OPINION ARTICLE

Anthracycline-Based Cancer Treatment that Induces Cardiotoxicity

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DESCRIPTION

The lifetime risk of cancer for individuals born after the 1960s is now predicted to be greater than 50%, which is much higher than the same estimate for those born in the 1930s (15% rise for males and 10.8% increase for women), in keeping with the ageing and growing population. Cancer survivability rates are increasing; however cardiotoxicity from anticancer therapy is a growing problem that can lead to cardiomyopathy and heart failure. The cost of treating cancer continues to be a significant burden on global healthcare systems. As a result, yesterday's cancer survivors are quickly evolving into today's heart failure patients. Indeed, some cancer patients are more likely to die from cardiovascular disease than from the cancer itself, as evidenced by the findings that CVD is the leading cause of death in female breast cancer patients and that people in remission from childhood cancers are more likely to die from CVD than from a cancer relapse. As a result, cardiotoxicity brought on by chemotherapy is posing new healthcare issues. Anthracyclines continue to be a prominent clinical tool for cancer treatment and are the class of chemotherapeutics against a variety of malignancies despite strong evidence that anthracyclines, including the most studied member of the anthracycline family, doxorubicin, DOX, can cause heart failure.

Up to 37.5% of chemotherapy patients experience ventricular dysfunction as a result of Anthracycline-Induced Cardiotoxicity (AIC). Arrhythmias, increased brain natriuretic peptide and troponin levels can all be acute toxicity symptoms of AIC, however these side effects are often reversible if medication is stopped. AIC can also manifest more often and clinically as late-onset chronic toxicity linked to the irreversible development to heart failure in the years or even decades following the end of medication. In up to 65% of patients treated with anthracyclines for juvenile malignancies and 12% of patients treated with anthracyclines for breast cancer, late-onset AIC manifests as pathological myocardial remodelling characterised by cardiomyocyte hypertrophy and increased fibrosis, which results in functional cardiac deterioration.

Clinical studies have demonstrated a small degree of cardio protective potential for dexrazoxane; however, not all patients respond equally to the drug, especially girls responding to therapy more effectively than men. Dexrazoxane may reduce the antineoplastic effects of anthracycline therapy, which raises further concerns. Ultimately, heart transplantation remains the sole curative option for people with end-stage heart failure brought on by AIC. However, due to a current or prior history of malignancy and worries about cancer recurrence in the context of immunosuppressive medication, individuals with AIC are frequently ineligible for transplant. As a result, many patients are unable to receive even this last-resort medication.

CONCLUSION

The acute subclinical changes to cardiac cellular biology that are asymptomatic and hidden are the root of late-onset cardiotoxicity. Over the course of months and years, these changes are thought to cause responsive pathological myocardial remodelling that eventually leads to symptomatic and fulminant heart failure. Cellular senescence is one potential cell fate that can develop after chemotherapy and contribute to the chronic/progressive character of AIC (and the related higher occurrence in CVD).

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