Molecular mechanisms and cardio protection techniques for cardio toxicity of anticancer medications.

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Introduction

Cardiology patients now have much better prognoses because to chemotherapy and tailored medicines. Certain anti-cancer medications, nevertheless, can also have unfavorable cardiovascular side effects, which can cause cardiac dysfunction to manifest suddenly or gradually. Regardless of the oncological prognosis, these common cardiovascular complications, also known as cardio toxicity, can have a significant impact on quality of life and overall survival. They may also necessitate the modification, suspension, or withdrawal of life-saving antineoplastic therapies, which runs the risk of reducing their efficacy. The type, dose, method, and duration of anticancer medication treatment, as well as unique risk factors, may all affect whether cardio toxicity manifests. Notably, the cardio toxic adverse effects may be reversible if heart function returns after medication is stopped or irreversible, as is the case with injury and loss of cardiac muscle cells. Anticancer treatments' subclinical cardiac dysfunction may later progress into symptomatic congestive heart failure. Hence, the development of cardio protective medicines is urgently required to minimize the acute or chronic expression of heart damage as well as to slow the start and progression of clinical and subclinical cardio toxicity. In this review, we provide an overview of our current understanding of the cellular and molecular processes involved in the beginning of cardio toxicity brought on by popular types of chemotherapy and targeted therapy medications. Also, we explore cardio protective preventative measures that might be a valuable addition to anticancer therapy as well as present and future techniques to deal with the cardio toxic side effects [1].

Unfortunately, many chemotherapy drugs have negative effects on the heart, which can cause cardiac dysfunction to manifest suddenly or gradually over time. This condition is known as cardio toxicity. Although there is no agreed-upon definition of cardio toxicity, in clinical practice, it frequently denotes deterioration in patients' heart function as shown by left ventricular ejection fraction (LVEF). Cardio toxicity has been defined by several organizations and clinical committees using various threshold LVEF decreases. Therapy with anthracyclines, the chemotherapy medication family that caused the most worries about cardio toxicity, is linked to an incidence of cardiac dysfunction that ranges from 2% to 48%. Cardio toxicity was defined by the Cardiac Review and Evaluation Committee (CREC) as a decrease in LVEF of at least 5% to below 55% with concurrent signs or symptoms of congestive heart failure (CHF), or a decrease in LVEF of at least 10% to below 55% without associated signs or symptoms. The CREC retrospective study evaluated the cardiotoxicity of the anti-HER2 agent trastuzumab with or without concurrent anthracycline treatment [2].

The major mechanism appears to be mediated by vascular malfunction and/or thromboembolic ischemia; however administration of alkylating medications and fluoropyrimidines may also cause cardiomyocyte death and hence irreparable cardiac damage. Anticancer medications, however, can potentially compromise cardiomyocyte performance without resulting in cell death. It is linked to a reduced incidence of HF and is often reversible heart dysfunction (type II cardio toxicity). The dysregulation of cardiomyocyte-intrinsic pathways and/or modification of other cardiac populations and external factors, particularly paracrine factors, which in turn affect cardiomyocyte function, have been proposed as potential mechanisms for reversible cardio toxicity. Targeting monoclonal antibodies or tyrosine kinase inhibitors (TKIs) are frequently linked to reversible cardiac damage, and their negative effects result from the signaling impairment of cardio protective molecules for cardiomyocytes, such as Neuregulin-1 (NRG1), or for other cardiac cell populations, such as vascular endothelial growth factor (VEGF), and platelet-derived growth factor [3].

Cardiovascular side effects of antineoplastic therapy pose serious risks to the health of cancer patients and may make the decision to continue or stop treatment difficult. Today, some medications have undergone clinical trials to combat the cardio toxicity associated with anticancer treatment. Here, we propose a further assessment of variables, which are currently mainly recognized for their role in cardiomyocyte proliferation and survival, as promising approaches for the protection and/or regeneration of the cardiac tissue. In order to guarantee cancer patients a long-term relapse-free survival and top-notch cardiovascular health, an increased synergistic effort would be needed for the oncologic and cardiologic research sectors [4].

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