Short Communication
Volume 10:12, 2022

Journal of Cardiovascular Diseases & Diagnosis

ISSN: 2329-9517 Open Access

Influence of Existing Cardiovascular Disease on Lung Cancer Patient Treatment Regimens and Survival Rates

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Introduction

Due to the cardiotoxicity of anticancer medications, patients receiving treatment for various cancers may experience serious side effects. A few of the factors that influence the severity of this toxicity include the patient's demographics, the molecular site of action, the immediate and cumulative dose, the mode of administration, the presence of any underlying cardiac conditions and the molecular site of action. Toxicity may also have been affected by previous or current treatment with other antineoplastic drugs. Cardiotoxic side effects may manifest immediately following drug administration or may take months or years to manifest after treatment has been administered. In this article, we investigate the likelihood of cardiotoxicity in a number of commonly used chemotherapy agents and recently approved drugs. To all the more unequivocally recognize which individuals are in danger of creating cardiotoxicity, more exploration will be required. Additionally, management plans and strategies for reducing cardiotoxicity must be established [1].

Discussion

The treatment of cancer has come a long way in recent years and many types of cancer's morbidity and mortality have been reduced significantly. A groundbreaking thought is that malignant growth can be controlled like diabetes or hypertension through early identification, routine observing and composed helpful independent direction. For cancer survivors, therefore, limiting concurrent conditions is essential. For many cancer survivors, the risk of developing heart disease is actually the same as the risk of developing recurrent cancer. Alternative treatments for cancer patients today include more advanced drug regimens, radiation therapy and surgical procedures. Several of these treatments, many of which have significant potential adverse cardiac effects, are likely to have a significant impact on patients' outcomes. Recognizing these consequences is therefore essential for effective management. The goal of this review is to highlight common cancer treatments and their 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 Center, 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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Received: 02 December, 2022, Manuscript No. jcdd-22-85126; **Editor assigned:** 03 December, 2022, PreQC No. P-85126; **Reviewed:** 16 December, 2022, QC No. Q-85126; **Revised:** 21 December, 2022, Manuscript No. R-85126; **Published:** 28 December, 2022, DOI: 10.37421/2329-9517.2022.10.531

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].

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

High doses of certain chemotherapy drugs only result in cardiotoxicity; CHF and pericarditis with platinum drugs, atrial fibrillation with melphalan, systolic dysfunction with pericarditis with cyclophosphamide and LV dysfunction with anthracyclines are all examples. At doses of 10 to 18 g/m^2, ifosfamide causes CHF, but only for five days at doses of 1.2 to 2 g/m2. Busulfan causes tachyarrhythmias, hypertension or hypotension and LV dysfunction when given intravenously, but not when taken orally. Cardiotoxicity can be reduced by changing the order in which medications are administered. For instance, compared to administering interferon and IL-2 simultaneously for the first two

weeks, administering interferon alone caused significantly less cardiovascular damage. The combination of paclitaxel and doxorubicin caused CHF in 20% of cases when there was a gap of 15 to 30 minutes between the two drugs. However, the cardiotoxicity of this combination decreased when there was a gap of 4 to 16 hours.

Acknowledgement

None.

Conflict of Interest

None.

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How to cite this article: Cheung, Winson. "Influence of Existing Cardiovascular Disease on Lung Cancer Patient Treatment Regimens and Survival Rates." J Cardiovasc Dis Diagn 10 (2022): 531.