



Case Report

A case of chemoradiotherapy-associated cardiotoxicity that developed into acute heart failure with progressive subendocardial fibrosis



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ABSTRACT

We describe the case of a 61-year-old Japanese woman who developed acute heart failure 5 years after chemoradiotherapy for breast cancer. The patient received less than the cardiotoxic dose of docetaxel, epirubicin, cyclophosphamide, and fluorouracil and experienced no cardiovascular complications in the 5 years between the onset of chemoradiotherapy and the onset of acute heart failure. Cardiac catheterization was performed and elevation of end diastolic pressure of both ventricles was observed. Endomyocardial biopsy showed progressive replacement fibrosis in the subendocardium. Normal thickness of the right endocardium is <20 μm . Surprisingly, our patient had a fibrous subendocardium that was 100–200 μm thick. Ultrastructural abnormalities similar to those observed in anthracycline cardiotoxicity were evident on electron micrographs. This case report demonstrates the unique pathophysiology of heart failure in a patient who received less than the cardiotoxic dose of antineoplastic agents. Recent protocols have decreased the dosage of cardiotoxic agents; however, even these reduced doses might not be safe for all Japanese individuals and may cause subclinical cardiovascular damage and late-onset heart failure. Clinicians should monitor cancer survivors carefully, even if antineoplastic agents were administered under the cardiotoxic dose.

<Learning objective: Intensive chemotherapy is commonly used to treat cancer patients. Recent protocols have decreased the dose of cardiotoxic agents; however, even these reduced doses might not be safe and may cause subclinical cardiovascular damage and late-onset heart failure. Clinicians should monitor cancer survivors carefully, even if antineoplastic agents were administered under the toxic dose.>

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Introduction

In Japan, the incidence of breast cancer has increased in recent years, but the survival of patients with newly diagnosed breast cancer has improved [1]. With advancements in chemotherapy regimens, mortality rates are decreasing by 2.3% annually [2], and the 5-year survival rate for advanced breast cancer has increased to 30% [3]. In the past, cardiovascular complications were rare in breast cancer patients because the lifespan of a patient with advanced cancer was too short for the issue to manifest. However, more recent reports indicate that there are now a considerable

number of cancer survivors who develop chemotherapy-related cardiac dysfunction and thus are under periodic observation by a cardiologist [4]. Here we describe a breast cancer patient who developed acute heart failure (HF) 5 years after chemoradiotherapy. We observed unique subendocardial fibrosis in the endomyocardial biopsy despite the fact that all antineoplastic agents had been delivered under the cardiotoxic dose.

Case report

A 61-year-old Japanese woman experienced shortness of breath in May 2012, 5 years after she had undergone a partial mastectomy and received subsequent chemoradiotherapy for breast cancer. Three weeks later, she developed dyspnea, general fatigue, and loss of appetite, and she was therefore admitted to our hospital with orthopnea. A chest radiograph revealed cardiomegaly, pulmonary

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congestion, and bilateral pleural effusion. At the time of hospital admission, the patient's weight had increased by 5 kg since her last regular medical check-up 4 weeks earlier. Physical examination showed moist rale in bilateral lower lung fields, and the patient was diagnosed with acute pulmonary edema.

The patient had been diagnosed with right breast cancer without metastasis in October 2006. The tumor was 5.3 cm in size and located in the lower medial region of the right chest (T3N1M0, stage IIIa). Initial chemotherapy with docetaxel (cumulative dose, 300 mg/m²) proved ineffective. After cardiovascular screening, she

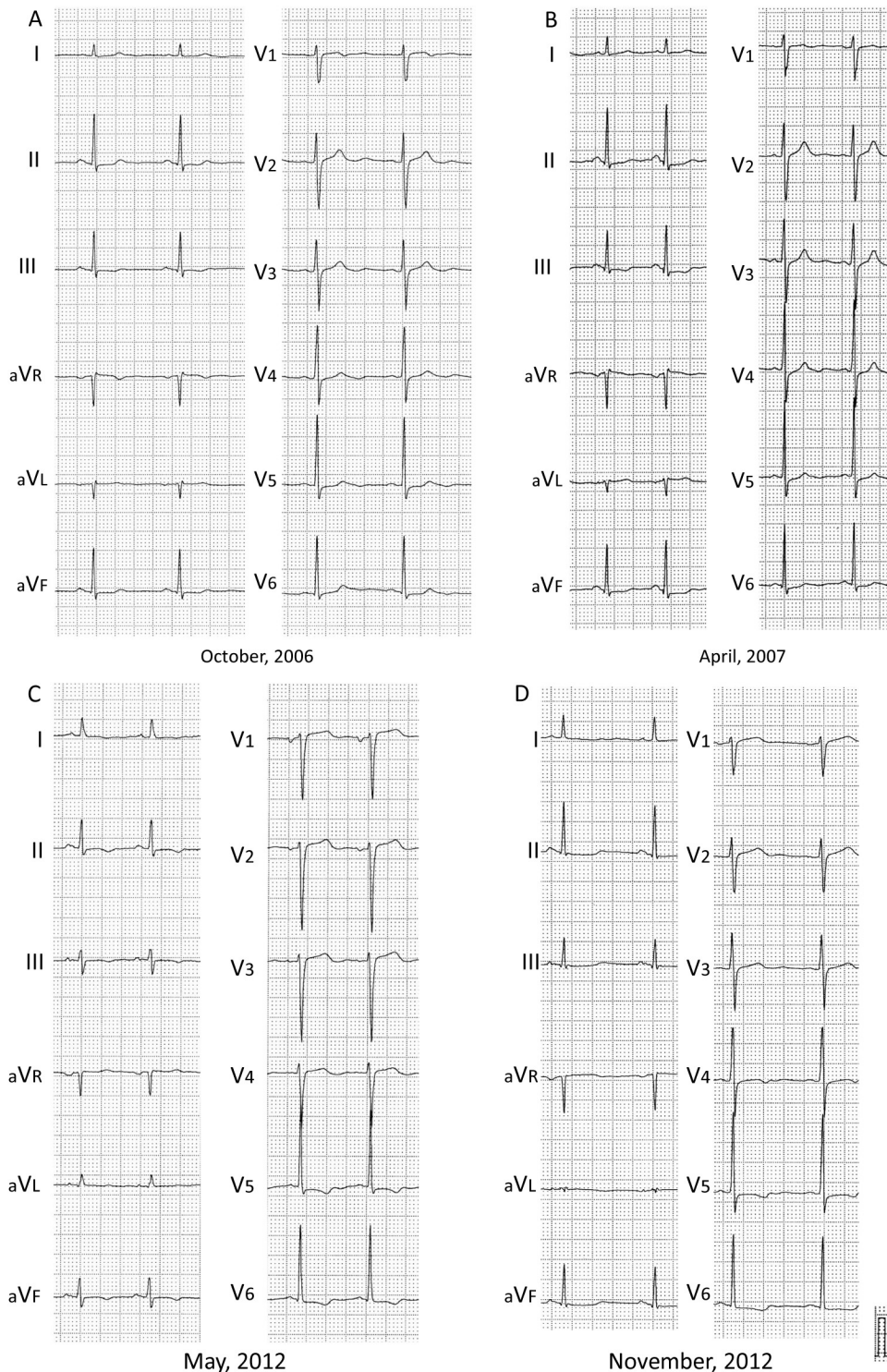


Fig. 1. Electrocardiograms before chemotherapy (October, 2006) (A), after chemotherapy (April, 2007) (B), at the time of hospital admission due to heart failure (May, 2012) (C), and 6 months later (November, 2012) (D) (10 mm/mV). Slight and non-specific ST segment depression was observed in (A) and (B), but there were no significant differences. Electrocardiogram at the time of heart failure admission showed diminished QRS amplitude, poor R waves in precordial leads, and ST segment depression associated with T-wave inversion in V_{5,6} (C). Six months later, the QRS amplitude almost normalized in precordial leads, but ST segment depression remained in II, III, aVF, V_{5,6} (D).

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