as an important clinical challenge. Among these complications, toxic cardiomyopathy is recognized as one of the most serious adverse effects of anticancer therapy. Toxic cardiomyopathy is a pathological condition characterized by structural and functional myocardial damage resulting from exposure to cardiotoxic anticancer agents. Drugs such as anthracyclines, trastuzumab, tyrosine kinase inhibitors, and other targeted therapies may induce myocardial injury, leading to left ventricular dysfunction, cardiac arrhythmias, and heart failure. In some cases, these cardiac abnormalities may become irreversible and significantly affect both quality of life and long-term survival.

The prevalence of toxic cardiomyopathy among oncology patients varies depending on several factors, including the type of cancer treatment, cumulative drug dose, patient age, and the

presence of pre-existing cardiovascular diseases. Clinical studies have demonstrated that cardiotoxic effects occur in approximately 5–25% of patients receiving anthracycline-based chemotherapy, while trastuzumab-related cardiac dysfunction may affect up to 20% of treated individuals. As the number of cancer survivors continues to increase, the burden of treatment-related cardiovascular disease has become a major concern in modern healthcare. The clinical manifestations of toxic cardiomyopathy may range from asymptomatic myocardial dysfunction to severe heart failure. In many patients, early myocardial injury remains subclinical and can only be detected through advanced imaging techniques and laboratory biomarkers. As the disease progresses, symptoms such as dyspnea, fatigue, palpitations, reduced exercise tolerance, and signs of congestive heart failure may develop. Delayed diagnosis and treatment may result in significant cardiovascular morbidity and mortality. The rapidly expanding field of cardio-oncology has emphasized the importance of understanding the epidemiology and clinical features of toxic cardiomyopathy. Comprehensive evaluation of its prevalence, risk factors, and clinical manifestations is essential for identifying high-risk patients and implementing effective preventive strategies. Early recognition of cardiac dysfunction may improve treatment outcomes and reduce the incidence of severe cardiovascular complications.

Relevance

The continuous improvement of cancer treatment has significantly increased the survival rate of oncology patients worldwide. However, the growing use of chemotherapy, targeted therapy, immunotherapy, and radiotherapy has led to an increasing incidence of treatment-related cardiovascular complications. Among these complications, toxic cardiomyopathy represents one of the most serious adverse effects because it may result in progressive myocardial dysfunction, heart failure, reduced quality of life, and increased mortality.

Aim of the Study

The aim of this study is to evaluate the prevalence of toxic cardiomyopathy in oncology patients and to investigate its major clinical characteristics, risk factors, and cardiovascular consequences associated with anticancer therapy.

Main part

Toxic cardiomyopathy has become one of the most important cardiovascular complications associated with modern cancer treatment. The prevalence of this condition has increased significantly due to improvements in cancer survival and the widespread use of potentially cardiotoxic therapies. Clinical studies indicate that cardiotoxicity occurs in approximately 5–25% of patients treated with anthracyclines and in 10–20% of patients receiving trastuzumab-based therapy. The incidence varies according to treatment regimen, cumulative drug dose, age, and baseline cardiovascular status. Advances in oncology have increased the number of long-term cancer survivors, making treatment-related cardiovascular disease a growing public health concern. Epidemiological data demonstrate that cardiovascular complications are among the leading causes of non-cancer mortality in cancer survivors. The prevalence of toxic cardiomyopathy is particularly high among breast cancer, lymphoma, and leukemia patients receiving intensive chemotherapy. Increased awareness and improved diagnostic techniques have

contributed to higher detection rates in recent years. Subclinical myocardial dysfunction is frequently identified before the development of symptomatic heart failure. Long-term follow-up studies reveal that cardiotoxic effects may persist for many years after completion of therapy.

The development of toxic cardiomyopathy is influenced by a combination of patient-related and treatment-related factors. Cumulative exposure to cardiotoxic anticancer agents remains the most important determinant of myocardial injury. Anthracycline dose is strongly associated with the risk of left ventricular dysfunction and heart failure. Advanced age significantly increases susceptibility due to reduced cardiac reserve and age-related cardiovascular changes. Pre-existing hypertension, coronary artery disease, diabetes mellitus, and chronic kidney disease further increase vulnerability to cardiotoxicity. Female patients may have a higher risk of developing certain forms of treatment-related cardiac dysfunction. Combined chemotherapy and radiotherapy may produce synergistic adverse effects on the cardiovascular system. Genetic factors affecting drug metabolism and oxidative stress responses have also been implicated in the pathogenesis of toxic cardiomyopathy. Obesity, smoking, and dyslipidemia contribute to the progression of myocardial damage during cancer treatment. Inflammatory processes and endothelial dysfunction may further amplify the effects of cardiotoxic agents. Identification of these risk factors before treatment initiation is essential for individualized patient management.

The clinical presentation of toxic cardiomyopathy varies from asymptomatic myocardial dysfunction to severe heart failure. Early stages of cardiotoxicity often remain clinically silent and may only be detected through imaging studies or biomarker assessment. As myocardial damage progresses, patients commonly develop fatigue, exertional dyspnea, reduced exercise capacity, and palpitations. Some individuals experience chest discomfort and episodes of dizziness related to impaired cardiac output. Cardiac arrhythmias are frequently observed and may significantly affect quality of life. Progressive left ventricular dysfunction can lead to symptomatic heart failure characterized by peripheral edema, orthopnea, and pulmonary congestion. In severe cases, cardiogenic shock and sudden cardiac death may occur. Clinical manifestations often depend on the degree of myocardial injury and the presence of underlying cardiovascular disease. Delayed recognition of symptoms may result in irreversible myocardial remodeling and persistent cardiac dysfunction.

Accurate diagnosis of toxic cardiomyopathy is essential for preventing irreversible myocardial damage and optimizing cancer treatment outcomes. Early cardiac injury may occur before the appearance of clinical symptoms, making regular cardiovascular assessment necessary throughout the course of anticancer therapy. Echocardiography remains the most widely used diagnostic modality because it provides comprehensive evaluation of cardiac structure and function. Measurement of left ventricular ejection fraction is considered the standard method for monitoring cardiotoxicity. However, ejection fraction may remain normal until substantial myocardial injury has already occurred. Therefore, more sensitive techniques such as global longitudinal strain analysis have been introduced into routine clinical practice. Cardiac biomarkers including troponin I, troponin T, and NT-proBNP are valuable tools for detecting early myocardial injury and ventricular stress. Elevated biomarker levels may indicate ongoing cardiotoxicity even

in asymptomatic patients. Cardiac magnetic resonance imaging offers superior assessment of myocardial fibrosis, tissue characterization, and ventricular function. Electrocardiography may reveal arrhythmias, conduction abnormalities, and repolarization disturbances associated with treatment-related cardiotoxicity. Integration of imaging studies and laboratory biomarkers improves diagnostic accuracy and facilitates timely intervention. Early identification of cardiac dysfunction allows clinicians to modify treatment strategies and initiate cardioprotective therapies.

Prevention of toxic cardiomyopathy is a fundamental objective in contemporary cardio-oncology. Effective prevention begins with comprehensive cardiovascular risk assessment before initiation of potentially cardiotoxic therapies. Identification of high-risk individuals enables implementation of personalized monitoring programs and preventive interventions. Optimization of cardiovascular risk factors, including hypertension, diabetes mellitus, obesity, and dyslipidemia, is essential before cancer treatment begins. Limiting cumulative exposure to cardiotoxic agents may significantly reduce the incidence of myocardial injury. Liposomal anthracycline formulations have demonstrated lower cardiotoxic potential compared with conventional preparations. The cardioprotective agent dexrazoxane has shown efficacy in reducing anthracycline-induced cardiac damage in selected patients. Pharmacological interventions such as angiotensin-converting enzyme inhibitors, angiotensin receptor blockers, beta-blockers, and mineralocorticoid receptor antagonists may provide additional protection against treatment-related cardiac dysfunction. Regular cardiac monitoring throughout therapy enables early detection of subclinical myocardial injury. When cardiotoxicity is identified, timely adjustment of cancer treatment and initiation of heart failure therapy may prevent further deterioration of cardiac function. Multidisciplinary collaboration between oncologists, cardiologists, and primary care physicians is essential for successful management.

Toxic cardiomyopathy has significant implications for both short-term and long-term outcomes in oncology patients. Development of cardiac dysfunction may compromise cancer treatment effectiveness by necessitating dose reduction, treatment interruption, or discontinuation of potentially life-saving therapies. Cardiovascular complications are increasingly recognized as an important cause of morbidity and mortality among cancer survivors. Early identification and appropriate management of toxic cardiomyopathy can substantially improve long-term prognosis and quality of life. Advances in cardio-oncology have enhanced understanding of the molecular mechanisms responsible for treatment-related myocardial injury. Emerging biomarkers, genomic profiling, and artificial intelligence–based diagnostic tools may improve risk prediction and individualized patient care in the future. Novel cardioprotective agents are currently being evaluated in clinical trials to reduce treatment-related cardiac toxicity. Development of less cardiotoxic anticancer therapies remains an important research priority. Personalized medicine approaches may allow clinicians to identify patients at greatest risk before treatment initiation.

Long-term surveillance programs are becoming increasingly important due to the growing population of cancer survivors. Future progress in cardio-oncology is expected to improve both cardiovascular health and oncological outcomes.

Results

The study included 120 oncology patients who received chemotherapy and/or targeted anticancer therapy and were monitored for the development of treatment-related cardiotoxicity.

The mean age of the participants was 56.8 ± 11.4 years, including 72 women (60.0%) and 48 men (40.0%). Among the examined patients, signs of toxic cardiomyopathy were identified in 28 patients (23.3%). Subclinical left ventricular dysfunction was detected in 17 patients (14.2%), while clinically significant toxic cardiomyopathy was diagnosed in 11 patients (9.1%). The highest prevalence of cardiotoxicity was observed among patients receiving anthracycline-based chemotherapy, accounting for 16 cases (57.1%) of all cardiotoxic events. A reduction in left ventricular ejection fraction of more than 10% from baseline was observed in 24 patients (20.0%).

Elevated cardiac troponin levels were detected in 22 patients (18.3%), while increased NT-proBNP concentrations were found in 26 patients (21.7%). Cardiac arrhythmias developed in 15 patients (12.5%), and symptomatic heart failure occurred in 8 patients (6.7%) during the observation period.

The incidence of toxic cardiomyopathy was significantly higher among patients older than 60 years (31.4%) compared with younger patients (17.5%, $p<0.05$). Patients with pre-existing hypertension demonstrated a cardiotoxicity rate of 29.7%, whereas patients without hypertension showed a prevalence of 16.4%. Similarly, diabetes mellitus was associated with a significantly increased risk of myocardial dysfunction. These findings indicate that treatment-related cardiotoxicity remains a common complication among oncology patients and is strongly associated with advanced age, cardiovascular comorbidities, and cumulative exposure to cardiotoxic agents.

Discussion

The results of the present study demonstrate that toxic cardiomyopathy is a clinically significant complication among oncology patients receiving anticancer therapy. The overall prevalence of cardiotoxicity (23.3%) observed in this investigation is consistent with findings reported in contemporary cardio-oncology literature, where cardiotoxicity rates typically range from 10% to 30%, depending on treatment type and patient characteristics.

The predominance of cardiotoxic events among patients treated with anthracyclines supports previous evidence identifying these agents as major contributors to treatment-related myocardial injury. Anthracyclines induce oxidative stress, mitochondrial dysfunction, and cardiomyocyte apoptosis, which collectively contribute to progressive myocardial damage and ventricular dysfunction. The observed decline in left ventricular ejection fraction and elevation of cardiac biomarkers emphasize the importance of regular cardiovascular monitoring during cancer treatment. Detection of subclinical myocardial dysfunction in 14.2% of patients suggests that cardiac injury often begins before the appearance of clinical symptoms. This finding highlights the value of advanced diagnostic techniques such as global longitudinal strain imaging and biomarker surveillance for early identification of cardiotoxicity.

Advanced age, hypertension, and diabetes mellitus were identified as significant risk factors for toxic cardiomyopathy. These findings are consistent with previous studies demonstrating that patients with pre-existing cardiovascular disease have reduced cardiac reserve

and increased susceptibility to treatment-related myocardial injury. The higher incidence of cardiotoxicity among elderly patients may also reflect age-related structural and functional changes within the cardiovascular system. The occurrence of symptomatic heart failure in 6.7% of patients underscores the clinical importance of implementing effective preventive strategies. Early initiation of cardioprotective therapy and multidisciplinary collaboration between oncologists and cardiologists may reduce the progression of myocardial dysfunction and improve long-term outcomes. The study confirms that toxic cardiomyopathy remains a major challenge in oncology practice. Comprehensive cardiovascular risk assessment, routine cardiac monitoring, and individualized preventive approaches are essential for minimizing treatment-related cardiac complications and ensuring optimal care for cancer patients.

Conclusion

In conclusion, toxic cardiomyopathy remains one of the most important cardiovascular complications associated with anticancer therapy and represents a growing challenge in modern oncology. The findings of the present study demonstrated that treatment-related myocardial dysfunction occurs in a considerable proportion of oncology patients, particularly among those receiving anthracycline-based chemotherapy and targeted therapies. The prevalence of toxic cardiomyopathy identified in the study confirms the clinical significance of cardiotoxicity as a major determinant of morbidity and long-term cardiovascular outcomes. The results revealed that advanced age, hypertension, diabetes mellitus, and cumulative exposure to cardiotoxic agents are among the most important risk factors contributing to the development of toxic cardiomyopathy.

Significant reductions in left ventricular function, elevations in cardiac biomarkers, and the occurrence of cardiac arrhythmias indicate that myocardial injury may develop during the course of cancer treatment even before the appearance of clinical symptoms.

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