

INFLAMMATION AS A COMMON PATHWAY IN CANCER AND CARDIOVASCULAR DISEASE

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RESUMO

Introduction: Inflammation has emerged as a direct link between cancer and cardiovascular disease. Studies show that both the tumor and its treatments trigger an immune response, endothelial dysfunction, and thrombotic tendencies. Inflammatory biomarkers, metabolic alterations, and myocardial damage reinforce the idea that this systemic inflammatory response directly links these two conditions, being mediated by pro-inflammatory cytokines such as Interleukin-6, Tumor Necrosis Factor Alpha, and C-reactive Protein, which promote chronic inflammation, endothelial injury, and the progression of atherosclerosis. **Objective:** Thus, this study investigates inflammation as a common biological pathway between cancer and cardiovascular diseases, studying the role of biological markers, immunological and metabolic alterations, and the effects of cancer treatments in the development of endothelial damage, atherosclerosis, and cardiotoxicity. The aim is to understand its mechanisms and impact on cardiovascular risk, including the activation of shared inflammatory and metabolic pathways such as NF- κ B, oxidative stress, and alterations in aerobic glycolysis and lipid metabolism, which favor insulin resistance, dyslipidemia, and a pro-atherogenic environment. **Methods:** A literature review was conducted using the PubMed database, including studies published in the last 5 years, with the descriptors: "Inflammation," "cancer," and "cardiovascular risk / cardio-oncology," selecting 9 articles for this study. **Results:** Cancer patients present increased cardiovascular risk driven by a complex network of inflammatory and metabolic alterations. At the molecular level, tumor-derived factors and cancer therapies promote activation of pattern recognition receptors, particularly toll-like receptors (TLRs), leading to downstream signaling via MyD88 and subsequent activation of nuclear factor-kappa B (NF- κ B) and signal transducer and activator of transcription 3 (STAT3). This results in sustained production of pro-inflammatory cytokines such as IL-6, TNF- α , and IL-1 β , reinforcing a chronic inflammatory state. Additionally, activation of the NLRP3 inflammasome contributes to IL-1 β and IL-18 maturation, further amplifying vascular inflammation. Oxidative stress plays a central role, with increased generation of reactive oxygen species (ROS) promoting mitochondrial dysfunction, lipid peroxidation, and endothelial damage. Redox-sensitive pathways, including MAPK (p38, JNK) and NF- κ B, are also activated, enhancing inflammatory gene expression and cellular proliferation. Cancer-related metabolic reprogramming, such as enhanced aerobic glycolysis (Warburg effect), contributes to lactate accumulation, which modulates immune cell function and sustains a pro-inflammatory microenvironment. Furthermore, endothelial dysfunction is exacerbated by reduced nitric oxide (NO) bioavailability and increased expression of adhesion molecules (VCAM-1, ICAM-1), facilitating leukocyte recruitment and atherogenesis. Prothrombotic pathways are also activated through tissue factor expression and platelet activation. Cancer therapies, particularly chemotherapy and radiotherapy, intensify these processes by inducing endothelial injury, mitochondrial damage, and persistent inflammatory signaling, ultimately contributing to myocardial dysfunction and accelerated atherosclerosis. **Conclusion:** Inflammation is the main pathway linking cancer and cardiovascular disease, involving inflammatory mediators and shared metabolic pathways that amplify vascular and cardiac damage. The tumor and its treatments increase cardiac risk. Biomarkers and early monitoring are essential for prevention and appropriate management.

Palavras-chave: Systemic Inflammation; Cardiovascular Diseases; Cancer.