A 66-year-old woman was resuscitated from a cardiac arrest after completing 1 cycle of cardiopulmonary resuscitation (Supplementary Figure 1). Echocardiography showed severe global akinesia and small pericardial effusion. Coronary angiography was normal. Endomyocardial biopsy was performed. Myocardial pathology confirmed her diagnosis of giant cell myocarditis (GCM) (Figure 1). As a treatment for GCM, she received anti-thymocyte globulin (ATG) followed by cyclosporin A and everolimus (Supplementary Figure 2).

Follow-up pathological examination showed resolving myocarditis on the 12th hospital day (Figure 2) at 5 weeks after starting treatment. Histological confirmation is essential for verifying GCM in myocarditis. Observations in experimental GCM on Lewis rate model suggest that autoimmune mechanisms mediated by CD4+ T lymphocytes are responsible for myocarditis.
Histologic Response for Giant Cell Myocarditis

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Conflict of Interest
The authors have no financial conflicts of interest.

Author Contributions
Conceptualization: Cho HJ; Data curation: Choi YJ, Cho HJ; Supervision: Cho HJ; Visualization: Choi YJ; Writing - original draft: Choi YJ, Cho HJ; Writing - review & editing: Choi YJ, Cho HJ.

for the pathogenesis of GCM. In acute phase, T helper cell type 1 (Th1) is dominant, which may lead to inflammation at the induction of myocarditis. ATG therapy induces a profound decrease in Th1 cell counts. Early ATG administered seems to play a key role in alleviating the fulminant inflammation of the heart and resulted in a better clinical course.

SUPPLEMENTARY MATERIALS

Supplementary Figure 1
The electrocardiogram (A) and chest x-ray (B) on the first day to the hospital. (A) Sinus rhythm with the low voltage of QRS complexes in the precordial and limb leads. There are no significant ST-segment changes. (B) The chest radiograph shows cardiomegaly with bilateral diffuse infiltrates which suggest cardiogenic pulmonary edema.

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Supplementary Figure 2
Summary for administration of immunosuppressive drugs.

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