An 80-year-old woman was referred to hospital because of complaints of dyspnea at rest and progressive weakness of the leg muscles. She had been experiencing progressive fatigue after walking short distances and dyspnea on exertion over the past 1 year. She had a past history of hypertension, dyslipidemia, diabetes mellitus, and angina pectoris. Physical examination at admission showed peripheral edema and slight weakness of the proximal skeletal muscles. Arterial pressure, heart rate, and temperature were 133/63 mmHg, 74 beats/min, and 37.2°C, respectively. On auscultation, coarse crackles were heard over all lung fields, and a grade III ejection systolic murmur at the cardiac apex. Chest radiograph indicated pulmonary congestion and cardiomegaly (cardiothoracic ratio, 62.8%). Resting electrocardiogram showed sinus rhythm, with left anterior hemiblock, and first-degree atrioventricular block. Cardiac enzymes (troponin, creatine phosphokinase, and creatine phosphokinase-MB) were increased, and B-type natriuretic peptide was elevated, at 482 pg/ml. Echocardiography indicated preserved left ventricular ejection fraction (50%). The patient underwent conventional heart failure therapy, but collapsed suddenly 2 days after admission. A nurse immediately started chest compressions, and cardiopulmonary resuscitation was performed for approximately 2 min. Primary arrhythmia documented on the monitor was asystole due to complete atrioventricular block. Permanent pacemaker was therefore implanted after rhythm was first restored with a temporary pacemaker. A few days after implantation of the permanent pacemaker, left femoral muscle biopsy indicated inflammatory cellular infiltration; on the basis of this, polymyositis was diagnosed. To rule out malignancy, heparin-loading 18F-fluorodeoxyglucose (FDG) positron emission tomography-computed tomography showing cardiac FDG uptake predominantly in the lateral wall of the left ventricle (arrows).
treated with prednisolone and i.v. immunoglobulin G, the systemic skeletal muscle weakness gradually progressed, and she died of respiratory failure on the 66th day of hospitalization. Autopsy showed increased heart weight of 350 g (Figure 2A). On microscopy, replacement fibrosis of cardiac muscle fibers in the interventricular septum was seen (Figure 2B). The inflammatory cell infiltration, however, was predominantly in the lateral region of the left ventricle, where the FDG uptake had been mainly observed (Figure 2C). On the basis of these findings, the patient was diagnosed with polymyositis combined with myocarditis.

Polymyositis is a chronic inflammatory muscle disease characterized by skeletal muscle weakness. Cardiovascular manifestations are reported to be the most common cause of death in polymyositis.1,2 Electrocardiographic abnormalities observed in polymyositis are conduction abnormalities, including right bundle-branch block, left anterior hemiblock, and atrioventricular block, which are associated with fatal outcome.3,4 No correlation has been found between the overall activity of the disease and cardiac involvement.5,6 In the present case, we speculate that chronic inflammation of the interventricular septum led to fibrosis, which resulted in complete atrioventricular block. FDG-PET can visualize inflammatory lesions of skeletal muscles.7,8 Furthermore, FDG-PET is a promising modality for detecting active lesions in various inflammatory cardiovascular diseases, including the cardiac involvement in sarcoidosis, myocarditis, and inflammatory vascular diseases.9 Proving the presence of inflammation in the myocardium on FDG-PET/computed tomography (FDG-PET/CT), however, is occasionally difficult because of the shift to glucose metabolism in the myocardium. In this case, we performed FDG-PET following i.v. injection of heparin (50 IU/kg) 15 min before FDG, with fasting for 18 h before FDG-PET/CT; this technique has been reported to suppress physiological FDG uptake.10 When the uptake of FDG is diffuse, it is considered to be non-specific; in the present patient, however, there was inhomogeneous uptake, and the FDG uptake in the lateral left ventricular wall corresponded to the pathological findings. In conclusion, FDG-PET/CT may have the potential to diagnose cardiac involvement in polymyositis.

References