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Case Report

Assessment of myocardial fibrosis using T1-mapping and extracellular volume measurement on cardiac magnetic resonance imaging for the diagnosis of radiation-induced cardiomyopathy

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ABSTRACT

Radiation-induced heart disease (RIHD) is a serious side effect of thoracic radiation therapy (RT) and is associated with significant morbidity and mortality. Radiation-induced cardiomyopathy (RICM) is one of the manifestations of RIHD, which represents with left ventricular (LV) systolic and diastolic dysfunction due to myocardial fibrosis. Although the diagnosis of RIHD is challenging and is generally an exclusion diagnosis, multimodality imaging including echocardiography, cardiac computed tomography and cardiac magnetic resonance (CMR) imaging could help the diagnosis. Herein, we report a case of 70-years-old male, who had been treated with chemo-radiation therapy for early esophageal cancer, was suffered from medically refractory heart failure due to severely reduced LV systolic function and constrictive pericarditis 8 years after chemo-radiation therapy. Although no gadolinium-enhancement (LGE) was detected on CMR, T1 mapping depicted increased extracellular matrix volumes of 45%, which suggested global myocardial fibrosis. Histopathological analysis by endomyocardial biopsy (EBM) revealed marked degeneration of myocytes and interstitial fibrosis, while vacuolation in myocytes which is characteristics of chemotherapy induced cardiomyopathy was not specific by electron microscopy. Therefore, we diagnosed that the present case was likely to the RICM.
<**Learning objective:** RICM is characterized by inflammation followed by the development of a diffuse, patchy interstitial fibrosis of the myocardium, which is usually obtained either by EBM or at autopsy. Native and post-contrast T1-mapping by CMR enables to estimate extracellular volume (ECV), which is believed to be increased as a result of diffuse myocardial fibrosis. The assessment of myocardial fibrosis using ECV should be useful for early detection of myocardial damage due to RT, and which probably taking place of EBM.>

Introduction

With the recent improvement in survival rate of thoracic malignancies with chemo-radiation therapy, radiation-induced heart disease (RIHD) has come to be recognized. Radiation-induced cardiomyopathy (RICM) is one of the manifestations of RIHD, which presents with left ventricular (LV) systolic or diastolic dysfunction due to myocardial fibrosis. We experienced medically refractory heart failure (HF) due to severely reduced LV systolic function suggesting RICM, and T1 mapping by cardiac magnetic resonance (CMR) image was useful to depict myocardial properties.

Case presentation

A 70-year-old man was admitted to Tottori University Hospital due to exertional dyspnea and bilateral leg edema.

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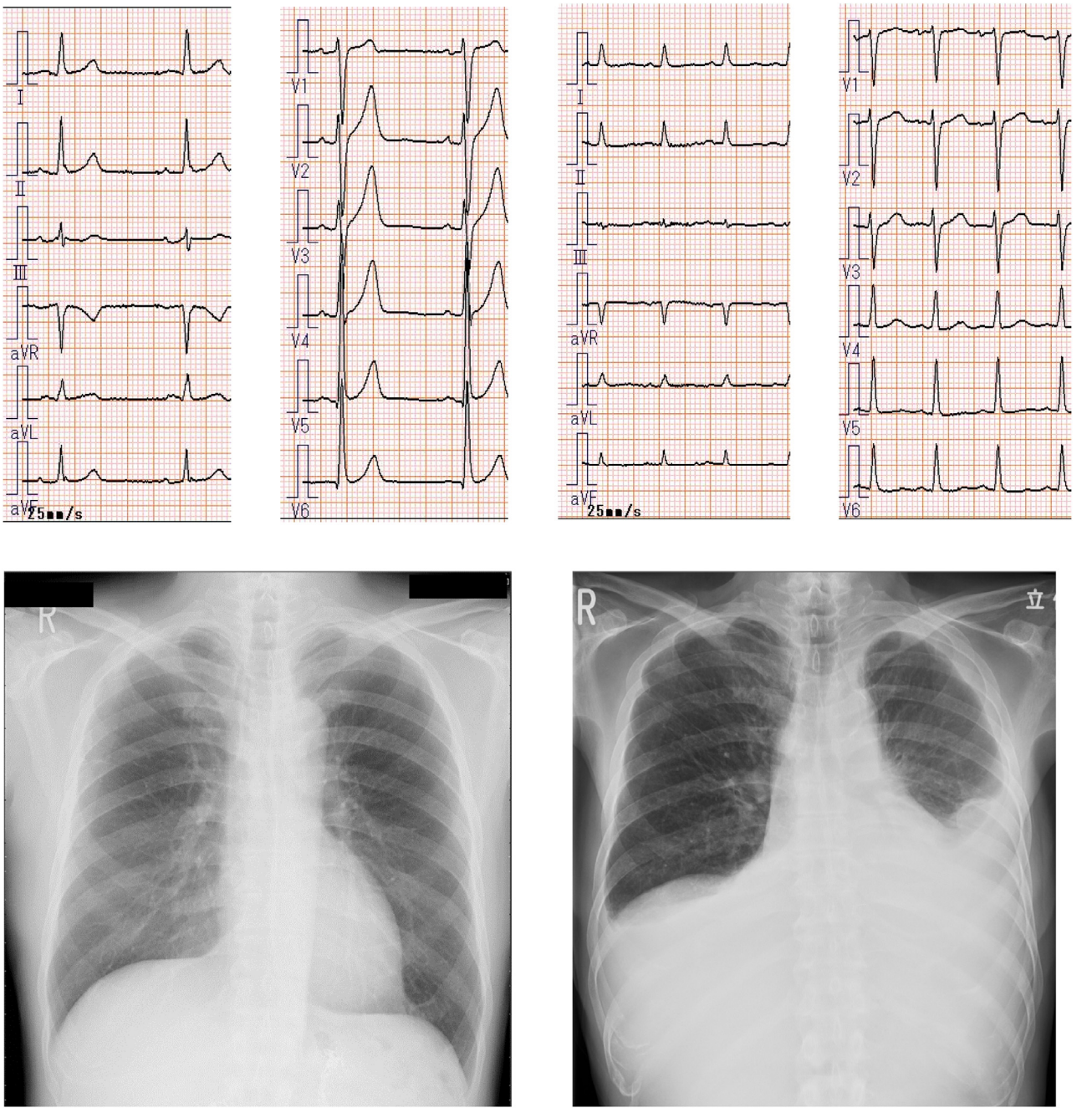

Fig. 1. (Upper) Electrocardiogram (ECG) and (Lower) chest X-ray before and after chemo-radiation therapy. Before chemo-radiation therapy (A), both ECG and chest X-ray showed no definite abnormality. On this admission (B), ECG showed low voltage in the extremity leads and poor R progression in the chest leads. Cardiomegaly and bilateral pleural effusion (left dominant) were revealed in the chest X-ray.

He had been treated with chemo-radiation therapy for early esophageal cancer (Stage 1; cT1N0M0) 8 years earlier. He underwent mediastinal external beam radiation with a total dose of 60 Gy/30 fr, with 48 Gy and 12 Gy using anterior–posterior fields and parallel–oblique fields, respectively. Docetaxel and 5-fluorouracil (5-FU) were administered and continued as an adjuvant chemotherapy for two years, then he achieved remission. He had no previous medical history of any cardiac disease. However, left pleural effusion was increased 4 years after chemo-radiation therapy suggesting radiation pleuritis. Despite taking diuretics, exertional dyspnea and bilateral leg edema developed and gradually worsened. He was referred to our cardiology department for the first time because he was suspected of HF. On admission, blood pressure was 141/69 mmHg and heart rate was 129 beats/min. He had hypoxemia of 95% of SpO_2 (room air) and jugular vein was distended. Blood examination revealed mild renal dysfunction (blood urea nitrogen of 27.3 mg/dl, creatinine, 1.07 mg/dl, estimated glomerular filtration rate, 51.6 ml/min/1.73m^2) and mildly elevated brain natriuretic peptide of 105 pg/ml. A 12-lead

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