Clinical Case Report

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Myocardial calcification found in Epstein–Barr viral myocarditis and rhabdomyolysis

A case report

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Abstract

Rationale: The Epstein–Barr (EB) virus has rarely been reported as a cause of fulminant myocarditis. To our knowledge, the present case is the first report on myocardial calcification in EB viral myocarditis and rhabdomyolysis.

Patient concerns: A 17-year-old man was admitted to the department with fever, chest tightness, and tachypnea that had been present for 2 days.

Diagnoses: The initial investigation showed elevated liver enzyme levels, creatine kinase levels, creatine kinase isoenzyme levels, and elevated serum myoglobin. Echocardiography showed that left ventricular motion amplitude decreased. Test for immunoglobin M and immunoglobin G antibodies against Epstein–Barr virus were positive. These findings were consistent with fulminant myocarditis, cardiogenic shock, and rhabdomyolysis.

Interventions: The patient was intensively treated with venoarterial extracorporeal membrane oxygenation (VA-ECMO), continuous renal replacement therapy (CRRT).

Outcomes: Myocardial calcification was observed in the left ventricle walls on CT examination 10 days after the admission. Four months later, the patient is still alive and with adequate daily life.

Lessons: This case indicates that this rare form of myocardial calcification may be associated with EB viral infection and rhabdomyolysis.

Abbreviations: CRRT = continuous renal replacement therapy, CT = computed tomography, EB = Epstein–Barr, LVEF = left ventricular ejection fraction, MRI = magnetic resonance imaging, VA-ECMO = venoarterial extracorporeal membrane oxygenation.

Keywords: case report, Epstein–Barr virus, myocardial calcification, myocarditis, rhabdomyolysis

1. Introduction

Myocardial calcification has been observed in necrotic tissue following fulminant myocarditis in new-borns and infants.[1,2] It can be used as a marker of severe myocardial injury and has high mortality.[3] It is rare for marked acute dystrophic cardiac calcification to develop in a young adult patient. We report a 17-year-old man who survived Epstein–Barr viral myocarditis and rhabdomyolysis with accompanying extensive calcification of left ventricular walls, demonstrated by repeated CT.

2. Case presentation

A 17-year-old man was admitted to the department with fever, chest tightness, and tachypnea that had been present for 2 days. The patient had no history of cardiac, renal, or other disorders. In physical examination on admission, the following was observed: temperature was 38.5°C, pulse rate was 139/min, respiratory rate was 33/min, and blood pressure was 89/56 mm Hg. Heart sounds were scarcely audible. Both lower limbs were weakened. Echocardiography showed that left ventricular motion amplitude decreased, left ventricular systolic and diastolic function decreased, and left ventricular ejection fraction (LVEF) was 40%. Laboratory results were as follows: glutamic oxalacetic transaminase 1080 U/L, glutamic pyruvate transaminase 1450 U/L, creatine kinase 12000 U/L, creatine kinase isoenzyme 2880 U/L, lactic dehydrogenase 19300

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Availability of data and materials: All the data supporting our findings are contained within the manuscript.

Competing interests

Ethics approval and consent to participate

Ethics approval and consent for this case report were waived.

Consent for publication

Written informed consent was obtained from the patient for publication of this case report and accompanying images. A copy of the written consent is available for review by the Editor of this journal.

The authors have no conflicts of interest to disclose.

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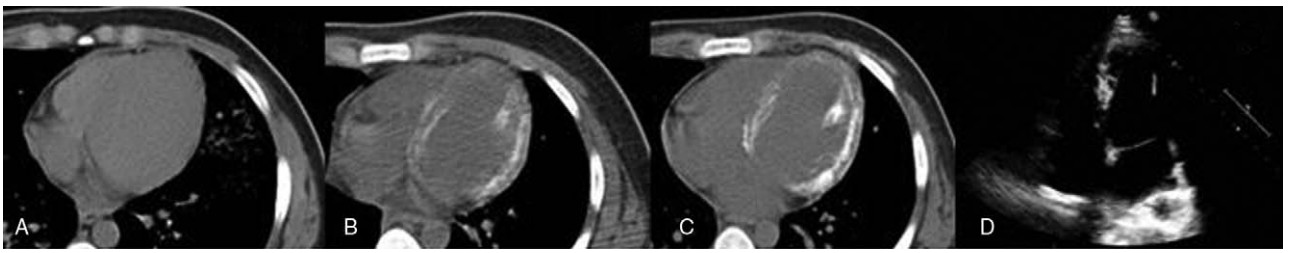

Figure 1. (A) Computed tomography (CT) scan showing no morphological abnormalities in the left ventricular wall on the day of admission. (B) Follow-up CT scan (10 days after admission) showing the left ventricular wall with increased density. (C) CT scan 30 days after admission showing extensive myocardial calcifications in the left ventricular wall with clearly increased density. (D) Echocardiography did not detect the myocardial calcifications. CT = computed tomography.

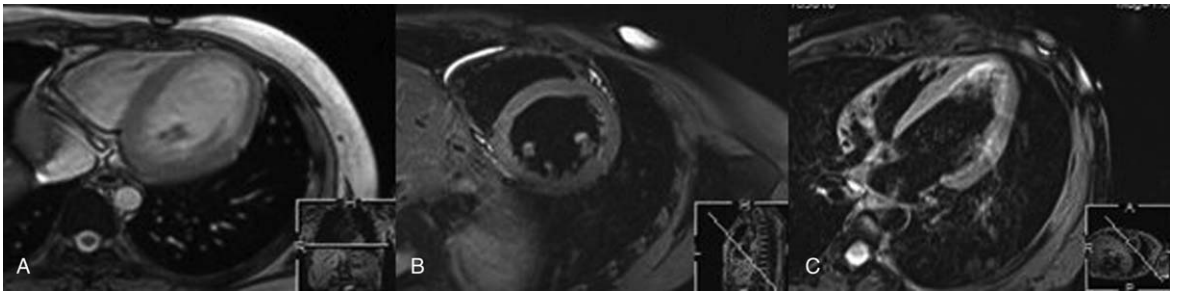

Figure 2. Magnetic resonance imaging scan 50 days after admission (4-chamber view [A] short-axis view [B] long-axis chamber view [C]) showing high-signal intensity in the left ventricular wall distribution.

U/L, serum myoglobin 1400 ng/mL, troponin I 4.01 μg/L, serum urea nitrogen 14.6 mmol/L, and serum creatinine 235 μmol/L. Immunoglobulin G (IgG) 8 g/L, antinuclear (ANA) and antismooth muscle antibodies (SMAs) were negative. After a few days, anti-Epstein–Barr virus antibodies IgG and IgM were positive. We made the diagnosis of fulminant myocarditis, cardiogenic shock, acute rhabdomyolysis and acute renal injury.

On the first day of admission, the patient suffered cardiac arrest resulting from acute left ventricular failure and malignant arrhythmias. Repeat echocardiography 1 day later demonstrated a LVEF of 35%. The patient was intensively treated with venoarterial extracorporeal membrane oxygenation (VA-ECMO) and continuous renal replacement therapy (CRRT) with unfractionated heparin as an anticoagulant without using citrates. A high dose of calcium gluconate was also intravenously infused for severe hypocalcaemia (1300 mg/day during the initial 5 days). The patient's clinical condition gradually improved, and VA-ECMO and the ventilator were withdrawn on days 3 and 5, respectively. CT examination showed no morphological abnormalities on the day of admission (Fig. 1A), but the left ventricular wall density increased 10 days after the examination (Fig. 1B), and more evident myocardial calcification was observed in the left ventricle, with a CT value of 180 HU (Fig. 1C) 30 days after the CT examination. Echocardiography did not detect the myocardial calcifications (Fig. 1D). There were no obvious abnormalities in blood phosphorus or parathyroid hormone levels. There was also no evidence of coronary artery abnormalities. Echocardiography (2 months after hospital admission) showed a LVEF of 56%. The patient was subsequently discharged with heart function class II on NYHA classification. Follow-up of a delayed gadolinium enhancement magnetic resonance imaging (MRI) examination performed 50 days after admission showed high signal intensity in the left ventricular wall without evidence of persisting inflammation, suggesting myocardial fibrosis/scarring resulting from myocardial injury/necrosis (Fig. 2A–C). Four months later, the patient is still alive and with adequate daily life.

3. Discussion

The Epstein–Barr virus has rarely been reported as a cause of acute myocarditis, accounting for approximately 1% of cases, but it has been associated with ventricular arrhythmias and sudden death.[4,5] Myocardial dystrophic calcification is a rare complication of myocarditis. We failed to find any other reports on myocardial calcification in EB viral myocarditis. CT, not MRI, is the gold standard for detecting myocardial calcification. [6] Echocardiography would likely be "blind" to this injury.

The primary cause of dystrophic calcification is calcium deposition in ischaemic and necrotic areas.[6] In this case, the patient's past clinical history and clinical examination did not indicate any muscle energy loss or ischaemic disease associated with muscle oxygen consumption (heat stroke, severe exercise, or seizures), except for some infection symptoms (high fever and chill), and EB viral antibodies were positive. This suggests that an EB viral infection may cause fulminant myocarditis and rhabdomyolysis, leading to myocardial injury and acute renal failure. Although the mechanism of myocardial calcification after acute viral myocarditis is not clear, it has been suggested that fulminant myocarditis resulting from viral infection can provoke more serious myocardial injury, resulting in a higher likelihood for calcium to deposit.

There are other possible mechanisms of cardiac calcification. First, abnormal calcium metabolism associated with rhabdomyolysis may result in hypocalcaemia and cardiac calcification.[7–9] It has been reported that hypocalcaemia is caused by 1,25-dihydroxy vitamin synthesis and bone muscles resistance to calcium action of the parathyroid hormone in the oliguric phase of acute renal failure. It has been reported that rhabdomyolysis itself can cause hypocalcaemia without acute renal injury or hyperphosphatemia. Hypocalcaemia is more apparent in rhabdomyolysis combined with acute renal injury. In this case, the myocardial calcification was found on a CT scan in the oliguria states; hyperphosphatemia did not occur. These results suggest that the abnormality in renal function and hormones may play a part in hypocalcaemia and myocardial calcification.

Second, extrinsic calcium resuscitation in rhabdomyolysis may be detrimental. In patients with rhabdomyolysis, the increase in intracellular calcium concentration activates intracellular neutral protease and leads to further cell destruction. Extrinsic calcium gluconate increases the intracellular calcium concentration even more, thus leading to greater protease activity.[10,11] This might intensify the calcium accumulation in myocardial or other soft tissues.

Author contributions

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