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Biventricular fulminant myocarditis: A case report

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Abstract

Fulminant myocarditis is associated with left ventricular (LV) dysfunction and subsequent hemodynamic collapse, but a good prognosis can be expected with aggressive LV support. We report a case of fulminant myocarditis, which involved not only the LV but also the right ventricle (RV). A 64-year-old man was admitted with general fatigue and appetite loss. An electrocardiography showed ST elevations in chest leads, but coronary angiography showed no stenosis. Intraaortic balloon pumping did not improve the shock state; the administration of percutaneous cardiopulmonary support was suggested but declined. The patient died nine hours after admission to our hospital. An autopsy showed lymphocyte infiltration in both LV and RV.

Keywords

biventricular; fulminant; left ventricle; myocarditis; right ventricle

Introduction

Fulminant myocarditis is characterized by hemodynamic instability due to left ventricular (LV) dysfunction, but can be cured with a good long-term prognosis when aggressive hemodynamic support is appropriately applied [1,2]. We experienced a fatal case of fulminant myocarditis, in which not only the LV but also the right ventricular (RV) involvement was confirmed by autopsy.

Case Report

A 64-year-old man was admitted to our hospital because of general fatigue and appetite loss. The patient had been well until a week before admission, when general malaise and appetite loss developed. Three days later, back pain and shoulder pain started, and he had difficulty moving or standing. On the day of admission, his colleague found him struggling at home and called an ambulance. His past medical history was unremarkable other than atopic dermatitis. He did not take any medication on admission. There was no family member who had cardiovascular diseases. On physical examination, he seemed to be exhausted but clear. The blood pressure was 103/93 mmHg, the pulse was 86 beats per minute and regular, the temperature was 36.5°C, and the oxygen saturation was 97% while he was breathing ambient air. An electrocardiography showed sinus rhythm with a heart rate of 91 beats per minute, ST-segment elevations and abnormal Q waves in leads V_1 to V_5 (Figure 1A). A chest radiograph showed cardiomegaly with a cardiothoracic ratio of 63% without pulmonary congestion or pleural effusion (Figure 1B). The

complete blood counts were normal, but liver and renal function tests were impaired. The level of creatine kinase was increased up to 1,315 U/L with a positive troponin test, and the brain natriuretic peptide level was 1,207 pg/mL. Echocardiography showed severely reduced LV contractility except for the lateral wall; the LV ejection fraction was estimated to be 10 to 15% on visual assessment. Neither valvular disease nor pericardial effusion was detected. The RV wall motion was also reduced without RV enlargement.

Emergency coronary angiography did not show any obstruction or stenosis in the coronary arteries. During the catheter procedure, however, the blood pressure dropped, leaving the patient in a shock state. Intraaortic balloon pumping (IABP) was introduced. Right-heart catheterization revealed a mean pulmonary artery pressure of 13 mmHg and a cardiac output of 2.68 L/min. His systolic blood pressure was around 80 mmHg with IABP support. The trachea was intubated and mechanical ventilation was started. The blood pressure remained low after the administering of dopamine, dobutamine, adrenaline, and noradrenaline. The use of percutaneous cardiopulmonary support (PCPS) was suggested but declined by his relatives. The patient died nine hours after admission to our hospital.

An autopsy showed that the heart weighted 830 g with significant dilation of both ventricles (Figure 2A). Pathological examination demonstrated neutrophil and lymphocyte infiltration irregularly in almost all the myocardium of the both ventricles, with partial loss of myocardium (Figures 2B to 2D).

Immunohistochemistry showed T-cell lymphocytes were predominant. A short axis image of the heart reconstructed after desmin staining showed severely damaged myocardium in the most part of the RV, the ventricular septum, and anterior and inferior walls of the LV (Figure 2E). A diagnosis of biventricular fulminant myocarditis with lymphocyte infiltration was confirmed.

Discussion

Fulminant myocarditis is a distinct entity from acute (nonfulminant) myocarditis because there are significant differences in clinical presentation, treatment strategy, and outcomes between the two categories[3]. Although the diagnostic criteria of fulminant myocarditis has not been fully established, guidelines for diagnosis and treatment of myocarditis by Japanese Circulation Society Joint Working Groups recommend that fulminant myocarditis should be considered when external circulation support (e.g., IABP, extracorporeal membrane oxygenation, PCPS, and LV assist device) is required to maintain hemodynamic stability [4]. Given the clinical course with IABP support in our case, it is reasonable that the present patient was diagnosed with fulminant myocarditis.

The LV function in our case was extensively reduced, but pulmonary congestion was not detected on chest radiography. Similarly, a national survey of fulminant myocarditis in Japan showed that 9 of 31 (29%) patients had no pulmonary congestion on chest radiographs[5]. The underlying mechanism remains uncertain, but we may safely consider that reduced RV function was the most likely cause, since RV plays a pivotal role in the development of pulmonary congestion [6]. RV dysfunction in the setting of impaired LV contractility is likely to lead to hemodynamic collapse, because it may be unable to maintain adequate LV preload [6]. This speculation can be applied to our case, given decreases in pulmonary artery pressure and cardiac output as assessed by right-heart catheterization.

Several mechanisms can be proposed as the cause of reduced RV function in our case. It is intuitive

to consider that lymphocyte myocarditis through the RV wall, which was confirmed on pathologic examination, was the primary etiology. Dehydration may be one of them, because of the small RV chamber on echocardiography performed in the emergency room. LV dysfunction should also be considered as the cause because experimental studies have shown that approximately 20 to 40% of RV function resulted from LV contraction [7]. Myocyte apoptosis or cytokine activation could also cause RV dysfunction [8,9], given that these factors have been thought to be associated with the development of acute myocarditis [10].

Reduced RV ejection fraction, as assessed by radionuclide angiography, rapid response thermodilution, or echocardiography, has been reported to be an independent prognostic factor in patients with heart failure [11-13]. Also, in patients with myocarditis, a previous study has provided evidence that RV dysfunction remains to be the most powerful predictor of adverse outcomes defined as death or need for cardiac transplantation [14]. These findings indicate that more attention should be paid to the detection of RV dysfunction, although the assessment of RV function is challenging because of its complex geometry. The diagnosis of fulminant myocarditis is not easy to make in emergency situations, but the current case highlights the importance of assessing pulmonary congestion on chest radiograph and RV wall motion abnormality on echocardiography for treatment decision and outcome prediction, when the possibility of acute myocarditis cannot be ruled out.

Figures

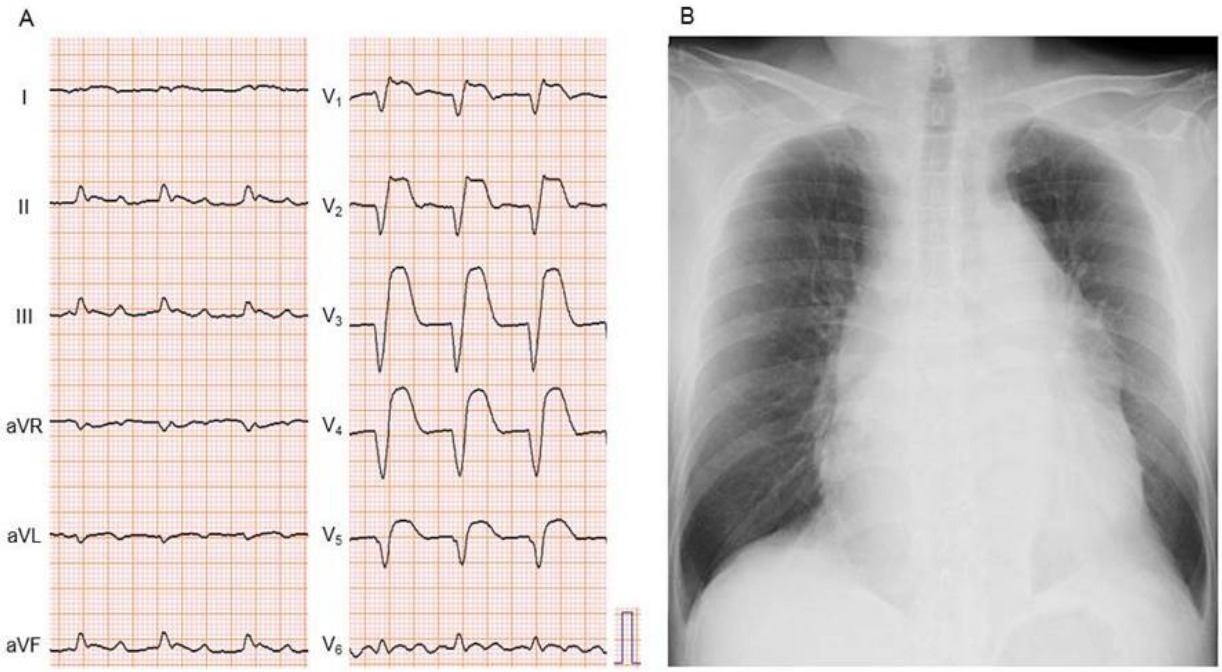

Figure 1: Electrocardiography and chest radiograph

ST-segment elevations with abnormal Q waves are shown in leads V_1 to V_5(A). Prolonged QRS durations, long QT intervals, and low voltage in limb leads are also present. Neither pulmonary congestion nor pleural effusion is obvious although cardiomegaly is noted (B).

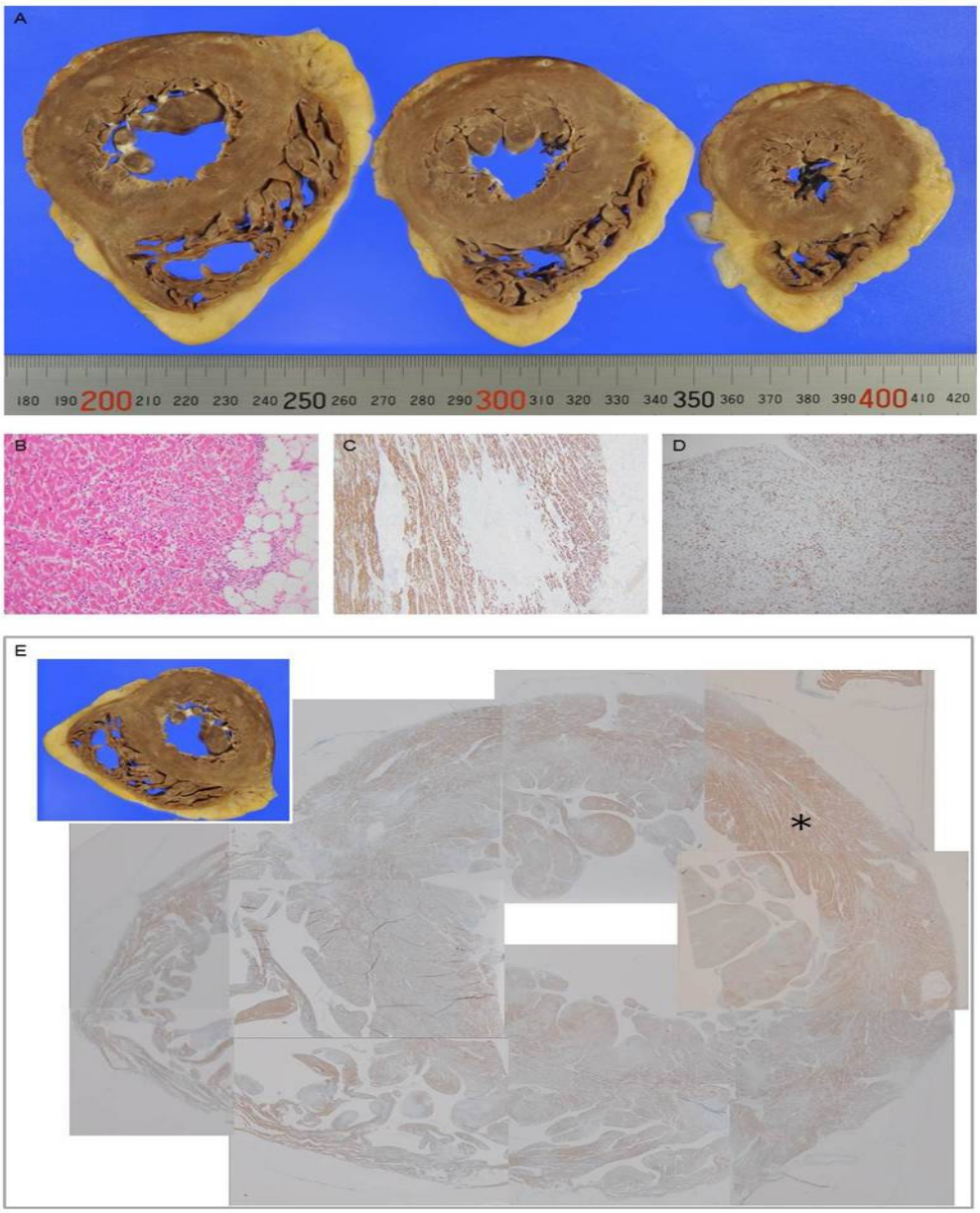

Figure 2: Pathological examination of the heart

Panel A shows patchy and yellowish degeneration of myocardium in dilated both ventricles as well as pericardial fat tissue degeneration around the heart (A). Panel B reveals widespread lymphocytic infiltration (hematoxylin and eosin). Panel C shows partially lost myocardium (desmin). Panel D demonstrates T cell lymphocytes infiltration into the myocardium (cluster of differentiation 3 or CD3). Desmin staining shows myocardial damage in both ventricles except for the LV lateral region (E, asterisk). The short axis image of the heart in Panel E was reconstructed by using 11 panels of desmin staining; the inset shows the same level as desmin staining.

References

1. Ginsberg F, Parrillo JE. Fulminant myocarditis. Crit Care Clin. 2013;29:465-483.

2. Maisch B, Ruppert V, Pankuweit S. Management of fulminant myocarditis: a diagnosis in search of its etiology but with therapeutic options. Curr Heart Fail Rep. 2014;11:166-177.

3. McCarthy RE 3rd, Boehmer JP, Hruban RH, Hutchins GM, Kasper EK, Hare JM, et al. Long-term outcome of fulminant myocarditis as compared with acute (nonfulminant) myocarditis. N Engl J Med. 2000;342:690-695.

4. JCS Joint Working Group. Guidelines for diagnosis and treatment of myocarditis (JCS 2009): digest version. Circ J. 2011;75:734-743.

5. Aoyama N, Izumi T, Hiramori K, Isobe M, Kawana M, Hiroe M, et al.; Japanese Investigators of Fulminant Myocarditis. National survey of fulminant myocarditis in Japan: therapeutic guidelines and long-term prognosis of using percutaneous cardiopulmonary support for fulminant myocarditis (special report from a scientific committee). Circ J. 2002;66:133-144.

6. Voelkel NF, Quaife RA, Leinwand LA, Barst RJ, McGoon MD, Meldrum DR, et al.; National Heart, Lung, and Blood Institute Working Group on Cellular and Molecular Mechanisms of Right Heart Failure. Right ventricular function and failure: report of a National Heart, Lung, and Blood Institute working group on cellular and molecular mechanisms of right heart failure. Circulation. 2006;114:1883-1891.

7. Santamore WP, Dell'Italia LJ. Ventricular interdependence: significant left ventricular contributions to right ventricular systolic function. ProgCardiovasc Dis. 1998;40:289-308.

8. Torre-Amione G, Kapadia S, Benedict C, Oral H, Young JB, Mann DL. Proinflammatory cytokine levels in patients with depressed left ventricular ejection fraction: a report from the Studies of Left Ventricular Dysfunction (SOLVD). J Am Coll Cardiol. 1996;27:1201-1206.

9. Schaper J, Elsässer A, Kostin S. The role of cell death in heart failure. Circ Res. 1999;85:867-869.

10. Liu PP, Mason JW. Advances in the understanding of myocarditis. Circulation. 2001;104:1076-1082.

11. Ghio S, Gavazzi A, Campana C, Inserra C, Klersy C, Sebastiani R, et al. Independent and additive prognostic value of right ventricular systolic function and pulmonary artery pressure in patients with chronic heart failure. J Am Coll Cardiol. 2001;37:183-188.

12. Karatasakis GT, Karagounis LA, Kalyvas PA, Manginas A, Athanassopoulos GD, Aggelakas SA, et al. Prognostic significance of echocardiographically estimated right ventricular shortening in advanced heart failure. Am J Cardiol.1998;82:329-334.

13. Gavazzi A, Berzuini C, Campana C, Inserra C, Ponzetta M, Sebastiani R, et al. Value of right ventricular ejection fraction in predicting short-term prognosis of patients with severe chronic heart failure. J Heart Lung Transplant. 1997;16:774-785.

14. Mendes LA, Dec GW, Picard MH, Palacios IF, Newell J, Davidoff R. Right ventricular dysfunction: an independent predictor of adverse outcome in patients with myocarditis. Am Heart J. 1994;128:301-307.

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