A 56-year-old woman was referred for investigation of worsening signs of heart failure. She had been treated with radiotherapy and chemotherapy for Hodgkin disease 19 years earlier. Because the patient had disease localized to the mediastinum, she had received mantle radiation, which is focused on the sternal area. She had also undergone MOPP chemotherapy (mechlorethamine, vincristine, procarbazine, and prednisone), an anthracycline-free regime. Eighteen months earlier, she had presented with exertional dyspnea. At that time, coronary angiography demonstrated ostial stenoses of the left and right coronary arteries (Figure, A and B; arrows), which were treated with percutaneous intervention and stenting. Repeat coronary angiography revealed that both stents were widely patent without any other significant coronary stenoses. Echocardiography revealed basal hypokinesia, an ejection fraction of 35%, and normal valvular function. Cardiac magnetic resonance imaging (CMR) was ordered to investigate the cause of her heart failure.

![Figure](image)

**Figure.** A and B, Initial coronary angiography demonstrating ostial stenoses of the right and left coronary arteries (arrows). C and D, Late inversion-recovery contrast-enhanced CMR images acquired 10 minutes after gadolinium injection in the 2-chamber (A) and short-axis (B) views demonstrate patchy basal fibrosis, not corresponding with a coronary territory.

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From the Institut Cardiovasculaire Paris Sud, Hôpital Jacques Cartier, 6 avenue du Noyer Lambert, 91 300 Massy, France.

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Correspondence to Dr Darach O h-Icí, MB, CMR Department, Institut Cardiovasculaire Paris Sud, Hôpital Jacques Cartier, 6 avenue du Noyer Lambert, 91 300 Massy, France. E-mail darachohici@gmail.com

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CMR revealed a nondilated hypokinetic left ventricle with akinesis of the anterior, septal, and lateral walls of the basal left ventricle and akinesis of the basal right ventricle. The ejection fraction was confirmed at 35%, predominantly the result of the basal akinesis. There were no significant valvular lesions on MRI. There were pleural effusions and a moderate pericardial effusion (Movies 1 and 2) without any evidence of pericardial thickening or constrictive pericardial disease. Delayed enhancement images demonstrated basal myocardial fibrosis (Figure, C and D; arrows). This fibrosis did not correspond to a coronary territory or to a pattern consistent with other cardiomyopathies such as amyloidosis or sarcoidosis. There was no evidence of myocardial infarction. She was diagnosed with radiation-induced cardiomyopathy. Her medical treatment was optimized and she was discharged from hospital.

As oncology treatments improve with longer survival rates, radiation-induced heart disease is likely to be increasingly recognized. Mediastinal radiotherapy significantly increases the risks of myocardial infarction, angina pectoris, heart failure, and valvular disorders. The coronary ostia are typically affected. Radiation leads to microvascular insufficiency and ischemia, resulting in diffuse and patchy interstitial myocardial fibrosis. Myocardial fibrosis is usually seen at radiation doses above 30 Gy and is often asymptomatic. A number of studies using single-photon emission computed tomography have found significantly increased rates of perfusion defects in patients who have undergone mediastinal radiation compared with control subjects. These defects have correlated well with the irradiated areas of the left ventricle.

Right ventricular abnormalities have not been described; however, single-photon emission computed tomography may not have the same sensitivity as CMR for assessing the right ventricle.

CMR patterns of delayed enhancement can help distinguish between the causes of cardiomyopathy, including ischemia, valvular, myocardial, and pericardial diseases. This information can be used before considering revascularization in patients with potential nonischemic causes of heart failure.

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None.

References

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Darach O h-Icí and Jérôme Garot

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