

Complete recovery of a patient with cardiogenic shock due to parvovirus B19 fulminant myocarditis after treatment with extracorporeal membrane oxygenation and intravenous immunoglobulin

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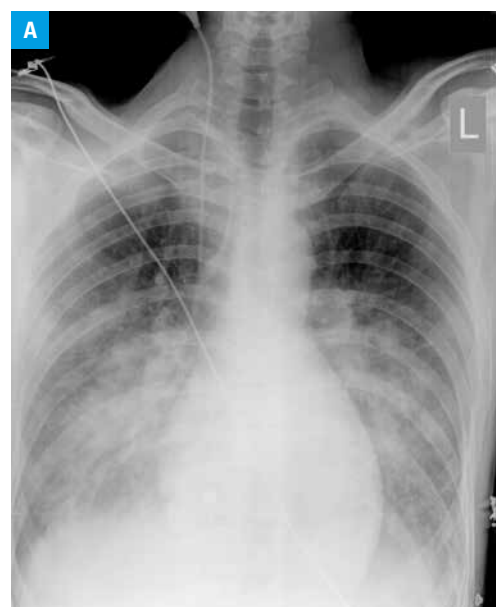
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Fulminant myocarditis (FM) is a rare subtype of acute myocarditis characterized by a rapid onset, dramatic course, and poor outcome.¹ Epidemiological data are scarce but only 27 of 1098 patients (2.5%) with more than 14 infiltrating lymphocytes in cardiac biopsy samples met the criteria of FM in the Marburg Myocarditis Registry.² The World Heart Federation consensus introduced a cut-off value of 50 infiltrating cells/mm² or higher as a diagnostic criterion.³ The etiology is noninfectious and infectious with predominance of cardiotropic viruses. Mechanical circulatory support including intraaortic balloon pump (IABP), extracorporeal membrane oxygenation (ECMO), and ventricular assist devices has become the cornerstone of treatment for circulatory collapse.⁴ A treatment of FM has not been precisely defined. Intravenous immunoglobulins are used in biopsy-confirmed viral myocarditis and immunosuppression in autoimmune reactive myocarditis.⁵

A previously fit and well 30-year-old physical worker was admitted to our intensive care unit in a critical state because of acute chest discomfort and cardiopulmonary collapse of an unknown origin. Initial electrocardiography showed ST-segment elevation in leads V₁–V₃ and deep ST-segment depression in leads V₅–V₆. The patient was referred for urgent coronary angiography but it did not reveal any abnormalities. Because of persistent hypotension of 80/40 mmHg, IABP was

inserted. Chest X-ray showed overt congestion (FIGURE 1A). The initial transthoracic echocardiography revealed extreme dysfunction of moderately dilated left ventricle (left ventricular ejection fraction [LVEF], 10%; left ventricular end-diastolic diameter [LVEDd], 61 mm). Right ventricular systolic function was normal and no signs of acute pulmonary embolism, cardiac tamponade, or valvular pathology were noted (FIGURE 1B). Baseline



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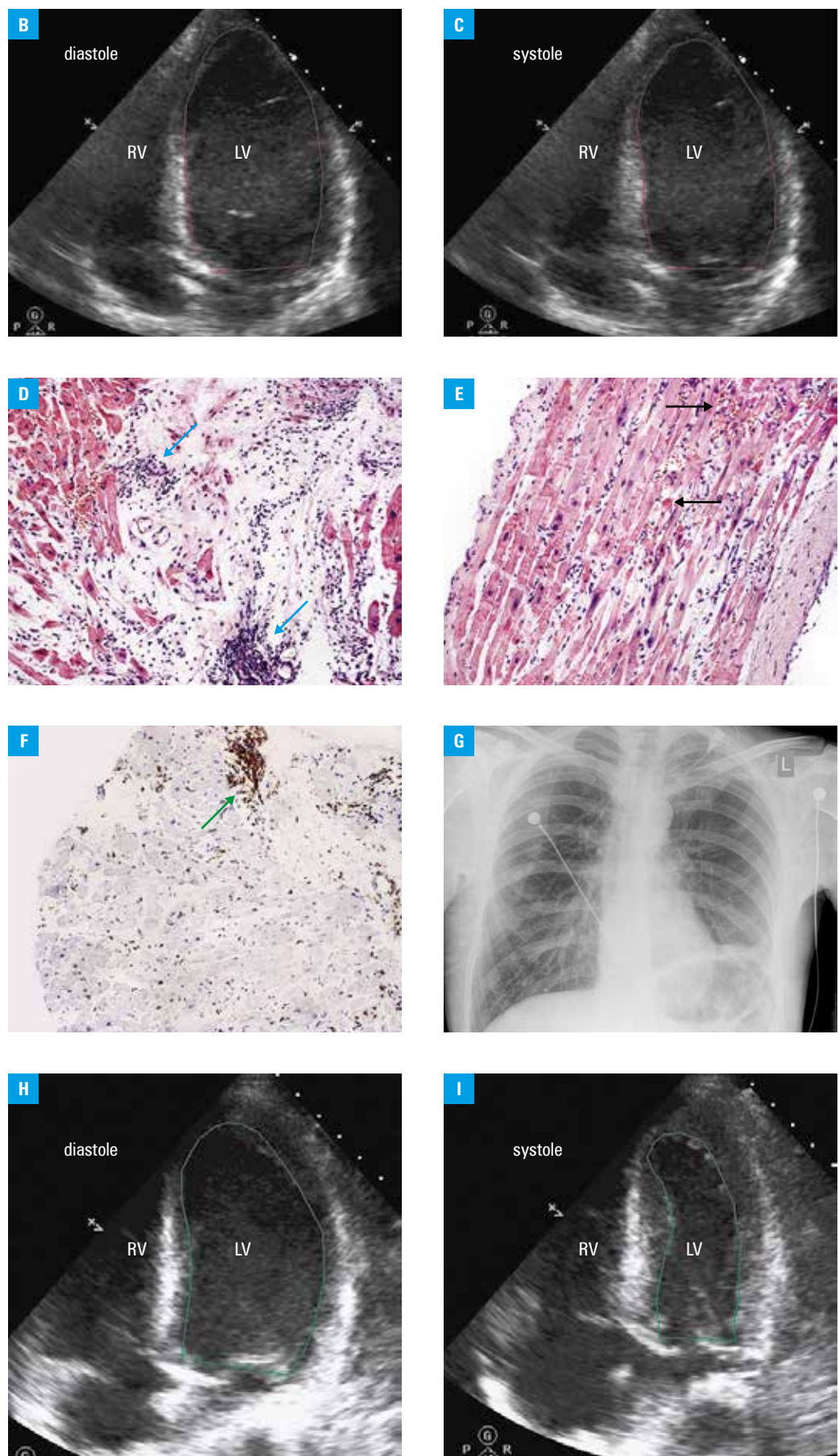


FIGURE 1 **A** – initial A-P chest X-ray with overt pulmonary congestion and enlarged cardiac silhouette; **BC** – admission transthoracic echocardiogram (TTE) documenting severe left ventricular (LV) dysfunction (LV ejection fraction [LVEF], 10%); **D** – LV endomyocardial biopsy sample (hematoxylin and eosin staining) showing lymphocytic infiltrates (blue arrows); **E** – destruction of cardiomyocytes and reactive fibrosis (black arrows), **F** – immunohistochemistry: CD3⁺ T cell infiltrates (green arrow); **G** – a marked decrease of pulmonary congestion and reduction of the cardiac silhouette size; **HI** – TTE on day 9 of hospitalization: near-to-normal LV contractility (EF, 50%)

laboratory tests were as follows: white blood cell count, 23 G/l; C-reactive protein (CRP), 120 mg/l (upper normal limit [UNL], <5); alanine transaminase, 106 U/l (UNL, <41); aspartate transaminase, 251 U/l (UNL, <40); international normalized ratio, 1.92 (UNL, 0.85–1.15); creatinine kinase (CK), 4583 U/l (UNL, <190); CK-MB, 126 U/l (UNL, <24); and high-sensitivity troponin T (hs-TnT), 4.03 ng/ml (UNL, <0.014). IABP was replaced by the venoarterial ECMO system. A working diagnosis of fulminant myocarditis was made, and the patient was scheduled for urgent endomyocardial biopsy, which was performed under fluoroscopic and echocardiographic guidance. Hematoxyline and eosin staining showed infiltration of mononuclear cells (**FIGURE 1D**, arrows), destruction of cardiomyocytes, and reactive fibrosis (**FIGURE 1E**, arrows). Immunohistochemistry indicated the infiltrating cells to be CD3⁺ T cells and CD68⁺ macrophages (**FIGURE 1F**, arrow). Histopathological and immunohistochemistry studies confirmed the diagnosis of myocarditis. Based on real-time polymerase chain reaction and in situ hybridization, the offending factor was diagnosed to be parvovirus B19. An intravenous immunoglobulin treatment with Pentaglobin[®] was administered for 3 days. The patient's clinical status and chest X-ray results were gradually improving (**FIGURE 1G**). On day 16, ECMO was safely removed. On day 28, the patient was transferred to a cardiac unit, and after 4 days, he was discharged home in a good clinical condition. Final echocardiography revealed normalization of the left ventricular cavity (LVEDd, 52 mm; LVESd, 32 mm) and improvement of LVEF to 50% (**FIGURE 1H**). The remaining parameters also normalized (CRP levels, 3.8 mg/l; CK, 78 U/l; CK-MB, 26 U/l; and hs-TnT, 0.028 ng/ml).

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