Opinion
Volume 10:10, 2022

Journal of Cardiovascular Diseases & Diagnosis

ISSN: 2329-9517 Open Access

Treatment and Evaluation of Cancer and Heart Disease Patients: Cardio-Oncology

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Introduction

Patients receiving treatment for various cancers may experience serious consequences due to the cardiotoxicity of anticancer drugs. The molecular site of action, the immediate and cumulative dose, the mode of administration, the presence of any underlying cardiac conditions, and the demographics of the patient are just a few of the variables that affect how severe this toxicity is. Additionally, treatment with other antineoplastic drugs in the past or present may have had an impact on toxicity. Cardiotoxic side effects may appear right away after drug administration or they may take months or years to become apparent after the patient has had treatment. In this article, we examine some recently approved drugs, as well as some regularly used chemotherapy agents, for their likelihood to produce cardiotoxicity. To more precisely identify which people are at risk for developing cardiotoxicity, more research will be needed. Plans for management and methods to lessen cardiotoxicity must also be established [1].

Description

Cancer treatment has advanced significantly in recent years, and significant strides have been made in lowering the morbidity and mortality from many types of cancer. A new idea is that cancer can be controlled like diabetes or hypertension through early detection, routine monitoring, and coordinated therapeutic decision-making. Therefore, limiting concomitant conditions is crucial for cancer survivors. The risk of heart disease for many cancer survivors is actually equal to the risk of developing recurrent cancer. Patients with cancer today have access to more sophisticated drug regimens, radiation treatment, and surgical procedures as treatment alternatives. The outcomes for patients are likely to be significantly impacted by several of these treatments, many of which have major potential adverse cardiac effects. Therefore, for these consequences to be effectively managed, recognising them is essential. This review's objective is to highlight drugs used often to treat cancer and their related cardiovascular side effects [2].

Each chemotherapeutic drug has distinct cardiac effects and the capacity to amplify the negative effects of other drugs. Another significant factor in amplifying damage is radiation therapy. It is crucial to keep in mind that intensively treated cancer patients frequently have severe illnesses, and cause-and-effect correlations are frequently ambiguous. So, using a review of the literature as well as the vast clinical expertise of the Department of Cardiology at The University of Texas M.D. Anderson Cancer Centre, we make an effort to summarise the current state of knowledge on the cardiovascular side effects of cancer therapy in this review.

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Date of Submission: 03 October, 2022, Manuscript No. jcdd-22-82103; **Editor assigned:** 04 October, 2022, PreQC No. P-82103; **Reviewed:** 14 October, QC No. Q-82103; **Revised:** 21 October, 2022, Manuscript No. R-82103; **Published:** 28 October, 2022, DOI: 10.37421/2329-9517.2022.10.522

Grading cardiovascular toxicity

Preclinical and clinical occurrences may reflect cardiovascular harm. Histopathological or biochemical methods can be used to detect preclinical toxicity. For instance, doxorubicin-induced myocardial damage may show up in endomyocardial biopsy specimens without necessarily increasing troponin T or I protein levels. Laboratory cardiovascular abnormalities are not taken into account in the grading scheme that the World Health Organization proposed in 1981 to standardise the reporting of medication side effects. The common toxicity criteria, a more thorough system created by the National Cancer Institute, takes into account all significant clinical and laboratory abnormalities [3].

Anthracyclines/Anthraquinolones

The anticancer medications with known cardiotoxicity that have the most research behind them are anthracyclines. Numerous hematologic and solid cancers are treated using the anthracyclines doxorubicin, daunorubicin, and epirubicin, which have received approval from the Food and Drug Administration. ST-segment and T-wave abnormalities are non-specific symptoms of acute cardiotoxicity. Contrary to early effects, late anthracycline cardiotoxicity builds up over time, is dose dependent, and, at sufficiently high doses, can cause congestive heart failure (CHF) and left ventricular (LV) dysfunction. The production of free radicals is assumed to be the mechanism for the direct myocardial damage. When individuals receive doxorubicin doses greater than 550 mg/m^2, the frequency of cardiomyopathy dramatically rises. However, more recent research has revealed that comparable cardiomyopathy can be brought on by smaller cumulative doses. Although the mortality rate among individuals who really experience late cardiotoxicity has been thought to be high, early diagnosis and treatment can significantly improve the grim prognosis. When taken at present levels, the anthracycline derivative mitoxantrone can also result in moderate cardiotoxicity comparable to that brought on by anthracyclines [4].

Risk factors for developing cardiovascular complications

A medicine's cardiotoxicity is influenced by a variety of factors that are both specific to the drug and unique to each patient. Understanding these elements might make cardiovascular adverse effects less common or less severe. Some crucial medication-related elements to take into account include the dose of the medicine administered during each session, the cumulative dose, the delivery schedule, the mode of administration, the combination of drugs given, and the order in which these drugs are administered. Age, prior cardiovascular disease, radiation therapy, metabolic disorders, and medication hypersensitivity are also patient-related factors. Focusing prevention efforts to reduce cardiotoxicity can be made easier by being aware of the risk factors for circulatory complications brought on by chemotherapy [5].

Conclusion

Some chemotherapy drugs only cause cardiotoxicity when given in high doses; examples include CHF and pericarditis in the case of platinum drugs, atrial fibrillation in the case of melphalan, systolic dysfunction and pericarditis in the case of cyclophosphamide, and LV dysfunction in the case of anthracyclines. Ifosfamide induces CHF at doses of 10 to 18 g/m^2, but only at doses of 1.2 to 2 g/m^2 each day for five days. When administered intravenously, busulfan causes tachyarrhythmias, hypertension or hypotension, and LV dysfunction but not when consumed orally. Changing the order in which medications are given can

help lessen cardiotoxicity. For instance, giving interferon alone for the first two weeks, then IL-2, had far less cardiovascular damage than the combination of IL-2 and interferon. If the gap between doxorubicin and paclitaxel was 15 to 30 minutes, the combination of paclitaxel and doxorubicin induced CHF in 20% of instances, but the cardiotoxicity of this combination was decreased when the gap was 4 to 16 hours.

Acknowledgement

None.

Conflict of Interest

None.

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How to cite this article: Bergh, Jonas. "Treatment and Evaluation of Cancer and Heart Disease Patients: Cardio-Oncology." J Cardiovasc Dis Diagn 10 (2022): 522.