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Clinical and Histological Response to Immunosuppressive Therapy in Giant Cell Myocarditis

You-Jung Choi, MD, and Hyun-Jai Cho, MD, PhD

Division of Cardiology, Department of Internal Medicine, Seoul National University Hospital, Seoul, Korea

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Correspondence to
Hyun-Jai Cho, MD, PhD
Division of Cardiology, Department of Internal Medicine, Seoul National University Hospital, 101, Daehak-ro, Jongno-gu, Seoul 03080, Korea.
E-mail: hyunjaicho@snu.ac.kr
hyunjaicho@gmail.com

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).[1] 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.[2] Observations in experimental GCM on Lewis rate model suggest that autoimmune mechanisms medicated by $CD4^+$ T lymphocytes are responsible

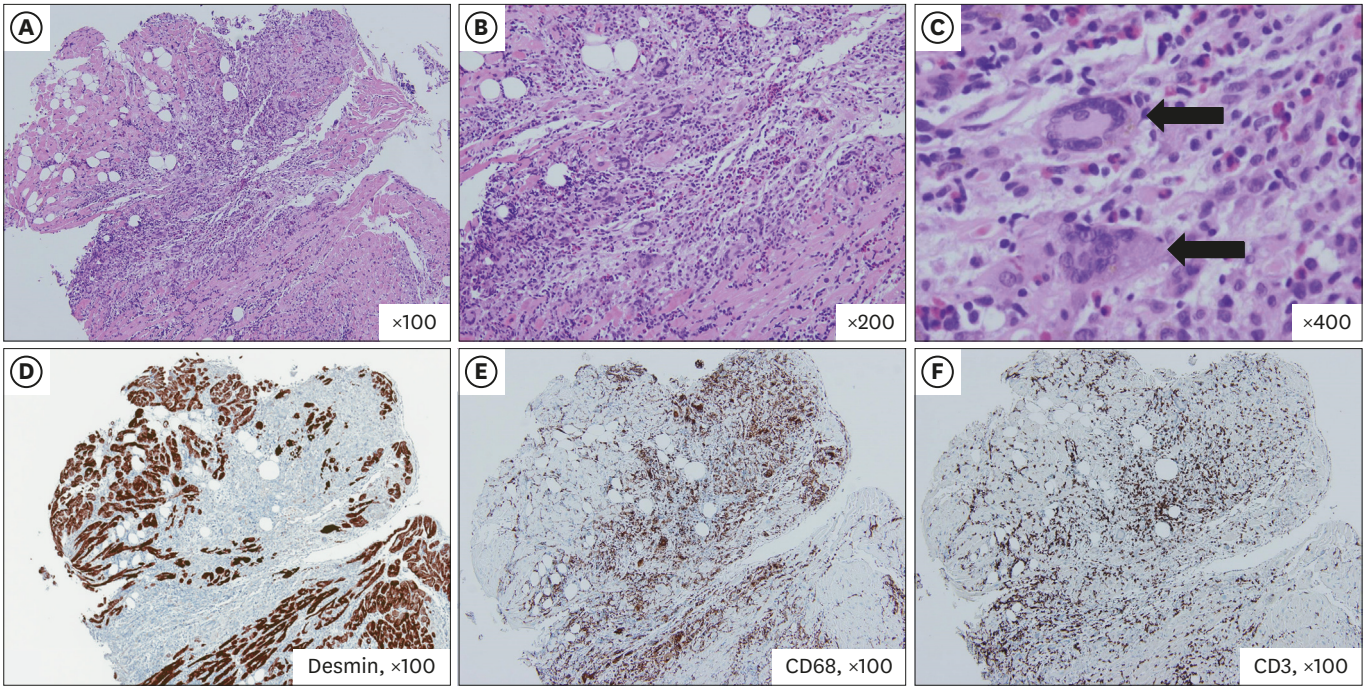

Figure 1. Histological findings on endomyocardial biopsy. (A) Diffuse geographic myocarditis with severe necrotic change and inflammatory infiltrate (H&E, ×100). (B) Numerous multinucleated giant cells in nongranulomatous background. (C) Collection of inflammatory cells including variable numbers of giant cells (arrows), histiocyte, T-lymphocyte, and eosinophils. (D) The area with necrotic change and severe inflammation showed negative of desmin. (E) Histiocyte infiltration in myocardium (CD68 staining). (F) T-lymphocyte infiltrate in myocardium (CD3, Pan-T cell marker staining).
H&E = hematoxylin and eosin.

ORCID iDs
You-Jung Choi
https://orcid.org/0000-0003-3512-855X
Hyun-Jai Cho
https://orcid.org/0000-0002-2779-4037

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.

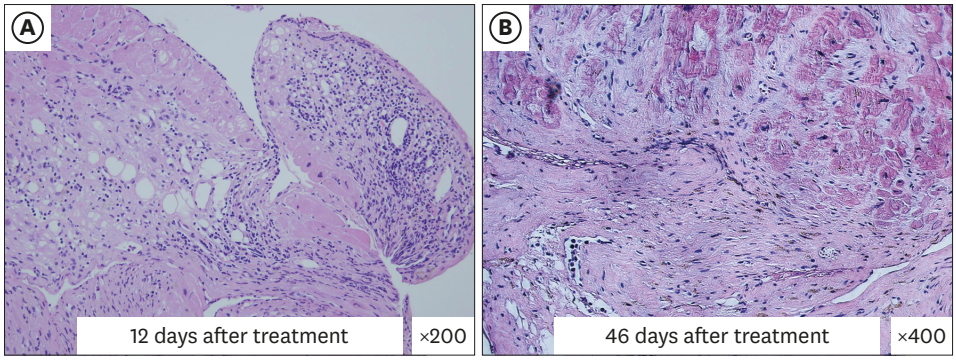

Figure 2. Follow-up endomyocardial biopsy. (A) On 12th day from starting immunosuppressive therapy, myocardial biopsy revealed a marked reduction of the inflammatory infiltrate and the absence of giant cells. The fibrotic and fatty change are seen in the area, where there was necrotic change before treatment (H&E, ×200). (B) On 46th day, massive granulation tissue and fatty change without active myocardial damage was observed (H&E, ×400).
H&E = hematoxylin and eosin.

for the pathogenesis of GCM.[3] 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.[4] 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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