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Cardiology

Ostial Stenosis of the Left Anterior Descending Artery after Left Breast Radiotherapy: An Avoidable Complication – A Case Report

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Abstract **Case Report**

Thoracic radiotherapy is an essential therapeutic modality in the management of breast cancer, particularly in locally advanced disease or following breast-conserving surgery. However, it is associated with potentially severe, often delayed cardiovascular complications. These conditions, frequently asymptomatic, are currently the leading non-malignant cause of death among cancer survivors treated with radiotherapy. Among late complications, radiation-induced coronary artery disease [RICAD] is the most concerning. We report the case of a 63-year-old woman with no significant cardiovascular risk factors, previously treated for left breast cancer with surgery, chemotherapy, and left-sided thoracic radiotherapy. A complete cardiological evaluation was performed following the onset of late chest pain. Echocardiography revealed segmental hypokinesia with reduced left ventricular ejection fraction. Coronary angiography demonstrated a severe ostial stenosis of the left anterior descending artery [LAD], without diffuse disease or calcification. Percutaneous coronary intervention with drug-eluting stent resulted in a favorable outcome, with partial recovery of ventricular function at 6 months. This case illustrates a severe and characteristic complication of left breast cancer treatment, likely secondary to radiation-induced fibrosing endarteritis. Diagnosis relies on lesion topography and the absence of classical atherosclerotic risk factors. Ostial LAD involvement should be suspected in any late ischemia in previously irradiated patients, even in the absence of conventional risk factors. Advances in radiotherapy techniques have reduced this risk, but long-term cardio-oncological follow-up remains essential.

Keywords: Radiotherapy, Breast cancer, Ischemic heart disease, Cardiac toxicity, Case report.

INTRODUCTION

Therapeutic advances in breast cancer have significantly improved overall survival. However, anticancer treatments, particularly radiotherapy and certain chemotherapies, are associated with an increased risk of long-term complications, notably cardiovascular events. Among these, radiation-induced cardiotoxicity is a well-documented phenomenon that is gaining increasing importance, particularly in left-sided breast irradiation, although it remains underrecognized [1]. First described in the 1960s, it may manifest several years after exposure and encompasses a spectrum of lesions affecting the pericardium, myocardium, valves, and coronary arteries [2]. After 10–15 years, cardiac exposure to radiotherapy has been shown to increase the incidence of heart failure, coronary artery disease, myocardial infarction, and cardiovascular mortality [3]. Radiation-induced coronary artery disease [RICAD], one of the most severe forms of this toxicity, is characterized by focal coronary lesions in segments exposed to radiation, which appear several years after treatment. The incidence of these lesions depends on the mean cardiac dose and the location of the radiation field. In left-sided breast cancer, the heart—particularly the anterior wall and the coronary ostia—is often partially included in the radiation field, exposing it to an increased risk of late ischemic damage. The literature primarily describes ostial or proximal stenoses of the LAD, the common trunk, or the left coronary arteries, and more rarely the right coronary arteries, depending on the topography of the radiation field.

We report a rare and illustrative case of severe radiation-induced ischemic heart disease occurring eight years after left breast radiotherapy in a patient without traditional cardiovascular risk factors, and discuss the mechanisms, diagnosis, and prevention strategies.

CASE PRESENTATION

2.1. Patient Presentation

A 63-year-old woman with no history of hypertension, diabetes, or smoking was treated in 2015 for invasive ductal carcinoma of the left breast, grade II[SBR II], hormone receptor-positive [HR+], HER2-negative.

2.2. Initial Oncological Treatment

She underwent radical surgery [mastectomy with axillary dissection], followed by adjuvant chemotherapy[anthracyclines]. Adjuvant radiotherapy was delivered to the chest wall and regional lymph nodes with a total dose of 50 Gy in 25 fractions over six weeks [2 Gy per fraction] [Figures 1 and 2]. Hormonal therapy with tamoxifen was prescribed for five years.

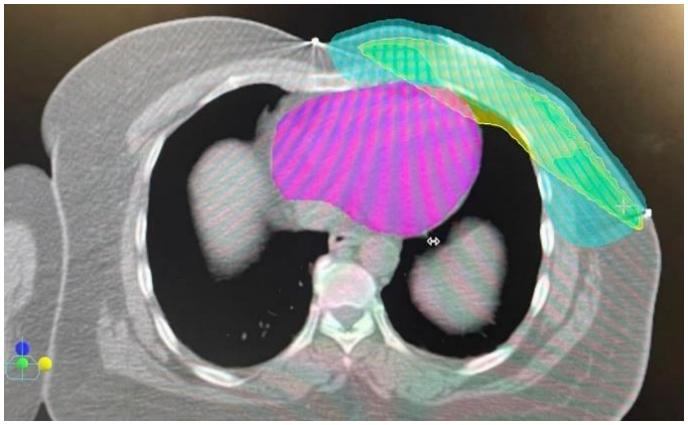

Figure 1: Axial isodose distribution showing inclusion of the heart within the left breast irradiation field

Figure 2: Thoracic dosimetric treatment planning

2.3. Timeline

Eight years after treatment completion, the patient presented with exertional dyspnea[NYHA II progressing to III] and intermittent constrictive chest pain, gradually worsening.

2.4. Diagnostic Workup

Clinical examination revealed regular heart sounds without murmurs and basal pulmonary crackles. Electrocardiography showed anterior ST-segment depression [Figure 3]. Troponin levels were mildly elevated [45 ng/L].

Transthoracic echocardiography demonstrated anterior hypokinesia with a left ventricular ejection fraction [LVEF] of 35% [Figure 4].

Coronary angiography revealed severe ostial stenosis of the LAD, without other significant lesions or diffuse calcifications [Figure 5].

Lipid and glycemic profiles were normal, the patient was a non-smoker, and no major cardiovascular risk factors were identified. The angiographic pattern, isolated topography, and absence of atherosclerotic lesions suggested a radiation-induced etiology.

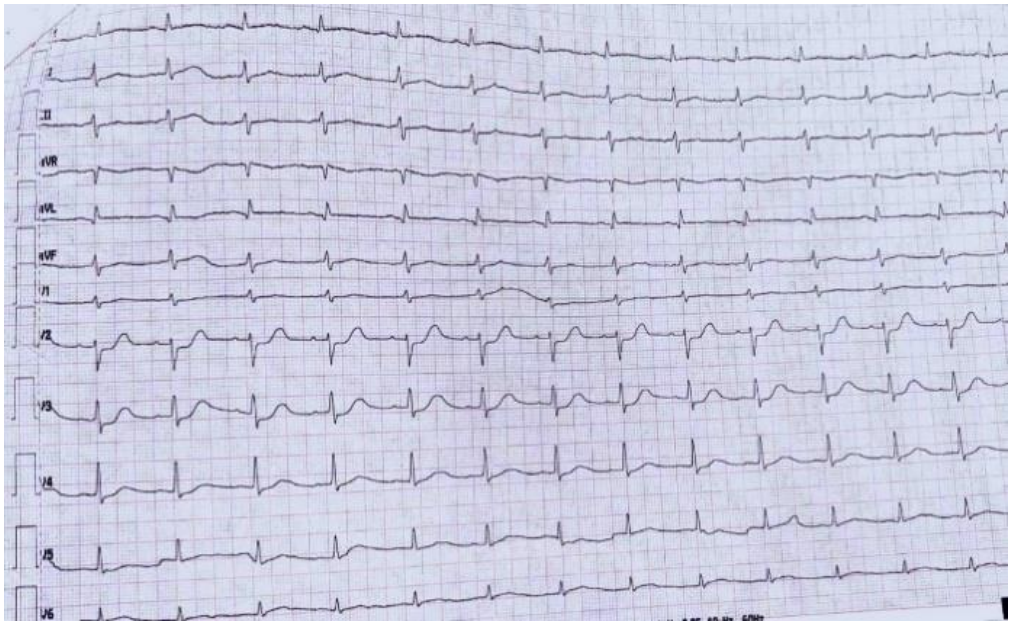

Figure 3: Anterior ST-segment depression on electrocardiogram

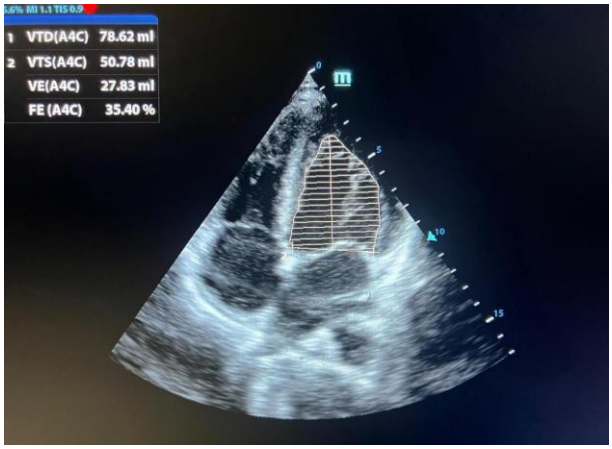

Figure 4: Initial transthoracic echocardiography showing a left ventricular ejection fraction[LVEF] of 35%

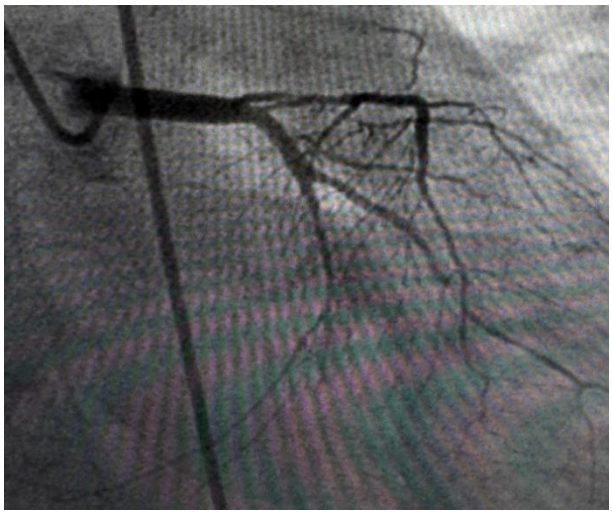

Figure 5: Severe ostial stenosis of the left anterior descending artery on coronary angiography

2.5. Therapeutic Intervention

Percutaneous coronary intervention with drug-eluting stent implantation was successfully performed, followed by dual antiplatelet therapy, statins, and beta-blockers.

2.6. Follow-up and Outcomes

The clinical course was favorable, with resolution of chest pain, improved exercise tolerance, and partial recovery of LVEF to 53% at six months [Figure 6]. No recurrent ischemic events were reported after 18 months of follow-up.

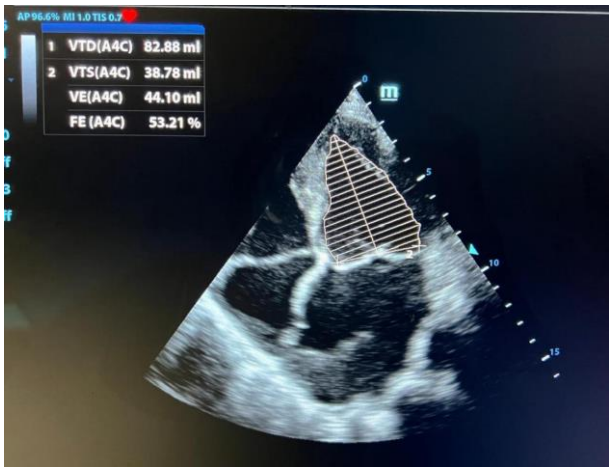

Figure 6: Follow-up transthoracic echocardiography showing an LVEF of 53%

DISCUSSION

2.7. Different forms of radiation-induced cardiac toxicity :

All cardiac tissues [pericardium, coronary arteries, myocardium, valves, and conduction tissue] can be damaged by radiation, leading to various complications. The severity of overall cardiac toxicity can be assessed according to the CTCAE v4.0 classification [Common Terminology Criteria for Adverse Events]. This is a descriptive terminology that can be used for reporting adverse events. A grading scale [or severity scale] is provided for each term [3].

Coronary damage [5–10%] is responsible for various late complications, occurring at least 5 years after radiation therapy [myocardial infarction, valvular heart disease, conduction disorders], particularly in patients with cardiovascular risk factors or those irradiated at a young age [3].

2.8. Epidemiology

Radiation-induced coronary lesions typically occur between 5 and 20 years after exposure, with an estimated incidence of 10% to 20% depending on the average cardiac dose.

A study of 35,000 patients treated with breast radiation therapy in Sweden and Denmark between 1976 and 2006 showed that the increase in cardiovascular mortality was also influenced by the lateralization of the breast tumor. In this population, the average dose absorbed by the heart for the group of patients treated on the right was 2.7 Gy, compared to 6.3 Gy for the group treated on the left. The left-to-right ratio was significant for myocardial infarction [1.22 [1.06–1.42]], angina [1.25 [1.05–1.49]], pericarditis [1.61 [1.06–2.43]], and valvular heart disease [1.54 [1.11–2.13] [3,4].

2.9. Pathophysiology

Exposure of the heart to ionizing radiation during radiation therapy for breast cancer increases the subsequent incidence of ischemic heart disease. This increase is proportional to the average dose to the heart, begins a few years after exposure, and continues for at least 20 years [5].

The pathophysiology is based on obliterative endarteritis secondary to radiation-induced arterial wall fibrosis, vascular endothelial dysfunction, chronic inflammation, and accelerated atherosclerosis, presenting a unique and aggressive pathological profile [6]. These lesions differ from classic atherosclerosis because they preferentially affect exposed segments, often in the absence of lipid plaque.

Cytokine-mediated fibrosis [TGF-β, IL-6] leads to concentric thickening of the vessel wall and a reduction in luminal diameter, primarily affecting the ostial and proximal segments.

The LAD is most exposed to the radiation beam during left breast irradiation, where the average cardiac dose frequently exceeds 5 Gy, despite intensity-modulated radiation therapy techniques.

Several recent studies confirm the correlation between the average dose received by the heart and the risk of cardiovascular events. Darby *et al.,* [N Engl J Med 2013] demonstrated that the risk of coronary artery disease increases by 7.4% for each additional Gy of average cardiac dose.

2.10. Clinical Features

Patients who have undergone radiation therapy to the left breast are at greater risk of coronary stenosis compared to those who have undergone radiation therapy to the right breast [7].

The LAD is a critical vascular structure, accounting for more than half of the heart's blood supply and thus playing a vital role in myocardial perfusion. Furthermore, it is located on the anterior and lateral surfaces of the myocardium, often receiving higher radiation doses [8]. Patients with this condition often have few or no cardiovascular risk factors.
The lesions are characterized by:

- Focal ostial or proximal involvement,
- A non-atheromatous appearance [absence of diffuse plaques or calcifications],
- A slow but progressive course.

In our observation, isolated ostial involvement of the LAD fits this presentation perfectly.

2.11. Clinical and diagnostic data

Radiation-induced heart damage has been recognized since the 1960s, but its true incidence is likely underestimated due to the long delay between exposure and the onset of symptoms.

Radiation-induced coronary lesions are most often ostial or proximal. The average time to onset of radiation-induced coronary disease is 10 to 20 years, but earlier cases have been reported, particularly in association with anthracycline-based chemotherapy.

The diagnosis is based on imaging: angiography reveals ostial or proximal stenoses, often solitary and non-calcified, in contrast to the usual diffuse atherosclerosis.

Our patient's presentation is atypical due to the early onset [age 8] and the isolated, non-atheromatous involvement of the LAD ostium. This profile has already been reported in recent observations, suggesting individual susceptibility or a synergistic effect with previously administered anthracyclines.

2.12. Literature data:

Darby *et al.,* [NEJM, 2013] demonstrated that the risk of a coronary event increases by 7.4% per Gy of average cardiac dose [5].

Lee *et al.,* [JACC CardioOncol, 2025] confirm that the LAD is the most commonly affected artery following left breast radiation therapy [8].

Takahashi *et al.,* [Front Cardiovasc Med, 2024] report a proximal IMA stenosis that occurred 9 years after radiation, with characteristics similar to our case.

2.13. Management

Percutaneous revascularization is the standard of care for accessible lesions. However, radiation-induced stenoses are associated with an increased risk of restenosis and fibrosis, warranting close follow-up.

Secondary prevention relies on optimal control of risk factors and comprehensive medical therapy [antiplatelet agents, statins, ACE inhibitors/ARBs, beta-blockers].

2.14. Prevention and Screening

The risk of cardiotoxicity depends on the mean cardiac dose [MCD]. According to Darby *et al.,* [N Engl J Med, 2013], each additional Gy increases the risk of a coronary event by 7.4%.

Modern techniques [conformal radiation therapy, accelerated partial breast irradiation [9], IMRT, respiratory gating, deep-breath-hold [10]] reduce the MCD to less than 2 Gy, thereby limiting future risk [11].

A combination of medical prevention and therapy with physical protection measures may be essential for achieving significant cardiac protection [12].

Advances in radiation technology have significantly reduced cardiovascular radiation dose and volume exposure [13].

Early studies in NSCLC have suggested LAD V15 ≥ 10% as a predictor of major adverse cardiac events [MACE] and all-cause mortality [14–17].

A study on reducing the dose to the LAD using the Deep Inspiration Breath Hold [DIBH] technique showed that the average and maximum doses to the LAD are significantly reduced with DIBH compared to standard radiation therapy [10].

Early detection relies on regular cardiac monitoring for all patients who have received thoracic radiation:

- Symptom assessment
- Baseline ECG and echocardiogram, followed by repeat testing every 2 to 3 years;
- Stress testing or perfusion imaging if symptoms are present;
- Noninvasive coronary evaluation [coronary CT or scintigraphy] for high-risk patients [>5 Gy CTC], or if symptoms are present or ischemia is suspected.
- Strict secondary prevention [lipid control, blood pressure control, antiplatelet therapy].

CONCLUSION

Cardiovascular disease represents a major long-term complication of cancer treatment. This case highlights the occurrence of severe, isolated, non-atherosclerotic coronary disease several years after left breast radiotherapy. Ostial LAD stenosis is a rare but characteristic complication. Prevention relies on optimized radiotherapy techniques and long-term cardio-oncology follow-up. Multidisciplinary collaboration is essential for early detection and management.

Conflicts of Interest: The authors declare no conflicts of interest.

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