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Impact of cancer in cardiovascular risk

Cáncer y su impacto en el riesgo cardiovascular

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INTRODUCTION

Cardiovascular diseases (CVD) and cancer are the leading causes of mortality worldwide. Cancer survivors experience a higher risk of CVD morbidity and mortality than the general population.[1] Cardiovascular risk factors are associated with more significant cardiotoxicity (CTOX) and lower long-term survival.

Epidemiological studies have reported that adherence to heart-healthy lifestyle habits could prevent the development of cancer and CVD, improving the survival of the general population.[1]

Determining cardiovascular risk (CVR) and early detection of myocardial damage from the therapies used are the cornerstone of CVD prognosis in cancer patients.[2]

Mechanisms of cardiotoxicity. Myocardial injury.

Cardiotoxicity includes the development of ventricular dysfunction, myocardial ischemia, arterial hypertension, arrhythmias, pulmonary thromboembolism, pulmonary hypertension, pericardial complications, peripheral vascular disease, and stroke, among others.

The myocardial injury occurs through 2 types of mechanisms

Type 1 cardiotoxicity is dose-dependent and irreversible; the damage occurs on the enzyme Topoisomerase II, with the generation of free radicals, the classic example being the anthracyclines.

Type 2 cardiotoxicity, generally reversible, is caused by the blockage of cell repair pathways that occur by inhibiting the HER2 receptor, ultimately leading to an acceleration of the myocyte death process and decreased functional recovery, mainly caused by trastuzumab.[3,4]

Tyrosine kinase inhibitors produce cardiotoxicity through other mechanisms characterized by mitochondrial dysfunction,[3] loss of membrane potential, cytochrome C release, and decreased ATP levels, ultimately leading to myocyte death.[5]

Radiation-induced CVD is characterized by endothelial dysfunction, considered a risk factor in the pathogenesis of accelerated atherosclerosis and heart failure, usually associated with preserved ejection fraction. For each dose of radiation measured in gray (Gy), the probability of the appearance of a significant CVD increases by 7.4%.

Chemotherapy cardiotoxicity: How to diagnose it? Prevention

Cardiotoxicity is defined as alterations at the level of the heart, vessels, and conduction system derived from antitumor treatment (chemotherapy and or radiotherapy); according to the European Society of Cardiology (ESC), ventricular dysfunction is the decrease in the fraction of left ventricular ejection (LVEF) > 10% compared to baseline LVEF, with the average cut-off point being 50%. The Spanish Image Society (ASE) and The Spanish Association of Cardiac

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Imaging (AEIC) suggest an average LVEF cut-off point of 53%.

The objective of monitoring patients is to make an early diagnosis of myocardial damage considering the following studies:

Biomarkers

1. Ultrasensitive troponin I baseline quantification is recommended, and after each cycle of treatment, it has a high negative predictive value; persistent elevation suggests a worse prognosis. The use of NT proBNP is not yet well defined.[6]

Multimodal image

1. The transthoracic echocardiogram is the most common method used for patients with antitumor treatment, it suggests serial evaluation of the LVEF, and in 2D, the biplanar method is preferred (intra and interobserver variability is 7.4%), whenever possible, the 3D (intra and interobserver variability Interobserver is 4%)[6,7] is preferred. LVEF is not considered a marker of myocardial function for subclinical diagnosis since it is altered once DV is established, which is generally irreversible.[3] The Global Longitudinal Strain (GLS) of the LV is the best predictor of cardiotoxicity, mainly with the use of anthracyclines; it is a robust measure for the subclinical diagnosis of DV,[8] a decrease between 10 and 15% is associated with symptomatic and asymptomatic CTOX. In some studies, GLS < 19% at the end of treatment is associated with the late development of CTOX.[7,8] Radial and circumferential strain have not been associated with the diagnosis of CTOX, and reproducibility is not as robust as GLS.[8,9] Studies show that for every 1% reduction in initial GLS, it is associated with a 1.48% chance for the development of CTOX.
2. The American College of Cardiology (ACC) recognizes cardiac magnetic resonance Imaging as a powerful imaging study for the detection of CTOX; its use is not recommended routinely unless discontinuation of CTOX treatment is considered and verification of LVEF is required. CT angiography is not used as a first-line image to assess LVEF.

Post-radiotherapy cardiovascular damage

Radiation therapy affects cardiac structures primarily when the heart is at the radiation site. Risk factors for post-radiotherapy heart damage: high doses of radiation (more than 30-35 Gy), adjuvant treatment with chemotherapy (mainly anthracyclines), irradiation in the left hemithorax, atherosclerotic risk factors (smoking, high blood pressure, hyperlipidemia and diabetes), cardiovascular disease (CVD) pre-existing.[10]

Radiation can cause pericardial, coronary artery, noncoronary atherosclerotic disease, myocarditis, cardiomyopathy, valvular heart disease, arrhythmias, and conduction disturbances, months to more than 20 years after radiation therapy.

There are no universally accepted clinical guidelines for post-radiotherapy damage stratification. Follow-up should begin five years after radiotherapy in high-risk patients and ten years in the rest, with subsequent evaluation every five years.

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