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A Case of Myocardial Biopsy in Fulminant Myocarditis

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Abstract: Clinical Data A 25-year-old female patient was admitted to our hospital on September 4, 2019, with "fever for 3 days and chest tightness and shortness of breath for 2 days". She had no previous history of coronary heart disease or hypertension. Three days before admission, she developed chills, low-grade fever, dry cough, and clear nasal discharge after catching a cold. Two days before admission, she experienced chest tightness and shortness of breath after mild physical activity, which relieved after rest. Admission physical examination: Temperature 37.3 ℃, blood pressure 85/55 mmHg, clear consciousness, poor mental state, pale complexion, respiratory rate 27 breaths/min, heart rate 108 beats/min, regular rhythm, no murmurs. Abdomen (-), no edema of lower extremities. Admission ECG: Sinus rhythm; ST segment elevation of 0.1–0.3 mv in leads II, III, aVF, V1-V6, and pathological Q waves in leads II, III, aVF. Initial diagnosis after admission: Fulminant myocarditis? Acute ST-segment elevation myocardial infarction (to be excluded). Laboratory tests on admission: D-dimer: 0.45 mg/L, B-type natriuretic peptide (BNP): 7401.8 pg/mL, high-sensitivity troponin I (cTnI): 11.41 ng/mL; creatine kinase isoenzyme (CK-MB): 59.2 ng/mL, myoglobin: 132.5 ng/mL; total white blood cell count: 3.74×10^9/L; hemoglobin: 125 g/L, C-reactive protein (CRP): 19 mg/L, erythrocyte sedimentation rate (ESR): 24 mm/1 h, procalcitonin: 0.07 ng/mL. Blood gas analysis (without oxygen inhalation): pH value: 7.403; partial pressure of carbon dioxide: 31.60 mmHg; partial pressure of oxygen: 45.50 mmHg; blood oxygen saturation: 79.00%; blood lactic acid (lac): 2.3 mmol/L. Transthoracic echocardiography showed: Left atrial diameter 31 mm, left ventricular diameter 35 mm, right atrial diameter 31 mm, right ventricular diameter 23 mm, interventricular septum 13 mm, left ventricular ejection fraction (LVEF) 60%. Chest CT plain scan: No exudative lesions in both lungs. Coronary CTA: No coronary artery stenosis. Cardiac magnetic resonance imaging (MRI): Normal diameters of each cardiac chamber, normal left ventricular function, no definite myocardial fibrosis or edema.

Keywords: Fulminant; Myocarditis; Myocardial biopsy

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1. Introduction

Fulminant myocarditis is myocardial injury caused by pathogen infection, immune factors, chemical drugs, etc.

It has an acute onset and severe condition, and can rapidly progress to heart failure or shock in a short period [1–4].

2. Case presentation

On September 5, 2019, right heart catheterization + myocardial biopsy was performed with intra-aortic balloon pump (IABP) support. After IABP insertion, blood pressure recovered from 85–90/50–60 mmHg to 100–105/60–70 mmHg. Results of right heart catheterization: Right atrial pressure 13/12/11 mmHg; right ventricular pressure 27/17/6 mmHg; mean pulmonary artery pressure 17 mmHg; pulmonary capillary wedge pressure 12 mmHg; cardiac output (CO) 2.4 L/min; cardiac index (CI) 1.8 L/min/m^2; pulmonary artery oxygen saturation 56.8%. Endomyocardial biopsy: Pathological specimens were taken from 3 different sites of the right ventricular septum. Postoperative immunohistochemical results showed: Inflammatory cells: CD3 (+), CD19 (-), CD20 (scattered few +), CD56 (-), CD68 (scattered +). Postoperative pathology: Myocardial cell degeneration, interstitial edema, mild proliferation of fibrous tissue, massive lymphocytic infiltration, occasional neutrophilic infiltration, consistent with myocardial interstitial inflammation.

The myocardial biopsy results supported the diagnosis of myocarditis. Treatment included methylprednisolone intravenous pulse therapy + intravenous immunoglobulin, diuretics, and symptomatic supportive care. Laboratory indicators such as BNP, cTnI, and lactic acid gradually decreased to normal, and hemodynamics was stable. Follow-up after 2 months showed no discomfort, normal exercise tolerance, normal myocardial enzymes, and normal echocardiography [5–9]. See **Figure 1** and **2**.

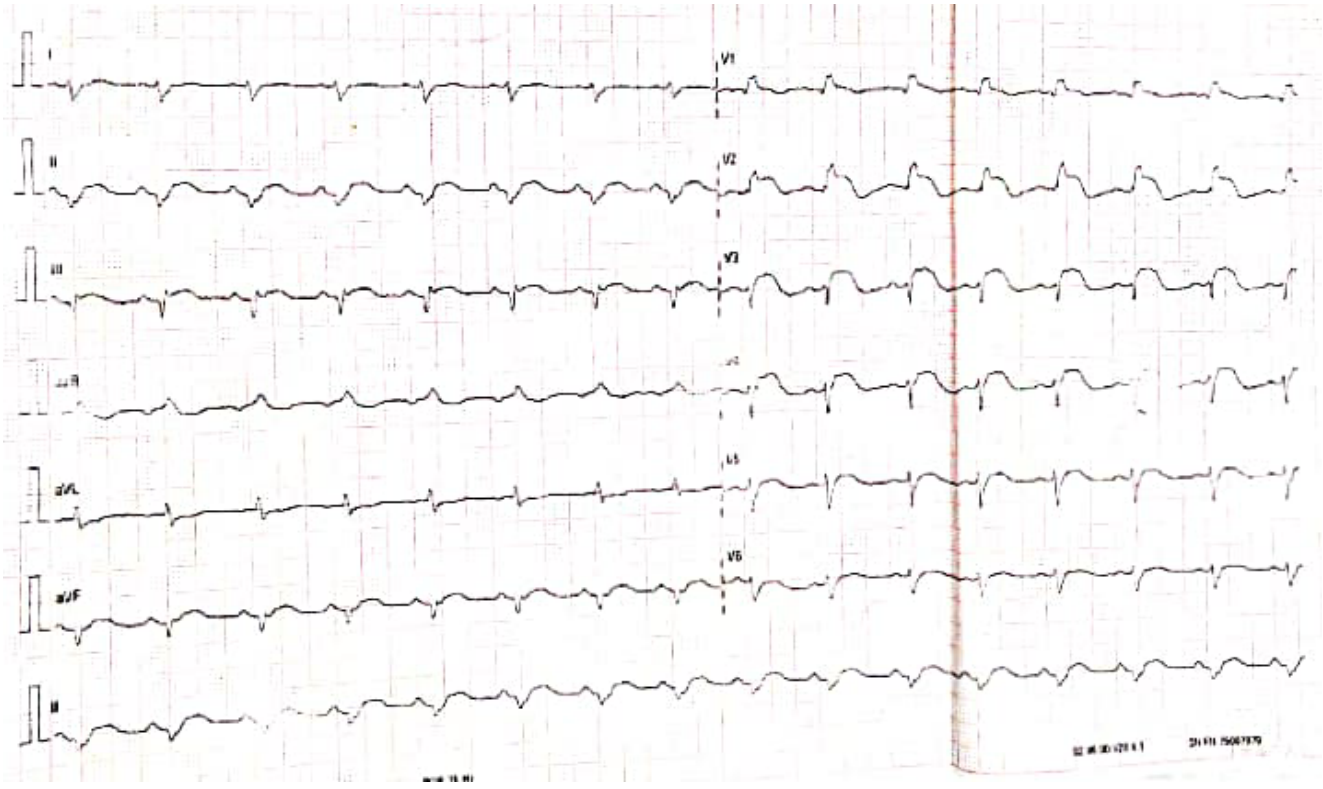

Figure 1. Admission ECG: ST segment elevation of 0.1–0.3 mv in leads II, III, aVF, V1-V6, and pathological Q waves in leads II, III, aVF.

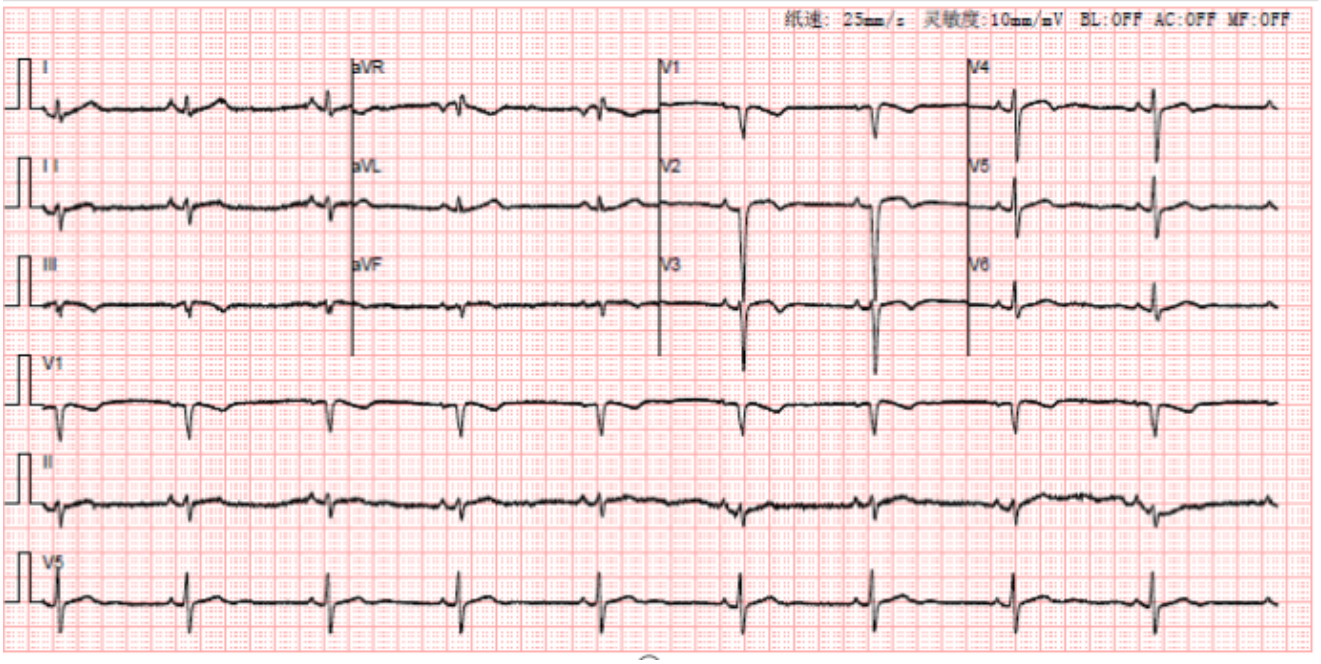

Figure 2. Pre-discharge ECG: ST segment elevation of 0.1–0.2 mv in leads V2, V4, and pathological Q waves in leads III, aVF

3. Discussion

Fulminant myocarditis is myocardial injury caused by pathogen infection, immune factors, chemical drugs, etc [9]. It has an acute onset and severe condition, and can rapidly progress to heart failure or shock in a short period. Myocardial injury indicators such as troponin and CK-MB are elevated. Acute phase is characterized by focal or diffuse myocardial tissue injury with massive inflammatory cell infiltration. Cardiac MRI may show characteristic myocardial changes such as edema, hyperemia, necrosis, and fibrosis. Endomyocardial biopsy provides an important basis for early diagnosis [10–12]. Differential diagnosis of myocarditis needs to exclude myocardial injury caused by acute coronary syndrome, sepsis, etc. After confirmation, treatment includes bed rest, nutritional support, anti-infection, glucocorticoids, intravenous immunoglobulin, and IABP or ECMO if necessary. IABP should be used as early as possible when hemodynamics is unstable. If cardiogenic shock persists despite IABP use, with cardiac index (CI) < 2.0 L/min/m^2 and blood lactic acid > 2 mmol/L, extracorporeal membrane oxygenation (ECMO) treatment can be used [13–15].

Characteristics of this case: The patient had an acute onset with prodromal respiratory tract infection symptoms, chest tightness, shortness of breath, hypotension, generalized ST segment elevation on ECG, elevated lactic acid, BNP and troponin. Coronary CTA showed no coronary stenosis, excluding acute myocardial infarction, and fulminant myocarditis was considered. However, echocardiography was normal, and cardiac MRI showed no typical manifestations of myocarditis such as myocardial edema, hyperemia, or fibrosis, so myocardial biopsy was necessary to confirm the diagnosis. The patient had hypotension, and right heart catheterization was performed under IABP support to maintain hemodynamic stability. Meanwhile, endomyocardial biopsy confirmed the diagnosis of myocarditis. Timely treatment with hormones and immunoglobulin resulted in a good prognosis. The patient's right heart catheterization showed CI: 1.80 L/min/

m^2, CO: 2.4 L/min, and blood lactic acid: 2.3 mmol/L. The patient had decreased cardiac output and elevated lactic acid, which indicated the possibility of ECMO placement. However, after IABP insertion, the patient's blood pressure recovered, circulation gradually improved, and blood lactic acid gradually decreased to 1.0 mmol/L, so ECMO was not ultimately used. After obtaining pathological specimens by myocardial biopsy, viral gene detection such as PCR, reverse transcription PCR, and nested PCR can be used to help identify pathogens. However, this examination has not been carried out in our hospital, which is a limitation of the diagnosis in this case.

4. Conclusion

This case demonstrates the critical role of timely endomyocardial biopsy in diagnosing fulminant myocarditis when imaging findings are inconclusive. Early mechanical circulatory support (IABP), combined with immunomodulatory therapy, effectively stabilized hemodynamics and led to a favorable outcome. Comprehensive evaluation including biomarkers, imaging, and histopathology is essential for accurate diagnosis and management of this potentially life-threatening condition.

Disclosure statement

The authors declare no conflict of interest.

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