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Editorial

Cardio-Oncology: At the Nexus of Two 21st Century Epidemics

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1. Introduction

A striking epidemiological shift has emerged in cancer survivorship, well-illustrated in breast cancer patients. For many undergoing contemporary treatments, the probability of dying from cardiovascular disease now rivals—if not exceeds—the risk of death from the original malignancy [1]. Cardiotoxicity is not a new discovery—anthracycline-related cardiac injury has been recognised for decades—but its consequences have now been reframed. As success against cancer has created a second front—that of protecting the heart—cardio-oncology has emerged and needs to be embedded within standard practice.

The context is that more than 385,000 new cancer diagnoses occur annually in the United Kingdom, with survival roughly doubled over the past half-century [2]. As more people live longer with and beyond cancer, the burden of therapy-related cardiovascular disease (CVD) grows. Cardio-oncology's purpose is straightforward: enable patients to receive optimal anti-cancer therapy safely, minimise unnecessary interruptions and mitigate cardiovascular toxicity throughout the treatment journey [1,3].

This editorial outlines the shared biology, linking cancer and CVD, by summarising major cardiotoxic phenotypes across contemporary cancer therapies and proposes pragmatic strategies—clinical, organisational and public health—to make cardioprotection the default in modern cancer care.

2. Shared Risks, Shared Biology

Cancer and cardiovascular disease (CVD) share a common soil of age, smoking, adiposity, and metabolic dysfunction. Lifestyle factors including poor diet and lack of exercise fuel the underlying pathogenesis of both diseases, as does compliance with medication in general. Genetic predisposition accounts for a meaningful subset of risk in both domains. Cancer and its treatments can accelerate cardiometabolic derangements: fatigue and deconditioning foster inactivity; corticosteroids and other adjuvants drive hypertension, dyslipidaemia and insulin resistance. Neoplasia-driven systemic inflammation with hypercoagulability also amplifies thrombotic risk [3].

The thrombotic diathesis is particularly salient. Cancer confers around a five-fold increased risk of venous thromboembolism (VTE) and a two-fold increase in arterial events such as stroke and myocardial infarction [3]. These realities demand that survivorship care is designed not as a postscript but as an integral, longitudinal pathway encompassing prevention, surveillance and timely intervention.

3. Cardiotoxicity Across Cancer Therapies

3.1 Anthracyclines and Classical Cytotoxics

Anthracyclines remain foundational for many solid-organ and haematological malignancies. Their cardiotoxicity is cumulative and dose-dependent; risks rise notably beyond doxorubicin-equivalent exposures of ~250 mg/m^2. High-dose cyclophosphamide may provoke acute heart failure and arrhythmias. Fluoropyrimidines (5-fluorouracil, capecitabine) can precipitate coronary vasospasm and myocardial ischaemia, often heralded by electrocardiogram (ECG) changes before symptoms [3].

3.2 HER2-Targeted Agents

Trastuzumab has transformed outcomes in human epidermal growth factor receptor 2 (HER2)-positive breast cancer but can induce (typically reversible) left ventricular (LV)-dysfunction [4]. These effects are usually manageable with early detection and judicious interruption or dose adjustment, provided there is a structured surveillance plan.

3.3 Radiotherapy

Chest and mediastinal radiotherapy can injure the pericardium, myocardium, valves and coronaries; late effects may manifest years after exposure. There is no established "safe" cardiac dose, reinforcing the principle that the heart is an organ at risk and that exposure should be kept as low as reasonably achievable [3]. Modern techniques—deep-inspiration breath-hold, intensity modulation, and proton therapy—reduce dose to the left anterior descending artery and other critical structures, though mean heart dose alone is an imperfect surrogate for focal coronary injury [5].

3.4 Targeted and Hormonal Therapies

Vascular endothelial growth factor (VEGF) inhibitors and tyrosine kinase inhibitors (TKIs) disrupt angiogenic and repair pathways, but these are shared by tumours and the cardiovascular system, leading to hypertension, en-

dothelial dysfunction and increased thromboembolic risk [3].

Hormonal therapies (aromatase inhibitors in breast cancer and androgen deprivation therapy in prostate cancer) exacerbate adverse lipid and glucose profiles. The oral gonadotrophin releasing hormone (GnRH) antagonist relugolix achieves rapid testosterone suppression and quicker recovery on cessation compared to leuprolide in men with advanced prostate cancer. This may partly explain reported lower rates of major cardiovascular events in some cohorts treated with relugolix [6]. The use of cyclin-dependent-kinase (CDK)-4/6 inhibitors, notably ribociclib, alongside endocrine therapy in women with hormone-receptor positive/HER2-negative metastatic breast cancer has shown increased predilection to QT-interval prolongation [7], making routine ECG monitoring around treatment essential.

3.5 Immunotherapy and Cellular Therapies

Immune checkpoint inhibitors (ICI) have greatly advanced oncology, but their related adverse events include myocarditis, arrhythmias and fulminant LV failure, albeit infrequently [8]. Late cardiac effects from ICI include progressive heart failure and potential pro-atherogenic effects [9]. Chimeric antigen receptor T-cell (CAR-T) therapy can trigger cytokine release syndrome (CRS) with resultant hypotension, tachyarrhythmias and transient LV dysfunction; cardiopulmonary mortality has approached 30% in some early series, though absolute event rates vary with indication, construct and supportive care [10]. Tocilizumab (anti-interleukin-6 receptor) remains a central countermeasure within established CRS protocols and may attenuate cardiovascular complications [11].

3.6 From Screening to Surveillance: Making Risk Visible

Cardiotoxicity risk is dynamic. It changes with cumulative exposure, intercurrent illness and evolving comorbidity. Consequently, a "baseline-only" approach is insufficient. The 2022 European Society of Cardiology (ESC) cardio-oncology guidelines advocate universal pre-treatment risk assessment and tailored surveillance using a combination of clinical review, biomarkers (troponin, natriuretic peptides), 12-lead ECG, and echocardiography with global longitudinal strain [3].

The Heart Failure Association–International Cardio-Oncology Society (HFA-ICOS) risk tool integrates comorbidities, prior exposures (e.g., anthracyclines, chest radiotherapy), and baseline investigations to stratify patients and set the cadence and modality of follow-up [12]. Cardiac magnetic resonance imaging (MRI) has a pivotal role in selected patients, particularly when echocardiography is suboptimal or myocarditis is suspected, offering tissue characterisation that can change management [13].

New or worsening cardiac symptoms, a $>10\%$ absolute fall in left ventricular ejection fraction (LVEF), or a $>15\%$ relative reduction in global longitudinal score should prompt urgent review, reconsideration of the cancer regimen and optimisation of cardioprotective therapy. Crucially, such decisions should be made in a cardio-oncology multidisciplinary team (MDT), balancing oncological efficacy with cardiovascular safety.

4. Cardioprotection: A Necessity in Routine Care

4.1 Aggressive Risk Factor Management

Cardioprotection should begin by addressing modifiable risk factors. Lifestyle intervention - smoking cessation, exercise, and dietary optimisation—can be framed as treatment, not advice. Hypertension, diabetes, and dyslipidaemia are to be treated to contemporary standards before and during therapy. In this context, as in others (such as pre-surgery), services increasingly include "cardiac prehabilitation" to improve functional reserve before cardiotoxic regimens begin.

4.2 Evidence-Based Pharmacology

To date, randomised controlled trials (RCTs) have, perhaps surprisingly, failed to identify any single agent—whether beta blockers, angiotensin-converting enzyme (ACE) inhibitors, statins or sacubitril/valsartan—to be of effective or relevant prospective cardioprotective benefit in the cardio-oncological context. Sodium-glucose cotransporter 2 inhibitors may offer hope on the horizon.

However, for the management of established disease, treatment is clearer. Statins form the backbone of lipid-profile optimisation. Beta-blockers and ACE-inhibitors/angiotensin-receptor blockers (ARBs) mitigate decline in LV-function during exposure to anthracyclines or trastuzumab and are supported by guideline-level recommendations [3]. Mineralocorticoid receptor antagonists can reduce fibrosis and fluid retention in selected patients.

4.3 Modifying Exposure and Ensuring Follow-up

When oncologically acceptable, it may be necessary to reduce cumulative anthracycline dose or use liposomal formulations. Meta-analysis supports superior cardiac safety of liposomal doxorubicin without compromising efficacy, particularly in older adults [14]. Dexrazoxane remains the only licensed pharmacological cardioprotectant against anthracycline cardiotoxicity in high-risk patients, with evidence of preserved LVEF and reduced clinical heart failure [15]. Such strategies should be standard topics in early treatment discussion within MDTs rather than reserved as salvage. Regular follow-up of patients after completion of cardiotoxic treatments ensures sustained morbidity and mortality benefit.

4.4 Service Design to Reduce Risk

The organisation of cardio-oncology services varies within healthcare systems but certain features in common

include the need for agreed pathways, triggers/red flags for early or emergency review, shared electronic prompts, rapid-access clinics for symptoms or rises in biomarkers; and, clear shared discussions and MDTs with handovers between oncology, cardiology and primary care teams. Hospitals that operationalise these basic systems of operation tend to see fewer unplanned interruptions of cancer therapy and better patient experience [1,3].

4.5 Policy and Public Health: Aligning Incentives With Outcomes

In addition to sharing treatment between healthcare professionals, the dual epidemics of cancer and CVD demand joined-up public health action: smoke-free policies, active-travel infrastructure, better food availability choices and equitable access to preventive care. Such measures deliver compound benefits with respect to cancer-related CVD incidence, treatment tolerance and survivorship.

At a systems level, healthcare providers and commissioners should establish cardio-oncology pathways for treatment regimens with known cardiotoxic risk. Automated prompts can be embedded for baseline risk-scoring and measurement of crucial parameters. This will enable treatment completion without cardiovascular interruption, preservation of LVEF, avoidance of unplanned admissions, thus improving patient-reported outcomes. National and international cardio-oncology societies have a crucial role to play in the education of clinicians and patients, to produce high-quality, evidence-based guidelines.

5. Conclusion

Cancer survivorship is one of modern medicine's defining successes. But it has brought to light an uncomfortable fact—without deliberate cardioprotection, some of our most effective anti-cancer therapies erode the very survival they secure. Cardio-oncology provides an antidote—a disciplined blend of prevention, surveillance and multidisciplinary care that preserves oncological intent whilst safeguarding the heart.

Key Points

• Cardio-oncology is a critically emerging field given the rapid improvements in cancer treatments and cancer survivorship in an already aging population.

• The aim of cardio-oncology is to safely allow patients to receive appropriate and optimal anti-cancer therapy whilst simultaneously mitigating cardiac toxicity throughout the patient journey.

• Cancer and cardiovascular disease share common risk factors which must be addressed in synchrony, from screening to treatment and onward surveillance, if we are to ensure optimal patient experience and patient outcomes.

• Treatments including anthracyclines, human epidermal growth factor receptor 2 (HER2)-targeted agents, endocrine therapies, radiotherapy and immunotherapy all play a significant role in observed cardiotoxic phenotypes.

• Standard organisational practice in delivering cancer treatment must embed cardioprotection, including aggressive risk factor management, surveillance, tailored investigations using a combination of clinical assessment, biomarkers and imaging.

• Ultimately, successful cardio-oncology requires multidisciplinary teams and systems-level changes in healthcare design and public health policy to safeguard the heart in cancer patients.

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Conflict of Interest

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