Cardio toxicity advances in pharmacogenomics and personalized medicine.

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Introduction

These agents can induce oxidative stress, disrupt cellular membranes, and lead to cardiomyocyte death. Type II cardio toxicity; on the other hand, is characterized by indirect damage to the heart, typically without dose dependence. Targeted therapies, such as tyrosine kinase inhibitors (TKIs), can cause this type of toxicity by interfering with signalling pathways involved in cardiac homeostasis. Several mechanisms contribute to cardio toxicity. Oxidative stress, a state of imbalance between free radicals and antioxidants, plays a crucial role in the development of both Type I and Type II cardio toxicity [1,2]. Oxidative stress can damage cellular components, including lipids, proteins, and DNA, leading to cellular dysfunction and death. Additionally, inflammation and immune responses triggered by drug-induced injury can exacerbate cardio toxic effects. Mitochondrial dysfunction is another key mechanism in cardio toxicity. Mitochondria are vital for energy production in cardiac cells, and impairment of mitochondrial function can lead to cellular energy depletion and compromised cardiac performance. Efforts to prevent and manage cardio toxicity involve various strategies. Cardio protective agents, such as dexrazoxane, have been developed to mitigate the toxic effects of anthracyclines in cancer patients. These agents act by chelating iron and reducing oxidative stress. Close monitoring of cardiac function through regular screenings, including echocardiography and biomarker assessments, is crucial, particularly during and after treatment with potentially cardio toxic drugs. Advancements in cardiac imaging techniques have also aided in early detection and monitoring of cardio toxicity [3]. Cardiac magnetic resonance imaging (MRI) can assess cardiac function, detect subtle changes in tissue structure, and provide valuable information about the extent and severity of cardio toxicity.

Additionally, an increased understanding of genetic predisposition to cardio toxicity has led to the development of pharmacogenomics approaches. Genetic testing can identify patients at higher risk of developing cardio toxicity, enabling personalized treatment plans and dosage adjustments. Research is on-going to identify novel therapeutic targets to prevent or mitigate cardio toxicity. For instance, the use of cardio protective agents, such as beta-blockers and angiotensin-converting enzyme (ACE) inhibitors, has shown promising results in some cases. Furthermore, emerging therapies like stem cell transplantation and gene therapy hold potential in repairing damaged cardiac tissue and improving cardiac function [4,5].

Conclusion

Cardio toxicity remains a significant concern in modern medicine due to its potential impact on patients' cardiovascular health. Understanding the mechanisms of cardio toxicity, implementing preventive strategies, and adopting advanced monitoring techniques are key to reducing its incidence and managing its effects. On-going research and personalized approaches offer hope for better outcomes in the future.

References

1. Piotrowska I. Early transcriptional alteration of histone deacetylases in a murine model of doxorubicin-induced cardiomyopathy. PloS One. 2017;12
2. Elrashidy RA. Cilostazol preconditioning alleviates cyclophosphamide-induced cardiotoxicity in male rats: mechanistic insights into SIRT1 signaling pathway. Life Sci. 2021;266:118822.
3. Yang Y. Sirt3 promotes sensitivity to sunitinib-induced cardiotoxicity via inhibition of GTSP1/JNK/autophagy pathway in vivo and in vitro. Arch Toxicol. 2019;93:3249–60.
4. Li P. Lysine acetyl transferases and lysine deacetylases as targets for cardiovascular disease. Nat Rev. Cardiol. 2020;17:96–115.
5. Hanf A. The anti-cancer drug doxorubicin induces substantial epigenetic changes in cultured cardiomyocytes. Chem Biol Interact. 2019;313:108834.

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