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CHEMOTHERAPY-INDUCED CARDIOTOXICITY: MECHANISMS, CLINICAL MANIFESTATIONS, AND MANAGEMENT STRATEGIES

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ABSTRACT

Chemotherapy-induced cardiotoxicity (CIC) is a major limitation in cancer therapy, contributing to long-term morbidity and mortality. Various agents such as anthracyclines and trastuzumab cause cardiac dysfunction through oxidative stress, mitochondrial damage, and apoptosis. Early diagnosis and preventive strategies are essential for improving outcomes.

INTRODUCTION

Advancements in chemotherapy have significantly improved cancer survival, but cardiovascular complications remain a concern. Cardiotoxicity includes heart failure, arrhythmias, and hypertension.

CLASSIFICATION

Type I cardiotoxicity is irreversible and dose-dependent, commonly caused by anthracyclines. Type II is reversible and associated with trastuzumab.

MECHANISMS

Mechanisms include oxidative stress, mitochondrial dysfunction, apoptosis, inflammation, and HER2 inhibition.

RISK FACTORS

High cumulative dose, age, pre-existing heart disease, diabetes, and combination therapies increase risk.

CLINICAL MANIFESTATIONS

Includes asymptomatic dysfunction, heart failure, arrhythmias, hypertension, and ischemia.

DIAGNOSIS

Echocardiography, cardiac MRI, and biomarkers like troponin and NT-proBNP are used.

PREVENTION

Dose limitation, liposomal formulations, dexrazoxane, ACE inhibitors, and beta-blockers.

MANAGEMENT

Heart failure therapy, chemotherapy modification, and multidisciplinary care.

CONCLUSION

Early detection and integrated cardio-oncology approaches are essential to reduce morbidity and mortality.

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