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Analyzing Oncomedicine Combinations and Mechanisms in Brief

Martine Sophia*

Department of Medical Oncology, Zhejiang University, Hangzhou, P.R. China

Abstract

Chemotherapy, radiation, targeted therapy, and immunotherapy have given cancer patients new hope. As cancer patient survival rates and clinical experience have expanded, interest in cancer therapy-induced cardiovascular disease has grown. To create effective preventative and treatment measures for the side effects of cancer treatments that can be deadly or result in long-term morbidity, a deeper understanding of the molecular biology underlying the disease is required. In addition to the typical cardio-protection drugs, traditional Chinese medicine formulations are efficient and can be expected to achieve "tailored treatment" from a range of perspectives. Also highlighting the urgent need for collaboration between cardiologists and oncologists is the emergence of "reverse cardio-oncology" as a result of the increased incidence of cancer in persons with cardiovascular disease.

Keywords: Cardiovascular disease • Radiation treatment • Antioxidants • Cardiomyocytes • Cardiovascular 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. Cardiotoxicity has also overtaken the tumor as the second leading cause of long-term mortality, according to a cohort study of pediatric cancer survivors. In 1976, the first report of cardiotoxicity in juvenile patients receiving DOX was made. Since then, additional initiatives have been launched to separate the cardiotoxic and anticancer effects of ANTs and further reduce toxicity. ANT's cardiotoxicity 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.

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. Evaluation and treatment of cardiotoxicity caused by anticancer medication or the malignant process it causes is the foundation of cardio-oncology. 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.

Researchers have focused on preventing and reducing cardiotoxicity associated with cancer treatment in order to accomplish this objective. Efforts to combine cardiovascular and anti-cancer medications while simultaneously achieving cardiac cleansing have contributed to the development of "tumour 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 cardiotoxicity.

Literature Review

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 cardiotoxicity is the leading cause of morbidity and mortality in cancer survivors [1]. The use of anti-nausea drugs during chemotherapy has been linked to known cardiotoxicity. On the other hand, relatively new medications like immunomodulation and target therapy have not been linked to many reports of cardiotoxicity. The method that ANTs use to fight cancer involves damaging DNA. They mostly affect cells that are growing in the S and G2 stages [2]. The parent nucleus of the anthracene 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 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 [3].

ANTs also have a direct impact on topoisomerase type 2, DNA unwinding, helicase activity, and the subsequent strand separation of DNA. Acute and chronic cardiotoxicity can be distinguished by the time of onset. Myocardial damage, interstitial edoema, and inflammatory cell infiltration are examples of rare acute cardiotoxicity caused by ANT therapy [4]. The pathology of

***Address for Correspondence:** Martine Sophia, Department of Medical Oncology, Zhejiang University, Hangzhou, P.R. China, E-mail: sophialmertina@gmail.com*

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this condition is comparable to that of abrupt toxic myocarditis. Dilated cardiomyopathy, on the other hand, is a more common symptom of chronic cardiotoxicity caused by ANTs in both human hearts and laboratory models. The pathology is characterized by dilated heart chambers and an increased heart weight. Vacuolar degeneration and sarcoplasmic reticulum swelling and consolidation are the most common patterns in both human tissue and animal models [5].

Discussion

Depending on when it first manifests, chronic cardiotoxicity is divided into early and late forms. HF is typically the outcome of early-onset chronic cardiotoxicity, which typically manifests as dilated and hypokinetic cardiomyopathy. It usually appears within a year of stopping therapy. Late-onset persistent cardiotoxicity 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 cardiotoxicity has on oxidative stress and the production of primitive ROS. Another intriguing aspect of ANT-induced cardiotoxicity is the fact that ANTs prefer the mitochondria in cardiomyocytes. ROS generation and mitochondrial dysfunction have been linked, according to studies. Cardiolipins, which are abundant on the inner mitochondrial membrane, are able to directly bind to DOX, which can then prevent the formation of ROS, obstruct the electron transport chain, and block complexes I and II. 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.

DOX hinders mitophagy by preventing Sestrin2 (SESN2) from interacting with Parkin and p62. Additionally, the DOX-induced regulation of mitochondrial autophagy may be linked to cytoplasmic p53, which binds to Parkin and prevents it from translocating to mitochondria to inhibit phagocytosis. Activation of the PINK1/Parkin pathway, which encourages PINK1/Parkin translocation to mitochondria, has been suggested by other studies to be the cause of cardiotoxicity and the signs of it. Reduced mitophagy also prevents cardiomyocyte cell death and reduces mitochondrial dysfunction. In addition to mitophagy, a process called "macroautophagic," in which DOX affects autophagy to cause cardiotoxicity, is investigated later. Autophagy's role in the cardiotoxicity caused by antitumor therapy has been questioned. Autophagy, a bulk breakdown pathway dependent on the lysosome, is necessary for maintaining cellular homeostasis. Autophagy begins with the activation of the adenosine 5'-monophosphate-activated protein kinase pathway and the inhibition of the mammalian target of rapamycin signaling pathway. However, autophagy inhibition-induced ANT-induced cardiotoxicity has been the focus of other studies. that DOX stimulates the phosphoinositide 3-kinase gamma/protein kinase B downstream signaling pathway of Toll-like receptor 9, activating mTOR, which in turn suppresses Ulk-1 and prevents autophagy from beginning. discovered that the administration of DOX therapy did not sufficiently trigger autophagy in the heart.

It is challenging to pinpoint the specific involvement of autophagy in DOX-induced cardiotoxicity, and those investigations lacked a thorough evaluation of autophagic flux. Increased LC3-II levels might indicate a problem with autophagosome fusion or improved autophagosome formation. There are some distinctions between the clinical symptoms of acute cardiotoxicity and chronic cardiotoxicity brought on by DOX, and the cardiotoxicity of DOX is amount of the drug. Controversial findings could potentially be a result of research on dosage and modelling duration being lacking. However, regardless of whether DOX causes cardio toxicity by activating or suppressing autophagy, it is highly intriguing that reversing autophagy at that moment can reduce cell death.

Conclusion

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, radiotherapy, and surgery are the primary 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.

Conflicts of Interest

None.

References

1. Totzeck, Matthias, Martin Schuler, Martin Stuschke and Gerd Heusch, et al. “Cardio-oncology-strategies for management of cancer-therapy related cardiovascular disease.” *Int J Cardiol* 280 (2019): 163-175.
2. Berger, James M., Steven J. Gamblin, Stephen C. Harrison and James C. Wang. “Structure and mechanism of DNA topoisomerase II.” *Nature* 379 (1996): 225-232.
3. Zhang, Ying-Yu, Chen Meng, Xin-Mu Zhang and Cai-Hua Yuan, et al. “Ophiopogonin d attenuates doxorubicin-induced autophagic cell death by relieving mitochondrial damage *in vitro* and *in vivo*.” *J Pharmacol Exp Ther* 352 (2015): 166-174.
4. Kong, Chun-Yan, Zhen Guo, Peng Song and Xin Zhang, et al. “Underlying the mechanisms of doxorubicin-induced acute cardiotoxicity: Oxidative stress and cell death.” *Int J Biol Sci* 2022 18 (2022): 760.
5. Montaigne, David, Xavier Marechal, Riadh Baccouch and Thomas Modine, et al. “Stabilization of mitochondrial membrane potential prevents doxorubicin-induced cardiotoxicity in isolated rat heart.” *Toxicol Appl Pharmacol* 244 (2010): 300-307

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