A Case of Fulminant Myocarditis With Three Recurrences and Recoveries

Yuya Matsue,1 MD, Leon Kumasaka,1 MD, Wataru Nagahori,1 MD, Masakazu Ohno,1 MD, Makoto Suzuki,1 MD, Akihiko Matsumura,1 MD, and Yuji Hashimoto,1 MD

Summary

Fulminant myocarditis is characterised by acute onset with severe haemodynamic deterioration. With intensive and appropriate bridging management, the prognosis is better than classic forms of myocarditis. Here, we report a patient who suffered from fulminant myocarditis 3 times over the last 8 years with recovery each time. (Int Heart J 2010; 51: 218-219)

Key words: Viral infection, Cardiogenic shock, Percutaneous cardiopulmonary support

Case Report

A 64-year-old man visited our clinic complaining of a fever and general fatigue on June 9, 2008. The symptoms persisted for 3 days. He had a past history of fulminant myocarditis with remission by use of mechanical circulatory assistance in 2000 and 2005. A cardioverter-defibrillator was implanted because of recurrent ventricular tachycardia in September 2005. Subsequently, amiodarone was added. He was also diagnosed with dermatomyositis in 1991 and was prescribed prednisolone at a dose of 2 mg per day. His global cardiac function had been well preserved and he had no restriction in daily activities. Echocardiography showed no left ventricle (LV) dilation and an LV ejection fraction of 66% in June 2007. The pathogen responsible for myocarditis in this case was not identified, and he had refused endomyocardial biopsy.

On admission, his body temperature was 38.6°C, pulse rate was 97 beats/min, and blood pressure was 145/97 mmHg. Laboratory examination revealed increased serum concentrations of creatine kinase (374 IU/L) and cardiac troponin I (9.06 ng/mL). Twelve-lead ECG showed convex ST segment elevations in the precordial lead. Chest radio-

Figure 1. Clinical course after admission. After admission day 1, PCPS and IABP were inserted for cardiogenic shock. On day 3 of admission, CK peaked out at 1,623 IU/mg. Solid line indicates EF; dotted line CK; broken line CK-MB; CK, creatine kinase; CK-MB, creatine kinase myocardial band; and EF, ejection fraction.

graphs showed no cardiomegaly and no pulmonary congestion. The first echocardiogram on admission showed no wall thickening or motion abnormalities of the LV and no pericardial effusion.

As he had suffered two previous episodes of fulminant myocarditis, we strongly suspected a third recurrence of fulminant myocarditis. His cardiac function became impaired rapidly after admission. LV ejection fraction decreased to 20% at 24 hours after admission. The systolic blood pressure dropped below 70 mmHg even with intraaortic balloon counterpulsation. Percutaneous cardiopulmonary support was also used as a bridge to recovery. The peak value of serum creatine kinase and its myocardial band were 1,623 IU/...
mg and 87 IU/mg, respectively, on the third hospital day. Cardiac function showed signs of recovery from the sixth hospital day, and mechanical support systems were stopped on the tenth day (Figure 1). His electrocardiogram returned to the prior findings (Figure 2). Examination of antibodies to viruses related to myocarditis showed no specific findings. Neither wall motion abnormalities nor chamber dilatation were detected on echocardiogram before discharge (Figure 3). He was discharged in an ambulatory state at the end of July 2008. We performed endomyocardial biopsies in March 2009. Histological examination showed a postinflammatory state, ie, myocardial atrophy and interstitial fibrosis. No active inflammation or cell infiltration was present. Simultaneous coronary arteriogram showed no coronary artery disease. The patient currently has no clinical symptoms.

**DISCUSSION**

Fulminant myocarditis has a better prognosis than acute myocarditis with appropriate management in the initial vulnerable state. Recently, we encountered a patient with fulminant myocarditis who had 3 recurrences over the previous 8 years, which is very rare. Although his cardiac function was severely deteriorated in the acute phases, he recovered each time and his ventricular contractility returned to normal with intensive bridging therapy for the term of cardiogenic shock. Although endomyocardial biopsy at the chronic phase did not show specific findings, such as active myocarditis, there was no doubt regarding the diagnosis of fulminant myocarditis. Endomyocardial biopsy is only considered as a class IIb recommendation. Chronic deterioration is not detected on serial echocardiograms.

There were no differences between the pathogenic mechanisms of fulminant and nonfulminant forms of myocarditis, and the host response to viral infection is also thought to be the same in acute lymphocytic myocarditis. Caforio and Iliceto reported a cardiac- or disease-specific antibody of the IgG class as a potential biomarker for identifying high-risk patients with myocarditis.

3. Dystrophin mutations in myocytes may facilitate myocarditis and cardiac failure during coxsackie B3 virus infection. Although such genetic factors have been suggested to be responsible for viral myocarditis, it is not clear why some viruses, which are common pathogens to which most people will be exposed, can lead to a fulminant form of myocarditis in some cases. An autoimmune mechanism of viral infection at the early stages of the disease should be considered as a more likely cause of fulminating morbidity. Unfortunately, it is difficult to clarify the pathophysiology underlying repeated myocarditis in our patient, and why the myocarditis was fulminant each time. A specific genetic background may play a role in the pathogenesis of the fulminant form of myocarditis. Our patient may have specific immunoreactivity for viral infection of his myocytes. Further basic and clinical examinations are necessary to elucidate the pathogenesis of this condition.

In summary, we have described a patient with fulminant myocarditis with a history of 3 recurrences over the previous 8 years. A hereditary factor for a specific pathogen is thought to be responsible for the repeated episodes in this patient.

**REFERENCES**