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Cancer Patient Survival Rates have increased and Clinical Experience

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Abstract

Chemotherapy, radiation, targeted therapy, and immunotherapy have given cancer patients hope. As cancer patient survival rates have increased and clinical experience has grown, interest in cardiovascular damage caused by cancer therapy has grown. To develop appropriate preventative and therapeutic strategies for the side effects of cancer treatment that can be fatal or cause long-term morbidity, a greater comprehension of the disease's molecular biology is required. In addition to common cardio protection medications, formulations of traditional Chinese medicine can be expected to achieve "tailored treatment" from a variety of angles. In addition, the emergence of "reverse cardio-oncology" in response to the rising incidence of cancer among cardiovascular disease patients emphasizes the urgent requirement for collaboration between cardiologists and oncologists.

Keywords: Cancer radiation therapy • Antioxidants • Mitochondrial autophagy • Chronic cardio toxicity

Introduction

As cancer mortality declines and the population that is still alive ages, the proportion of cancer patients who also have heart disease rises. Cancer treatment is linked to an increased risk of cardiovascular disease in cancer survivors, who are more likely to develop it than to have a tumor recur. Heart failure and arrhythmia are two common cardiovascular side effects of anticancer drugs. Older breast cancer survivors have a higher risk of dying from heart disease than from the disease itself. Cardio toxicity has also overtaken the tumor as the second leading cause of long-term mortality, according to a cohort study of pediatric cancer survivors. Cardio toxicity in juvenile patients receiving DOX was made. Since then, additional initiatives have been launched to separate the cardio toxic and anticancer effects of ANTs and further reduce toxicity. ANT's cardio toxicity has also received more attention at the same time. The cumulative dose, administration schedule, and age all had an effect on the risk, according to studies. Additionally, the DOX-induced regulation of mitochondrial autophagy may be linked to cytoplasmic p53, which binds to Parking and prevents it from locating to mitochondria to inhibit phagocytosis. Activation of the pathway, which encourages translocation to mitochondria, has been suggested by other studies to be the cause of cardio toxicity and the signs of it. Reduced mitophagy also prevents cardiomyocyte cell death and reduces mitochondrial dysfunction.

Description

As a result of a thorough understanding of the mechanism by which cancer develops, monoclonal antibodies, inhibitors, immunotherapy, and other medications have been developed. The cardio toxicity that these medications cause is a major factor in patient survival, prognosis, and quality of life, despite their effectiveness in treating cancer. As a result, the field of "cardio-oncology" is now more advanced. Primary cardiomyopathy may be to blame for decreased heart function following cancer treatment because myocardial cells are directly damaged. Secondary cardiomyopathy is caused by changes in the innervation or the hormonal system. Instead, myocarditis is caused when inflammatory cells invade the myocardium. The groundwork of cardio-oncology. The other hand the dangerous cycle it. It is essential to strike a balance between antitumor efficacy and cardiac events associated with cancer therapy in order to pursue optimal cancer care, which includes extending meaningful lives and providing individuals with supportive cardiovascular care. The mitochondria expand as a result of ROS action, which opens the mitochondrial permeability transition pore and reduces the mitochondrial membrane potential. Abnormal mitochondrial structure and mitochondrial damage have indeed been observed in neonatal rat cardiomyocytes exposed to DOX [1].

Researchers have focused on preventing and reducing cardio toxicity associated with cancer treatment in order to accomplish this objective. Attempts to combine cardiovascular and anticancer medications without affecting the effectiveness against cancer while simultaneously cleansing the heart have led to the development of "tumor cardiology." Traditional cardio-oncology focuses primarily on cancer patients' cardiovascular disease risk. The likelihood that people with cardiovascular disease will develop cancer in the future is the foundation of reverse cardio-oncology. The two disorders appear to interact in a complex way through these two developmental pathways. An understanding of this bidirectional interaction can be beneficial to the prevention and treatment of linked disorders, as well as the collaboration between cardiology and cancer. In this article, we look at how cancer treatment, combination therapy, and some recent developments in cardio-oncology reverse cardio toxicity. Advanced cancer is mostly treated with chemotherapy, radiation, targeted therapy, and immunotherapy; every single one of them has been shown to be bad for the cardiovascular system. Chemotherapy and radiation therapy-related cardio toxicity is the leading cause of morbidity and mortality in cancer survivors [2].

The use of anti-nausea drugs during chemotherapy has been linked to known cardio toxicity. On the other hand, relatively new medications like target therapy and immunomodulation have not been linked to many reports of cardio toxicity. The method that ANTs use to fight cancer involves damaging DNA. They mostly affect cells that are growing in the S and G2 stages. The parent nucleus of the anthracite ring forms a relatively stable complex by non-specific insertion parallel to the DNA base pair. The positive charge of the parent nucleus has a strong affinity for DNA because it has a

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negative charge. The Quinone structure of the molecule makes it simple for it to participate in oxygen free radical-generating electron transfer processes. ANTs, on the one hand, are embedded in DNA and prevent DNA replication and transcription. However, the use of ANTs increases the production of reactive oxygen species, which in turn results in double-strand breaks in DNA and oxidative damage to nucleic acids. ANTs also have a direct impact on topoisomerase DNA unwinding, helicase activity, and the subsequent strand separation of DNA. Acute and chronic cardio toxicity can be distinguished by their onset period. Myocardial damage, interstitial edema, and inflammatory cell infiltration are examples of rare acute cardio toxicity caused by ANT therapy. The pathology of this condition is comparable to that of abrupt toxic myocarditis. Dilated cardiomyopathy, on the other hand, is a more common symptom of chronic cardio toxicity caused by ANTs in both human hearts and laboratory models [3].

The pathology is characterized by dilated heart chambers and an increased heart weight. Vacuolar degeneration phosphoinositide 3-kinase gamma/protein kinase B activates mTOR in human tissue and animal models, suppressing Ulk-1 and preventing autophagy from beginning. Discovered that the administration of DOX therapy did not sufficiently trigger autophagy in the heart. Due to the lack of a comprehensive assessment of autophagy flux, it is difficult to pinpoint the specific role of autophagy in DOX-induced cardio toxicity. A problem with autophagosome fusion or improved autophagosome formation may be the cause of elevated LC3-II levels. There are some differences between the clinical symptoms of acute and chronic DOX cardio toxicity, and the amount of the drug is what causes DOX cardio toxicity. Lack of dosage and duration modeling research may have contributed to controversial findings. However, it is intriguing that reversing autophagy at that time can reduce cell death, regardless of whether DOX causes cardio toxicity by activating or suppressing autophagy [4].

Depending on when it first manifests, chronic cardio toxicity is divided into early and late forms. HF is typically the outcome of early-onset chronic cardio toxicity, which typically manifests as dilated and hypokinetic cardiomyopathy. It usually appears within a year of stopping therapy. Late-onset persistent cardio toxicity may appear years or even decades after chemotherapy has ended. The latter results in the production of H2O2, which can be transformed into a variety of ROS-related compounds that, in the end, trigger a DNA damage response and cause the death of numerous cardiomyocytes. Using antioxidants like N-acetyl cysteine, vitamin E, and coenzyme can reduce the cardio toxicity of ANTs, according to numerous studies. However, in a number of animal tests, the expected outcome was not achieved by using antioxidants consistently. So, it's not clear how much of an impact ANT's cardio toxicity has on oxidative stress and the production of primitive ROS. Another intriguing aspect of ANT-induced cardio toxicity is the fact that ANTs prefer the mitochondria in cardiomyocytes. ROS production and mitochondrial dysfunction are linked, according to studies. Cardiolipins, which are abundant on the inner mitochondrial membrane [5].

Conclusion

In addition to mitophagy, a process called "macroautophagic," in which DOX affects autophagy to cause cardio toxicity, is investigated later. Autophagy's role in the cardio toxicity caused by antitumor therapy has been questioned. Autophagy is a bulk breakdown pathway that is dependent on the lysosome and is necessary for maintaining cellular homeostasis. Autophagy begins with the activation of the adenosine protein kinase pathway and the inhibition of the mammalian target of kanamycin signaling pathway. However, autophagy inhibition-induced ANT-induced cardio toxicity has been the focus of other studies that DOX stimulates the signaling pathway that follows Toll-like receptor. This study discusses new developments in reverse cardio-oncology, the use of antineoplastic and cardio protective medications in combination, and the processes by which cancer therapy causes cardiovascular toxicity. Chemotherapy, radiation therapy, and surgery are the main treatments for cancer. Radiotherapy can be used to treat cancers of the breast, thyroid, prostate, and head and neck. Ionizing molecules in radioactively damaged tissue, preventing DNA replication, and eliminating tumor cells are the primary ways that radiotherapy damages DNA.

Acknowledgement

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Conflict of Interest

None.

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