LETTER TO THE EDITOR

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Letter to the Editor: In Response to Cardiac Toxicity of Cancer Therapies: Mechanisms, Surveillance, and Clinical Implications

Sara Khan, Mariyam Siddiqui, Maham Shamim

Karachi Metropolitan University (KMDC) Faculty of Medicine, Karachi, Pakistan

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To the Editor,

We read with great interest the recent article titled "Cardiac Toxicity of Cancer Therapies: Mechanisms, Surveillance, and Clinical Implications", published in the *International Journal of Cardiovascular Academy*.[1] The review presents an insightful overview of the mechanisms and monitoring strategies for therapy-induced cardiotoxicity. However, we highlight two emerging aspects that warrant further discussion.

While the paper provides an exhaustive review of cardiac toxicity mechanisms and surveillance methods, it neglects to mention the emerging role of molecular and genetic predictors in cardio-oncology. According to recent studies, a patient's genetics and ethnicity play a major role in their susceptibility to therapy-induced cardiomyopathy. For example, Black race/African ancestry is associated with 71% higher odds of cardiotoxicity after cancer chemotherapy.[2] Several common genetic variants have been identified as either increasing or reducing the risk of cancer therapy-induced cardiomyopathy. These genetic variants influence drug metabolism, efficacy, and susceptibility to adverse effects.[3]

Secondly, the description of the drug, dexrazoxane in the Management and Prevention section claims its administration as controversial based on issues of decreased efficacy of chemotherapy and secondary malignant neoplasms, especially among children. The latest evidence and global consensus, though, offer a more balanced view. International Late Effects of Childhood Cancer Guideline Harmonization Group concluded that "if cardiac damage risk is high, it could be reasonable to administer dexrazoxane to children and cancer patients with adults exposed to anthracyclines", highlighting an individualized risk-benefit evaluation (de Baat et al.,[4]). It recommends dexrazoxane as a clinically justifiable cardioprotective approach when properly administered in high-risk populations.

In conclusion, we appreciate the authors for their valuable contribution and hope these perspectives may guide future discussion in this evolving field by acknowledging the importance of genetic screening and updated dexrazoxane evidence would further strengthen the article's clinical applicability and comprehensiveness.

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Address for Correspondence: Sara Khan, Karachi Metropolitan University (KMDC) Faculty of Medicine, Karachi, Pakistan
E-mail: khanskk221@gmail.com
ORCID ID: orcid.org/0009-0002-9610-8093

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