

Review article

Radiation-induced coronary artery disease: prevention, screening and contemporary management

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ABSTRACT

Radiotherapy has become an integral component of modern cancer treatment, contributing substantially to improved survival among patients with thoracic malignancies. However, as the number of long-term cancer survivors continues to increase, the recognition of late cardiovascular complications has become an important aspect of survivorship care. Among these complications, radiation-induced coronary artery disease (RICAD) represents one of the most clinically significant manifestations of radiation-associated cardiotoxicity, contributing to considerable long-term morbidity and mortality. The pathogenesis of RICAD is multifactorial and differs from conventional atherosclerosis, involving radiation-induced endothelial injury, chronic inflammation, oxidative stress, and progressive vascular fibrosis, ultimately leading to accelerated coronary artery disease. The clinical presentation is frequently delayed by many years after completion of radiotherapy and may remain asymptomatic or present with atypical manifestations, making timely diagnosis particularly challenging. The risk of RICAD is influenced not only by the radiation dose delivered to the heart but also by patient characteristics, cardiovascular comorbidities, tumour location, and advances in radiotherapy techniques. As cancer survival continues to improve, identification of high-risk patients, implementation of appropriate screening strategies, and selection of optimal diagnostic and therapeutic approaches have become increasingly important in clinical practice. This review summarizes the current evidence regarding risk factors, screening recommendations, diagnostic evaluation, contemporary management, and preventive strategies for radiation-induced coronary artery disease.

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INTRODUCTION

Advances in cancer treatment have significantly improved long-term survival, leading to a growing population of cancer survivors at risk of late treatment-related complications. Among these complications, cardiovascular disease represents one of the most important causes of morbidity and mortality. Radiotherapy remains an essential component of treatment for many malignancies; however, exposure of cardiac structures to ionizing radiation may result in long-term cardiovascular injury.

Radiation-induced coronary artery disease (RICAD) is a clinically significant manifestation of radiation-induced cardiotoxicity, characterized by accelerated atherosclerosis, preferential involvement of proximal coronary segments, and a prolonged latency period between radiation exposure and clinical presentation. The risk of RICAD depends on multiple factors, including radiation dose, irradiated cardiac volume, age at treatment, and conventional cardiovascular risk factors.

Despite increasing recognition of radiation-associated cardiovascular complications, RICAD remains frequently underdiagnosed due to its delayed onset and often asymptomatic course. Early identification of high-risk patients, appropriate surveillance strategies, and optimization of cardiovascular prevention are essential to reduce long-term morbidity.

This review summarizes current knowledge regarding the pathophysiology, risk factors, screening strategies, diagnostic approaches, management, and prevention of RICAD in cancer survivors.

MATERIALS AND METHODS

A literature search was performed to identify relevant studies addressing radiation-induced coronary artery disease (RICAD) and radiation-induced cardiovascular toxicity. The search was conducted using PubMed/MEDLINE, Scopus, and Web of Science databases. The search strategy included combinations of the following keywords: “radiation-induced cardiotoxicity”, “radiation-induced coronary artery disease”, “thoracic radiotherapy”, “coronary artery disease”, “cardiovascular complications”, and “cancer survivors”.

Relevant publications, including original research articles, systematic reviews, meta-analyses, clinical guidelines, and expert consensus statements, were considered. Studies were selected based on their relevance to the epidemiology, pathophysiological mechanisms, risk factors, screening strategies, diagnostic approaches, treatment options, and prevention of RICAD. Particular attention was given to studies involving patients treated with thoracic radiotherapy for Hodgkin lymphoma, breast cancer, lung cancer, and oesophageal cancer.

The identified literature was critically evaluated, and findings were synthesized narratively according to the main clinical aspects of RICAD. Due to heterogeneity in study populations, radiation exposure parameters, diagnostic methods, and clinical outcomes, a qualitative synthesis was performed rather than a quantitative meta-analysis.

CHARACTERISTIC FEATURES OF RADIATION-INDUCED CARDIOTOXICITY

Radiation-induced cardiotoxicity represents a multifactorial and complex pathological process involving numerous molecular and cellular pathways. Exposure to ionizing radiation leads to cellular damage through direct disruption of DNA structures as well as indirect mechanisms associated with the formation of reactive oxygen species (ROS) [1, 2].

Within the cardiovascular system, endothelial damage affecting the coronary arteries is considered one of the earliest manifestations of radiation exposure. This initial injury triggers a persistent inflammatory response associated with enhanced oxidative stress, increased ROS generation, and the secretion of pro-inflammatory mediators. These mechanisms further aggravate vascular injury and compromise genomic stability. The subsequent inflammatory cascade promotes endothelial dysfunction, platelet activation, thrombus formation, and the replacement of injured intimal cells by myofibroblasts [3, 4]. This process results in a gradual reduction of coronary vessel diameter, predominantly affecting the proximal portions of the arteries, including the areas surrounding the coronary ostia.

RICAD may present with a wide range of clinical manifestations, from clinically silent coronary artery narrowing to symptomatic angina, acute coronary syndromes, and myocardial infarction. Individuals previously treated with radiotherapy may have an increased incidence of silent myocardial infarction, potentially due to altered pain perception [5]. The location and extent of coronary lesions are strongly associated with the anatomical region exposed to radiation. Mediastinal radiotherapy most frequently affects the left anterior descending artery (LAD) and the right coronary artery (RCA), while irradiation of the left breast is primarily associated with involvement of the LAD [6]. A distinctive characteristic of RICAD is the preferential occurrence of lesions in the proximal portions of coronary arteries, particularly near their origins [6].

WHICH PATIENTS ARE AT RISK?

RICAD occurs predominantly in patients treated with thoracic radiotherapy, particularly survivors of Hodgkin lymphoma, breast

cancer, and thoracic malignancies such as oesophageal and lung cancer [3]. The risk is primarily determined by the radiation dose delivered to the heart, with higher cardiac exposure translating into a greater likelihood of subsequent coronary events [7, 8]. However, radiation dose alone does not fully explain the variability in risk. Patient-related factors, including age at treatment, pre-existing cardiovascular risk factors, and lifestyle characteristics, further influence the absolute probability of developing coronary heart disease after radiotherapy [7, 8]. Consequently, these patient populations require particular attention during long-term cardiovascular follow-up.

Among all thoracic malignancies, Hodgkin lymphoma survivors constitute one of the highest-risk groups because mediastinal radiotherapy historically exposed the heart to substantially higher cumulative radiation doses than those used for most other cancers [7]. Coronary risk increases linearly with increasing mean heart dose, resulting in an approximately 2.5-fold higher risk of coronary artery disease in patients receiving a mean heart dose of 20 Gy compared with those who were not treated with mediastinal radiotherapy [7]. The relative effect of radiation appears to be greater in patients treated at a younger age, whereas the absolute risk is further amplified by conventional cardiovascular risk factors. Hypertension is the most consistently identified predictor, while diabetes mellitus, hypercholesterolaemia, obesity, recent smoking, and a positive family history of coronary heart disease also contribute to increased risk. Conversely, regular physical activity has been associated with a lower incidence of coronary artery disease in Hodgkin lymphoma survivors [7].

Breast cancer represents the largest population at risk because of the widespread use of adjuvant radiotherapy [8]. Although modern treatment techniques have substantially reduced cardiac exposure, mean heart doses remain highly dependent on tumour laterality and treatment fields. Typical doses are approximately 1–2 Gy for right-sided breast cancer, whereas irradiation of left-sided tumours, particularly when the internal mammary nodes are included, may result in substantially higher cardiac doses approaching 10 Gy [8]. Similar to Hodgkin lymphoma, the incidence of major coronary events increases proportionally with increasing mean heart dose and without an apparent threshold [8]. The excess risk becomes evident within the first 5 years after treatment and persists for at least 2 decades. While patients with pre-existing cardiovascular risk factors have a higher baseline incidence of coronary events, the proportional increase associated with radiation exposure is similar irrespective of baseline cardiovascular status [8].

Patients treated for oesophageal cancer are also particularly vulnerable because the heart is frequently included within the high-dose radiation field due to its close anatomical relationship

with the oesophagus [9, 10]. Unlike breast cancer and Hodgkin lymphoma, cardiac complications may become clinically apparent relatively early, with a significantly increased risk of heart disease-related death detectable as early as 8 months after radiotherapy [9]. Radiotherapy remains an independent predictor of cardiac mortality, and the absolute risk continues to increase over long-term follow-up, reaching 9.4% at 20 years [9]. Older age and tumours located in the lower oesophagus further increase the probability of cardiac death following treatment [10]. In lung cancer, the risk of radiation-induced cardiac injury is additionally influenced by the complexity of treatment and the high burden of pre-existing comorbidities. Current studies report cardiac complications in up to one-third of patients after thoracic radiotherapy [11]. Besides cardiac radiation dose, important determinants of risk include tumour lobar location, treatment modality, pre-existing cardiovascular and pulmonary disease, and smoking history, all of which should be considered when estimating an individual's susceptibility to RICAD [11].

WHEN SHOULD WE SCREEN?

The timing of screening should reflect the natural history of radiation-induced coronary injury. Clinical RICAD most commonly becomes evident after a latency period of approximately 15 years following radiotherapy [12], although the onset of cardiovascular risk may occur considerably earlier depending on the underlying malignancy and clinical endpoint. In breast cancer survivors, the incidence of coronary heart disease begins to increase within the first decade after radiotherapy, whereas excess cardiac mortality becomes apparent from the second decade of follow-up [13]. These findings indicate that radiation-related coronary damage develops progressively over many years, supporting the need for continued long-term cardiovascular monitoring even in patients treated with contemporary radiotherapy techniques [13].

The importance of surveillance is particularly evident in survivors of Hodgkin lymphoma, who generally achieve excellent long-term cancer-specific survival but remain at increased risk of late cardiovascular complications, including coronary artery disease. Because cardiovascular disease represents one of the leading causes of excess late mortality in this population, early detection of coronary artery disease is essential to enable timely preventive and therapeutic interventions [14].

Overall, the available evidence indicates that screening strategies should be based on the delayed but progressive nature of radiation-induced coronary injury. Long-term follow-up is warranted because coronary disease may remain clinically silent for years before presenting, and the cardiovascular risk persists well beyond the completion of cancer treatment [12–14].

DIAGNOSTIC APPROACH

The diagnostic evaluation of RICAD follows the general principles applied to conventional coronary artery disease; however, several important differences should be considered. Patients previously exposed to thoracic radiotherapy are more likely to develop ostial lesions, diffuse epicardial disease, and microvascular dysfunction, while clinical presentation may be atypical or even silent. Consequently, diagnostic strategies should combine anatomical and functional assessment to detect both obstructive coronary lesions and radiation-related microvascular abnormalities [15].

Coronary computed tomography angiography (CCTA) is one of the most valuable non-invasive imaging modalities for the evaluation of RICAD because of its nearly 100% negative predictive value for excluding significant coronary artery disease [14]. Unlike functional tests, CCTA allows direct visualization of coronary atherosclerosis before myocardial ischaemia develops, facilitating earlier preventive interventions [15]. In a prospective cohort of Hodgkin lymphoma survivors, CCTA identified coronary abnormalities in 26% of patients, with significant stenosis requiring surgical treatment in 5.5% [14]. Most lesions were non-ostial, although ostial stenoses were also common, reflecting the characteristic distribution of radiation-induced disease [7]. Importantly, coronary abnormalities were detected as early as the first 5 years after radiotherapy, with their prevalence increasing significantly after 10 years and reaching approximately 35% after 15 years [14]. When intermediate lesions are identified, CT-derived fractional flow reserve (CT-FFR) may further determine their functional significance and guide subsequent management [15].

Coronary artery calcium (CAC) scoring provides an additional CT-based method for cardiovascular risk stratification. Although it detects only calcified plaques, it requires no contrast administration and exposes patients to lower radiation doses than CCTA [15]. Before radiotherapy, CAC scoring predicts future cardiovascular events more accurately than the Framingham risk score, while after thoracic irradiation its high negative predictive value makes it useful for excluding significant coronary artery disease. Because thoracic CT examinations are routinely performed in oncology patients before and after treatment, assessment of coronary calcium from these studies represents an attractive opportunity for cardiovascular risk stratification without additional imaging [15].

In symptomatic patients, functional imaging should be selected according to standard recommendations, although each modality has specific advantages in the post-radiotherapy population [15]. Stress echocardiography demonstrates high specificity (89%) and positive predictive value (87%) for detecting functionally significant coronary artery disease and predicting future coronary events, despite only moderate sensitivity (59%) [15]. By contrast,

SPECT myocardial perfusion imaging provides higher sensitivity but substantially lower specificity after thoracic radiotherapy, while fixed perfusion defects may reflect myocardial fibrosis rather than active ischaemia, limiting diagnostic accuracy [15].

Compared with SPECT, positron emission tomography (PET) offers superior spatial and temporal resolution, lower radiation exposure, and higher diagnostic accuracy [15]. Moreover, quantitative assessment of absolute myocardial blood flow and myocardial flow reserve enables reliable evaluation of both diffuse epicardial disease and coronary microvascular dysfunction, abnormalities that are particularly relevant after thoracic irradiation and cannot be accurately assessed by SPECT [15].

CONTEMPORARY MANAGEMENT

Pharmacological strategies in RICAD

The treatment strategy for RICAD largely follows the principles applied in patients with conventional coronary artery disease who have not been exposed to radiation. Effective management requires a comprehensive and multidisciplinary approach. Intensive secondary prevention measures are essential and should include strict control of modifiable cardiovascular risk factors, such as tobacco use, excessive body weight, elevated cholesterol levels, impaired glycaemic control, and hypertension. These factors may interact synergistically with previous radiation exposure, substantially increasing the long-term probability of major coronary events, from approximately 2% to 7% [16].

Statin therapy has been associated with a lower incidence of major adverse cardiovascular events (MACE) in patients with breast cancer treated with breast-conserving surgery followed by adjuvant whole-breast radiotherapy [17]. Among available statins, the hydrophilic agents rosuvastatin and pravastatin appear to provide the greatest cardioprotective benefit [17]. These findings support the potential use of statins as a strategy to reduce radiation-related cardiovascular toxicity, although further prospective studies are needed to establish their optimal role in breast cancer survivors receiving radiotherapy.

Pharmacological treatment of established coronary artery disease plays a crucial role in alleviating symptoms, slowing the progression of atherosclerotic disease, lowering the incidence of cardiovascular complications, and improving overall survival. Unless contraindicated, all patients with coronary artery disease should receive low-dose acetylsalicylic acid therapy (75–100 mg once daily). Furthermore, intensive statin therapy is recommended regardless of baseline lipid levels, with the objective of reducing low-density lipoprotein cholesterol (LDL-C) concentrations by at least 50% from the initial value and achieving a target LDL-C level below 1.4 mmol/L [18].

ACE inhibitors are recommended in patients with chronic coronary syndromes who have left ventricular dysfunction, hypertension, diabetes, or chronic kidney disease, particularly when additional cardiovascular risk reduction is indicated. Beta-blockers and/or calcium channel blockers are recommended as first-line anti-anginal therapies for symptom control in patients with chronic coronary syndromes. HbA_{1c} targets should generally be individualized, with a value below 53 mmol/mol (<7%) appropriate for many patients [18].

Coronary artery bypass grafting and percutaneous coronary intervention

Management of RICAD presents several challenges that may influence the choice of revascularization strategy. Although coronary artery bypass grafting (CABG) remains an established treatment option, previous radiation exposure may compromise surgical outcomes due to fibrosis and stenosis of arterial and venous graft conduits, impaired wound healing, increased bleeding risk, radiation-induced pulmonary fibrosis, and extensive calcification of the thoracic aorta. These factors contribute to increased perioperative risk and should be carefully considered before surgical intervention [16, 19].

The long-term outcomes of CABG and percutaneous coronary intervention (PCI) in patients with RICAD remain uncertain. Previous studies have reported higher postoperative morbidity and mortality after cardiac surgery in patients with prior mediastinal radiation, although other analyses have shown comparable long-term outcomes in selected patients [16, 19]. Similarly, outcomes following PCI remain controversial, with some studies demonstrating acceptable results, while others suggesting increased mortality and adverse events compared with non-irradiated patients [20]. As direct comparisons between PCI and CABG in patients with RICAD are lacking, the optimal revascularization strategy remains unclear. Therefore, treatment decisions should be individualized and based on patient characteristics, coronary anatomy, surgical risk, and multidisciplinary heart team assessment. In patients with increased operative risk, PCI may represent a reasonable alternative, whereas surgical revascularization requires careful evaluation of radiation-related anatomical changes and potential complications [16, 21].

NEW RADIOTHERAPY TECHNIQUES TO PREVENT RADIATION-INDUCED CARDIOTOXICITY

As advances in cancer therapy improve tumour control and overall survival, the prevention and management of treatment-related adverse effects have become increasingly important. A primary goal of modern radiation oncology is to achieve effective tumour

eradication while minimizing radiation exposure to surrounding normal tissues at risk. Modern radiotherapy techniques can significantly reduce cardiac exposure and may lower the risk of radiation-induced cardiovascular complications. Deep inspiration breath-hold (DIBH) reduces the volume of the heart exposed to radiation, decreases mean cardiac dose, and lowers the dose delivered to the left anterior descending artery (LAD), potentially reducing the risk of radiation-related cardiac events [8]. Prone positioning may further decrease cardiac exposure in selected patients with left-sided breast cancer by reducing the amount of heart tissue included in the radiation field [22]. Advanced treatment modalities, including intensity-modulated radiotherapy (IMRT) and proton therapy, allow further reduction of cardiac irradiation and may provide additional protection of cardiac structures compared with conventional photon-based techniques [23]. The optimal cardioprotective strategy should be selected individually, considering tumour characteristics, patient anatomy, comorbidities, and available radiotherapy technology, with minimizing cardiac dose remaining a major goal of thoracic radiotherapy [8].

CONCLUSIONS

RICAD represents a unique and increasingly relevant form of coronary artery disease in the era of improving cancer survival. Because its development is often delayed and clinically silent, identifying patients at highest risk and implementing appropriate long-term surveillance are fundamental to reducing cardiovascular morbidity and mortality. Multimodality imaging, with coronary CT angiography as a cornerstone, enables earlier detection of radiation-associated coronary lesions and should be integrated with functional assessment when clinically indicated. While current therapeutic strategies largely mirror those used for conventional coronary artery disease, management should account for the distinctive anatomical and pathological features associated with previous thoracic irradiation. Further advances in heart-sparing radiotherapy techniques, combined with optimized cardiovascular prevention and continued clinical research, are expected to improve the long-term cardiovascular prognosis of patients receiving thoracic radiotherapy.

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