

Mini review

Oncocardiology: Far beyond the cardiotoxicity

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Approximately 73.4% of global deaths are caused by chronic non-communicable diseases, among them, cardiovascular and cerebrovascular diseases, tumors, and chronic respiratory diseases ranked in the top 3 respectively [1]. An accumulating body of evidence showed that the risk of all-cause mortality in cancer patients with cardiovascular disease (CVD) was 3.78 times higher than that of those without CVD and 8.8% of cancer survivors died of CVD [2]. Heart failure (HF) is a serious manifestation or terminal stage of various heart diseases. Although myocardial damage and dysfunction are the main causes of HF, the cardiovascular injury caused by the tumor itself and the detrimental effect of cancer treatment also play an important role. More recently, the data has suggested that up to 25% - 30% of patients with HF have histories of cancer for about 10 years; and cancer also determines the prognosis of heart HF [3].

Oncocardiology, an interdisciplinary subject, emerged as the times require. Oncocardiology is originally defined as cardiotoxicity related to cancer therapy and is now a term coined to describe a medical subspecialty that focuses on the identification, prevention, and treatment of cardiovascular complications during the treatment of cancer [4]. On August 26, 2022, the annual meeting of the European Society of Cardiology (ESC 2022) issued the first guidelines for oncocardiology [5]. In these guidelines, the basic concept, discipline orientation, development direction of echocardiology, and various types of cancer therapy-related cardiovascular toxicity (CTR-CVT) were reviewed in detail.

More recently, several lines of data suggest that CVD and tumors may have a synergistic effect. The evidence for the hypotheses included that both CVD and tumor have some common risk factors, including genetic susceptibility, obesity, smoking, hyperlipidemia, diabetes, sedentariness, and old age; cardiovascular risk factors are up-regulated in cancer survivors; progressive cellular senescence is a common denominator that could increase CVD risk in cancer, and progressive telomere shortening has been associated with the development of CVD.

More Information

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CVD in cancer patients can precede the diagnosis of cancer or can be related to the malignancy itself or its therapy. Many cardiovascular phenotypes have been identified as common across different cancer treatments. Lipshultz, et al. [6] found that survivors of childhood cancer, regardless of exposure to cardiotoxic treatments, had cardiovascular abnormalities related not only to abnormal left ventricular structure and function but also to increased traditional risk factors for atherosclerotic disease and systemic inflammation. Most recently, the relationship between atrial fibrillation (AF) and Stroke/ transient ischemic attacks (TIA) in cancer patients was conducted in a case-control study, the CHA₂DS₂-VASc score, which components such as heart failure, hypertension, old age, and diabetes mellitus were found to be associated with stroke in the general population, significantly increases the risk of stroke in cancer patients regardless of the presence of AF [7].

The potential role of relatively short-term insults given by cancer treatments in significant vascular effects and increasing CVD events is currently under investigation. Several studies demonstrated that telomere shortening and dysfunction had a significant impact on CVD. Accelerated telomere shortening, particularly in endothelial cells, was observed in atherosclerotic plaques and areas exposed to disturbed flow. In addition, DNA damage and apoptosis, excess inflammation, mitochondrial dysfunction, and reactive oxygen species (ROS) production were found to be associated with the risk of CVD [8,9].



Recent advances in oncology have contributed to the improvement of the prognoses of cancer patients. However, adverse effects on the cardiovascular system can cause a significant deterioration in patients. The current research is limited to the different mechanisms of cardiovascular injury related to cancer therapy. There is still a lack of reliable evidence on the control of common risk factors of tumor and cardiovascular disease, the new biomarkers of the tumor with CVD and the exploration of new anti-tumor therapy with higher cardiovascular safety. Thus, large-scale, multicenter randomized controlled clinical trials are needed. These are exciting challenges and opportunities that will test the ingenuity and persistence of oncocardiologists.

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