
Postgraduate Certificate in Cardio-Oncology (Belgium)

Cardiotoxicity of Cancer Therapies

Cardiotoxicity refers to the deleterious effects of cancer therapies on the structure and function of the heart. The term encompasses a spectrum that ranges from transient, subclinical changes in myocardial contractility to overt heart failure, arrhythmias, hypertension, and coronary artery disease. In the context of a postgraduate certificate in cardio-oncology, a precise understanding of the terminology is essential for accurate assessment, timely intervention, and effective communication within multidisciplinary teams.

Anthracyclines are a class of cytotoxic agents that intercalate DNA and generate free radicals. Examples include doxorubicin, epirubicin, and idarubicin. Their dose-dependent cardiotoxicity is one of the most extensively studied phenomena. For instance, a cumulative doxorubicin dose exceeding 300 mg/m² markedly increases the risk of symptomatic heart failure. Clinicians must therefore balance oncologic efficacy with cardiac risk, often employing dose-reduction strategies or cardioprotective agents such as dexrazoxane.

HER2-targeted therapy includes monoclonal antibodies (trastuzumab, pertuzumab) and antibody-drug conjugates that inhibit the human epidermal growth factor receptor 2 pathway. While these agents revolutionized the treatment of HER2-positive breast cancer, they also pose a distinct, often reversible, cardiotoxic risk. Unlike anthracyclines, the injury is frequently non-dose-dependent and may manifest as a decline in left ventricular ejection fraction (LVEF) during or shortly after therapy. Close monitoring with echocardiography and biomarkers is therefore recommended.

Tyrosine kinase inhibitors (TKIs) such as sunitinib, sorafenib, and imatinib target intracellular signaling cascades involved in tumor proliferation. Their cardiovascular side-effects include hypertension, left ventricular dysfunction, and, less commonly, myocardial ischemia. The mechanisms are multifactorial, involving endothelial dysfunction, reduced nitric oxide bioavailability, and off-target inhibition of kinases that regulate vascular tone. Practical application requires baseline blood pressure assessment and periodic monitoring throughout treatment.

Immune checkpoint inhibitors (ICIs) – agents that block CTLA-4, PD-1, or PD-L1 pathways – have transformed the management of melanoma, lung cancer, and several other malignancies. However, they can precipitate immune-mediated myocarditis, a potentially fatal condition that often presents with chest pain, arrhythmias, or rapid decline in LVEF. Early recognition hinges on a high index of suspicion, serial troponin measurement, and cardiac magnetic resonance imaging when available. Prompt initiation of high-dose corticosteroids is the cornerstone of therapy.

Radiation-induced heart disease (RIHD) encompasses a variety of cardiac pathologies that arise after exposure of the thorax to ionizing radiation. Fibrosis of the pericardium, coronary artery atherosclerosis, valvular thickening, and restrictive cardiomyopathy may emerge years after treatment. The latency period underscores the need for lifelong cardiovascular surveillance in survivors of mediastinal radiotherapy. Modern techniques such as intensity-modulated radiation therapy aim to limit cardiac dose, thereby

reducing long-term risk.

Left ventricular ejection fraction (LVEF) is the proportion of blood ejected from the left ventricle with each systolic contraction, typically expressed as a percentage. An LVEF below 50% is generally considered abnormal, though specific thresholds may vary by imaging modality. LVEF remains the primary quantitative measure for detecting chemotherapy-related systolic dysfunction, but its sensitivity for early, subclinical changes is limited. Consequently, many guidelines now incorporate more sensitive parameters such as strain imaging.

Global longitudinal strain (GLS) quantifies the percentage of myocardial shortening along the long axis of the left ventricle. A reduction of > 15% from baseline is often regarded as indicative of subclinical cardiotoxicity, even when LVEF remains within normal limits. GLS can be measured using speckle-tracking echocardiography and provides a valuable early warning sign that may prompt cardioprotective interventions before irreversible damage occurs.

Cardiac biomarkers play a pivotal role in the early detection of myocardial injury. Cardiac troponin I or T, when elevated above the 99th percentile, signals myocyte necrosis. Natriuretic peptides – B-type natriuretic peptide (BNP) and its N-terminal pro-hormone (NT-proBNP) – reflect ventricular wall stress and are useful for monitoring heart failure progression. In cardio-oncology, serial measurement of troponin and natriuretic peptides before, during, and after chemotherapy assists in risk stratification and guides therapeutic decisions.

Acute cardiotoxicity describes cardiac dysfunction that emerges within days to weeks of exposure to a cardiotoxic agent. It may manifest as arrhythmias, myocarditis, or sudden drops in LVEF. For example, high-dose cyclophosphamide can cause acute hemorrhagic myocarditis, presenting with chest pain and elevated troponin. Management typically involves immediate cessation of the offending agent, supportive care, and, in some cases, immunosuppression.

Chronic cardiotoxicity refers to delayed cardiac effects that develop months to years after therapy completion. This form includes progressive heart failure, coronary artery disease, and valvular pathology. Chronic cardiotoxicity is particularly relevant for survivors of childhood cancers, who may experience cumulative cardiovascular burden decades later. Long-term surveillance strategies, including periodic echocardiography and risk factor modification, are essential components of survivorship care.

Dose-dependent cardiotoxicity is directly proportional to the cumulative exposure of a drug. Anthracycline-related heart failure exemplifies this relationship, with a clear threshold beyond which the probability of clinical dysfunction rises sharply. Conversely, dose-independent cardiotoxicity, such as that associated with trastuzumab, does not correlate with cumulative dose but rather with concurrent or prior exposure to other cardiotoxic agents, underlying cardiac reserve, and individual susceptibility.

Subclinical cardiotoxicity denotes myocardial injury that is detectable by sensitive imaging or biomarker techniques but has not yet produced overt clinical symptoms or a decline in LVEF. Identification of subclinical changes enables clinicians to intervene early, often with cardioprotective medications, thereby preventing progression to symptomatic heart failure. The concept underscores the shift from a reactive to a

proactive approach in cardio-oncology.

Cardioprotective agents are pharmacologic interventions that mitigate the cardiac side-effects of cancer therapies. Dexrazoxane chelates iron and reduces anthracycline-mediated free-radical formation; it is approved for patients receiving high cumulative doses of doxorubicin. Beta-blockers such as carvedilol and bisoprolol improve myocardial energetics and attenuate oxidative stress. Angiotensin-converting enzyme (ACE) inhibitors and angiotensin receptor blockers (ARBs) reduce afterload and inhibit maladaptive remodeling. Clinical trials have demonstrated that initiating these agents before or early during chemotherapy can preserve LVEF and reduce the incidence of heart failure.

Surveillance protocols outline the timing and modalities for cardiac monitoring in patients undergoing potentially cardiotoxic therapy. A typical schedule may involve baseline echocardiography, repeat imaging at 3-month intervals during treatment, and follow-up assessments at 6-month or yearly intervals after completion, depending on the agent and cumulative dose. The European Society of Cardiology and the American Society of Clinical Oncology provide consensus recommendations that incorporate LVEF, GLS, and biomarker measurements to tailor surveillance intensity.

Risk factors for cardiotoxicity are multifactorial. Age greater than 65 years, pre-existing cardiovascular disease, hypertension, diabetes, and dyslipidemia increase vulnerability. High cumulative anthracycline dose, combined modality therapy (e.g., Anthracyclines plus trastuzumab), and prior chest radiation further amplify risk. Genetic predisposition, such as polymorphisms in the NAD(P)H oxidase pathway, is an emerging area of investigation that may eventually inform personalized risk assessment.

Mechanisms of injury vary by drug class. Anthracyclines generate reactive oxygen species that damage mitochondrial DNA and impair calcium handling. HER2-targeted agents disrupt protective signaling pathways that normally safeguard cardiomyocytes against stress. TKIs may cause endothelial dysfunction, leading to hypertension and reduced coronary perfusion. ICIs trigger immune activation that can target cardiac antigens, resulting in myocarditis. Understanding these mechanisms guides both preventive strategies and therapeutic interventions.

Clinical manifestations of cardiotoxicity are diverse. Heart failure may present with dyspnea, fatigue, peripheral edema, and reduced exercise tolerance. Arrhythmias, ranging from atrial fibrillation to ventricular tachycardia, can be precipitated by electrolyte shifts, direct myocardial injury, or inflammation. Hypertension is a common side-effect of VEGF-targeted TKIs and may require antihypertensive therapy to avoid further cardiac strain. Coronary artery disease can be accelerated by radiation-induced endothelial injury, necessitating aggressive lipid management.

Management strategies are guided by the severity and type of cardiac involvement. For asymptomatic LVEF decline, guideline-directed medical therapy (GDMT) with beta-blockers and ACE inhibitors is often initiated, and the offending cancer drug may be temporarily paused or dose-reduced. Symptomatic heart failure mandates full GDMT, possible hospitalization, and multidisciplinary discussion regarding continuation of oncologic therapy. In cases of ICI-related myocarditis, high-dose corticosteroids are administered, followed by tapering based on clinical response.

Cardiomyopathy is a generic term describing disease of the heart muscle that impairs its ability to pump blood effectively. In cardio-oncology, the most common form is dilated cardiomyopathy resulting from anthracycline exposure. However, restrictive cardiomyopathy may arise after radiation therapy due to extensive myocardial fibrosis. Distinguishing the underlying etiology influences both treatment and prognosis.

Myocarditis denotes inflammation of the myocardium and can be infectious, autoimmune, or drug-induced. Immune checkpoint inhibitor-associated myocarditis is an exemplar of drug-induced myocarditis, often presenting with elevated troponin, diffuse ST-segment changes on ECG, and reduced LVEF. Cardiac magnetic resonance imaging can reveal late gadolinium enhancement, confirming the diagnosis. Early recognition and immunosuppression are critical to improve outcomes.

Pericarditis involves inflammation of the pericardial sac and may manifest as chest pain, pericardial friction rub, and, in severe cases, tamponade. Radiation therapy to the mediastinum is a leading cause of chronic pericardial disease, while certain chemotherapeutic agents (e.g., Cyclophosphamide) can precipitate acute pericarditis. Management may require anti-inflammatory agents, pericardiocentesis, or surgical pericardiectomy in refractory cases.

Coronary artery disease (CAD) in the oncology setting can be accelerated by radiation-induced endothelial injury, leading to premature atherosclerosis. Patients receiving chest irradiation should be evaluated for traditional CAD risk factors and may benefit from low-dose aspirin, statin therapy, and lifestyle counseling. Non-invasive stress testing or coronary CT angiography can be employed for early detection of obstructive lesions.

Imaging modalities each provide unique information. Echocardiography is the first-line tool, offering real-time assessment of LVEF, GLS, and diastolic function. Cardiac magnetic resonance (CMR) provides gold-standard quantification of ventricular volumes, tissue characterization (including edema and fibrosis), and is particularly valuable when echocardiographic windows are suboptimal. Multigated acquisition (MUGA) scan offers high reproducibility for LVEF measurement but involves ionizing radiation; its use has declined with the advent of advanced echo techniques. Positron emission tomography (PET) may be employed to detect inflammatory activity in ICI-related myocarditis.

Strain imaging extends beyond GLS to include radial and circumferential strain, offering a comprehensive view of myocardial deformation. Reduced strain values precede LVEF decline and correlate with histological myocardial injury. In practice, clinicians may set a threshold of a 10-15% relative drop in GLS to trigger cardioprotective therapy, even if LVEF remains unchanged.

Fractional shortening is a simple M-mode echocardiographic measure of systolic function, calculated as the percentage reduction in left ventricular internal diameter from diastole to systole. While less sensitive than GLS, it remains useful in settings where advanced speckle-tracking software is unavailable. Its interpretation must consider loading conditions and heart rate.

Pharmacovigilance in cardio-oncology involves systematic monitoring of adverse drug reactions, including cardiac events. Reporting systems such as the FDA's MedWatch or the European Medicines Agency's

EudraVigilance collect data on cardiotoxicity incidence, enabling post-marketing safety analyses. Clinicians are encouraged to document and share any unexpected cardiac events to refine risk estimates and guide future guidelines.

Pharmacodynamics describes how a drug exerts its therapeutic and toxic effects at the molecular level. For cardiotoxic agents, pharmacodynamic considerations include the affinity for cardiac ion channels, the propensity to generate reactive oxygen species, and the interaction with cardioprotective signaling pathways. Understanding these principles assists in selecting agents with favorable cardiac safety profiles when multiple therapeutic options exist.

Pharmacokinetics encompasses absorption, distribution, metabolism, and excretion of a drug. Variability in these processes can influence cardiotoxic risk. For example, polymorphisms in the CYP3A4 enzyme affect the clearance of certain TKIs, potentially leading to higher plasma concentrations and increased hypertension. Dose adjustments based on renal or hepatic function are therefore critical to mitigate cardiac side-effects.

Onco-cardiology (also termed cardio-oncology) is an emerging subspecialty that bridges oncology and cardiology. Practitioners in this field must be fluent in both oncologic treatment regimens and cardiovascular disease management. The multidisciplinary team typically includes medical oncologists, cardiologists, radiation oncologists, pharmacists, and nursing specialists, all collaborating to balance optimal cancer control with preservation of cardiac health.

Multidisciplinary team (MDT) dynamics are central to successful cardio-oncology care. Regular case conferences allow for real-time adjustment of cancer therapy based on cardiac findings, and vice versa. For instance, if a patient develops a 12 % reduction in GLS during trastuzumab treatment, the MDT may decide to introduce an ACE inhibitor, postpone the next trastuzumab infusion, and schedule more frequent cardiac imaging.

Risk stratification tools such as the Heart Failure Association-International Cardio-Oncology Society (HFA-ICOS) risk score integrate patient demographics, treatment variables, and baseline cardiac parameters to predict the likelihood of cardiotoxicity. These tools guide the intensity of surveillance and the threshold for prophylactic therapy. Validation studies have demonstrated that a high-risk score correlates with a 3- to 5-fold increase in symptomatic heart failure incidence.

Electrocardiogram (ECG) changes may herald early cardiotoxicity. New-onset ST-segment depression, T-wave inversion, or conduction abnormalities should prompt further evaluation. In ICI-related myocarditis, diffuse ST-segment elevation and ventricular arrhythmias are common. Serial ECGs, especially before and after high-risk chemotherapy cycles, provide a low-cost method for early detection.

Arrhythmogenesis in the setting of cancer therapy can be precipitated by electrolyte disturbances (e.G., Hypokalemia from cisplatin), direct myocardial injury, or autonomic imbalance. Atrial fibrillation, a frequent complication, may be exacerbated by systemic inflammation and volume shifts. Management follows standard anti-arrhythmic protocols but must account for drug-drug interactions, especially with agents that prolong the QT interval.

QT prolongation is a recognized adverse effect of several oncology drugs, including arsenic trioxide, certain TKIs, and some anti-emetics. Prolonged QT predisposes to torsades de pointes, a life-threatening polymorphic ventricular tachycardia. Baseline ECG, correction of electrolyte abnormalities, and avoidance of concurrent QT-prolonging medications are essential preventive measures. Continuous telemetry may be indicated for high-risk patients.

Hypertension management during VEGF-targeted therapy requires vigilant blood pressure monitoring. Even modest elevations ($\geq 140/90$ mmHg) can increase the risk of subsequent left ventricular dysfunction. First-line antihypertensives include calcium channel blockers and ACE inhibitors; however, selection must consider the patient's renal function, drug interactions, and potential impact on tumor perfusion.

Vascular toxicity extends beyond hypertension to include endothelial dysfunction, thromboembolic events, and accelerated atherosclerosis. Agents such as bevacizumab and sunitinib can increase the incidence of arterial thromboembolism. Prophylactic antiplatelet therapy is controversial and should be individualized based on bleeding risk and cancer type.

Biomarker-guided therapy utilizes serial troponin or natriuretic peptide measurements to inform treatment decisions. A rise in troponin above the upper reference limit after an anthracycline infusion may trigger the initiation of a beta-blocker, even in the absence of imaging abnormalities. Similarly, a progressive increase in NT-proBNP can signal impending heart failure, prompting earlier escalation of GDMT.

Exercise prescription is an increasingly recognized component of cardiotoxicity mitigation. Structured aerobic training, performed before, during, or after chemotherapy, can improve myocardial reserve, enhance endothelial function, and reduce fatigue. Clinical trials have shown that moderate-intensity exercise (e.g., 150 Minutes per week) can attenuate LVEF decline in patients receiving anthracyclines, though individualized programs are necessary to accommodate cancer-related fatigue and immunosuppression.

Nutrition and lifestyle interventions complement pharmacologic cardioprotection. A Mediterranean-style diet rich in omega-3 fatty acids, antioxidants, and whole grains supports vascular health and may reduce oxidative stress. Smoking cessation, weight management, and glycemic control are vital, particularly for patients with pre-existing cardiovascular risk factors.

Genetic susceptibility research has identified polymorphisms in genes such as SLC28A3, RARG, and NADPH oxidase that influence anthracycline-related cardiotoxicity. While routine genetic testing is not yet standard practice, emerging data suggest that in the future, genotype-guided therapy could personalize cardioprotective strategies, selecting patients who would benefit most from dexrazoxane or intensified monitoring.

Long-term survivorship care addresses the delayed cardiovascular sequelae that emerge years after cancer treatment. Survivorship clinics often incorporate periodic echocardiography, lipid panels, glucose monitoring, and counseling on physical activity. The goal is to detect late-onset cardiotoxicity early, manage modifiable risk factors, and integrate cardiovascular health into the overall survivorship plan.

Clinical trial design in cardio-oncology must incorporate cardiac endpoints. Traditional oncology trials focus on tumor response and overall survival, but inclusion of LVEF, GLS, and cardiac event rates provides a more

holistic assessment of therapeutic benefit versus risk. Adaptive trial designs may allow for early stopping of cardiotoxic agents or the addition of protective co-therapies based on interim cardiac safety data.

Regulatory considerations influence drug labeling and post-marketing surveillance. The FDA and EMA require manufacturers to include cardiac safety data in the prescribing information for agents known to affect heart function. Labeling may recommend baseline cardiac assessment, periodic monitoring, and dose modifications for patients who develop cardiotoxicity. Understanding these regulatory mandates helps clinicians adhere to best practice standards.

Electronic health records (EHR) integration facilitates systematic cardiac monitoring. Embedding alerts for scheduled echocardiograms, flagging abnormal biomarker trends, and providing decision-support tools for dose adjustments streamline workflow and reduce missed appointments. Data extraction from EHRs also supports real-world evidence studies on cardiotoxicity incidence and outcomes.

Patient education is a cornerstone of effective cardiotoxicity management. Patients should be informed about the potential cardiac side-effects of their cancer therapy, the importance of reporting symptoms such as shortness of breath, palpitations, or swelling, and the need for adherence to scheduled cardiac evaluations. Educational materials, tailored to health literacy levels, improve engagement and early detection.

Psychosocial aspects of cardiotoxicity include anxiety related to the dual burden of cancer and heart disease. Psychological support services, counseling, and peer support groups can help patients cope with the uncertainty of treatment-related cardiac risk. Addressing mental health improves overall quality of life and may enhance adherence to both oncologic and cardiac therapies.

Future directions in cardio-oncology research include development of novel biomarkers (e.G., Micro-RNA panels), advanced imaging techniques such as 4-D flow MRI, and integration of artificial intelligence to predict individual cardiotoxicity risk. Ongoing trials are evaluating the efficacy of combination cardioprotective regimens, including simultaneous beta-blocker and mineralocorticoid receptor antagonist therapy, to further reduce the incidence of heart failure in high-risk patients.

Telemedicine has emerged as a valuable tool for remote cardiac monitoring, especially for patients undergoing long-term therapy or residing far from specialized centers. Home-based blood pressure cuffs, portable ECG patches, and mobile applications that record symptom diaries enable clinicians to track cardiac status in real time, adjust medications promptly, and reduce unnecessary clinic visits.

Interdisciplinary research collaborations between oncologists, cardiologists, pharmacologists, and basic scientists accelerate the translation of mechanistic insights into clinical practice. For instance, studies elucidating the role of mitochondrial permeability transition pores in anthracycline injury have led to experimental therapies targeting these pathways, with the aim of preserving myocardial energetics without compromising anti-tumor efficacy.

Health economics considerations are increasingly relevant as cardio-oncology services expand. Cost-effectiveness analyses compare the expense of intensive cardiac surveillance and prophylactic medication against the long-term costs of managing heart failure, hospitalizations, and reduced

productivity. Evidence suggests that early detection and intervention, though initially resource-intensive, yield overall savings by preventing severe cardiac events.

Ethical considerations arise when balancing life-extending cancer therapy against potential cardiac harm. Informed consent processes must transparently discuss the probability and severity of cardiotoxicity, allowing patients to make autonomous decisions regarding treatment continuation. Ethical frameworks also guide allocation of limited cardiology resources to cancer patients, ensuring equitable access to specialized care.

Training and certification pathways for cardio-oncology professionals are being formalized in many countries. Fellowships, dedicated curricula, and competency assessments aim to standardize expertise, ensuring that clinicians can competently interpret cardiac imaging, manage complex pharmacologic interactions, and lead multidisciplinary teams. Continuous professional development, through conferences and journal clubs, maintains up-to-date knowledge of rapidly evolving therapies.

Case studies illustrate the application of terminology in real-world scenarios. A 58-year-old woman with HER2-positive breast cancer receives trastuzumab after completing an anthracycline regimen. Baseline LVEF is 62 %, and GLS is –20 %. After three months of trastuzumab, LVEF remains 60 % but GLS declines to –17 %, representing a 15 % relative reduction. The MDT initiates an ACE inhibitor, continues trastuzumab with close monitoring, and repeats imaging in six weeks. This example demonstrates how subclinical strain changes trigger cardioprotective measures before overt systolic dysfunction emerges.

Another example involves a 45-year-old man with metastatic melanoma treated with nivolumab. He presents with chest discomfort and a troponin rise to 2 × ULN. ECG shows diffuse ST-segment elevation, and CMR reveals myocardial edema with late gadolinium enhancement. A diagnosis of ICI-related myocarditis is made, and high-dose prednisone is started. Within 48 hours, troponin levels decline, and LVEF improves from 45 % to 55 %. This case underscores the importance of rapid biomarker assessment, imaging, and immunosuppression in managing immune-mediated cardiac toxicity.

A third scenario features a 70-year-old survivor of Hodgkin lymphoma who received mantle-field radiation 20 years prior. Routine surveillance detects a 12 % increase in coronary calcium score and a mild reduction in LVEF to 48 %. The patient is started on a statin, low-dose aspirin, and a beta-blocker, and is referred for a stress cardiac MRI to assess for inducible ischemia. This illustrates the long-term cardiovascular vigilance required after thoracic radiation exposure.

Terminology summary provides a quick reference for learners. Terms such as dose-dependent, subclinical, strain imaging, and multidisciplinary team are central to the cardio-oncology lexicon. Mastery of these concepts enables clinicians to translate complex pathophysiological mechanisms into practical patient care strategies, ultimately improving both oncologic and cardiac outcomes.

Practical tip for clinicians: Always document baseline cardiac parameters before initiating a known cardiotoxic agent. Use a standardized template that includes LVEF, GLS, troponin, and NT-proBNP. This baseline serves as a reference point for detecting early changes and facilitates communication across specialties. Moreover, integrating alerts into the EHR for scheduled follow-up imaging reduces the

likelihood of missed assessments.

Challenges in the field include variability in imaging quality across institutions, limited access to advanced modalities like CMR, and heterogeneity in biomarker assay standardization. Additionally, the rapid introduction of novel therapies, such as chimeric antigen receptor (CAR) T-cell therapy, brings new, incompletely understood cardiac toxicities that require ongoing research and guideline development.

Emerging therapies such as CAR T-cell therapy can cause cytokine release syndrome, which may precipitate acute heart failure, arrhythmias, or myocardial stunning. Monitoring for cardiac involvement in patients receiving CAR T-cells involves serial troponin, BNP, and echocardiography, with readiness to provide inotropic support or immunomodulation if severe cardiac dysfunction develops.

Precision medicine approaches aim to tailor cancer treatment based on molecular tumor characteristics while simultaneously considering the patient's cardiac risk profile. Pharmacogenomic testing may identify individuals at heightened risk for anthracycline cardiotoxicity, guiding dose adjustments or selection of less cardiotoxic alternatives. Integration of cardiac risk algorithms into tumor board discussions exemplifies precision cardio-oncology.

Data registries such as the Cardio-Oncology Registry collect longitudinal information on cancer patients receiving potentially cardiotoxic therapies. These databases enable real-world analysis of cardiotoxicity incidence, risk factors, and outcomes, informing future guideline updates and identifying gaps in care delivery.

Policy implications highlight the need for health systems to allocate resources for cardiac monitoring, develop reimbursement models that support preventive cardiology in oncology, and promote research funding for cardio-oncology initiatives. Advocacy by professional societies can influence policy to ensure that cardiac health is incorporated into cancer care pathways.

Key take-away for learners: Understanding the precise definitions of cardiotoxic terms, recognizing the mechanisms behind each therapeutic class, and applying evidence-based monitoring strategies are essential to mitigate cardiac risk while delivering optimal cancer treatment. Mastery of this vocabulary equips practitioners to navigate the complex interplay between oncologic efficacy and cardiovascular safety, ultimately improving patient survival and quality of life.