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Two Cases of Acute Myocarditis with Suspected Acute Myocardial Infarction

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Abstract: Case 1: The patient is a 19-year-old male who was admitted to the hospital with a cough for three days and chest pain for a day, aggravated for two hours. Electrocardiogram showed 1. Sinus tachycardia 2. First-degree atrioventricular block 3. ST-T changes, not excluding acute extensive anterior wall myocardial infarction. Case 2: The patient was a 47-year-old woman admitted to the hospital with a fever for three days. Electrocardiogram showed: 1. Sinus tachycardia, 2. Complete block of the right bundle branch, 3. ST-T changes, not excluding acute extensive anterior myocardial infarction. The study of these two cases strengthened the departmental staff's knowledge of ECG in acute myocarditis and provided a rapid and accurate ECG diagnosis in the clinic.

Keywords: Acute myocardial infarction; Myocarditis; ECG diagnosis

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1. Case 1

The patient is a 19-year-old male, who was admitted to the hospital with cough for three days, chest pain for one day and aggravation for two hours. The patient experienced a cold three days ago and had a runny nose after catching a cold without a fever. He took medication orally on his own and felt that his cold symptoms had improved. The next day, when drinking alcohol after getting up in the morning, coughing symptoms develop and are accompanied by chest pain; chest pain is located in the sternum, accompanied by upper limbs losing strength. However, because the pain can be tolerated by the patient, so he did not pay attention. Yesterday afternoon at about 17:00, the patient felt that the pain worsened, during which there was nausea and vomiting once, so he came to the hospital. He was physically fit and denied any history of hypertension or diabetes mellitus. Examination: T 36.7 ℃, P 85 beats/min, R 20 beats/min, BP 100/60 mmHg, heart rate 85 beats/min, regular rhythm, no pathologic murmur, the abdomen is flat and soft, no pressure and rebound pain, liver and spleen were not touched under the ribs, and there was no swelling of both lower limbs. Auxiliary examination: electrocardiogram (**Figure 1**): (1) Sinus tachycardia, heart rate 101 beats/min; (2) First-degree atrioventricular block, PR interval 0.20 seconds; (3) ST-T changes, not excluding acute extensive anterior wall myocardial infarction, with obvious bow-back elevation of the ST segments of the I, avL and V2–V6 leads, the

amplitude of which was around 0.2–0.5 mv, and fusion of the T wave and the ST segment in a T-wave and ST-segment fusion is a single curve. Chest X-ray: Enlarged heart shadow; Cardiac ultrasound: Small mitral valve regurgitation, small tricuspid valve regurgitation, left ventricular systolic function measurement is normal; Emergency series: K 3.52 mmol/L, blood glucose 7.28 mmol/L, AST 199 IU/L, ALT 69 IU/L, creatine kinase 1641 IU/L, creatine kinase isoforms 116.65 U/L; Coagulation function: Normal; Blood routine: WBC 9.36×10^{9}/L, basophil percentage 0.2%; Blood gas analysis was normal; Infarction three: CK-MB 62.85 ng/ml, cTnl 29.14 ng/ml, MYo 324.8 ng/ml; Preliminary clinical diagnosis: Acute extensive anterior wall myocardial infarction, cardiac function grade I. After admission, emergency surgery was performed: Percutaneous selective arterial cannulation, intracoronary local drug release therapy and percutaneous super-selective arteriography. Coronary angiography confirmed: The coronary artery was right dominant, the left main trunk did not show significant stenosis, the anterior descending branch did not show significant stenosis, distal flow TIMI (Grade 3); The diagonal branch did not show significant stenosis, distal flow TIMI (Grade 3); The gyratory branch did not show significant stenosis, distal flow TIMI (Grade 3); The obtuse marginal branch did not show significant stenosis, distal flow TIMI (Grade 3). Angiographic diagnosis: Normal coronary artery. Postoperative review of electrocardiogram compared with the previous electrocardiogram showed no significant changes. The clinical diagnosis was clarified: Acute myocarditis and pulmonary infection. The ECG was reviewed 1 day after admission (**Figure 2**): (1) Sinus rhythm, heart rate 76 beats/min; (2) First-degree atrioventricular block, PR interval 0.23 seconds; (3) Premature ventricular contractions, seen early appearance of wide and aberrant QRS-T wave clusters; (4) ST-T changes, I, avL, V2–V6 ST segments upward elevation has been gradually retracted, I, avL inverted. After anti-inflammatory and myocardial nutritional treatments, the patient was discharged from the hospital with improved condition.

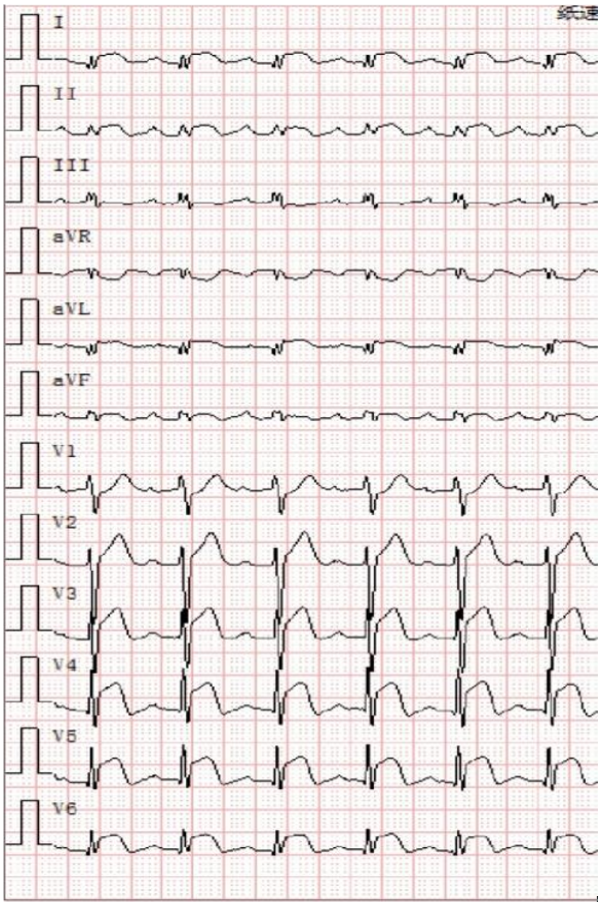

Figure 1. Admission emergency electrocardiogram of case 1.

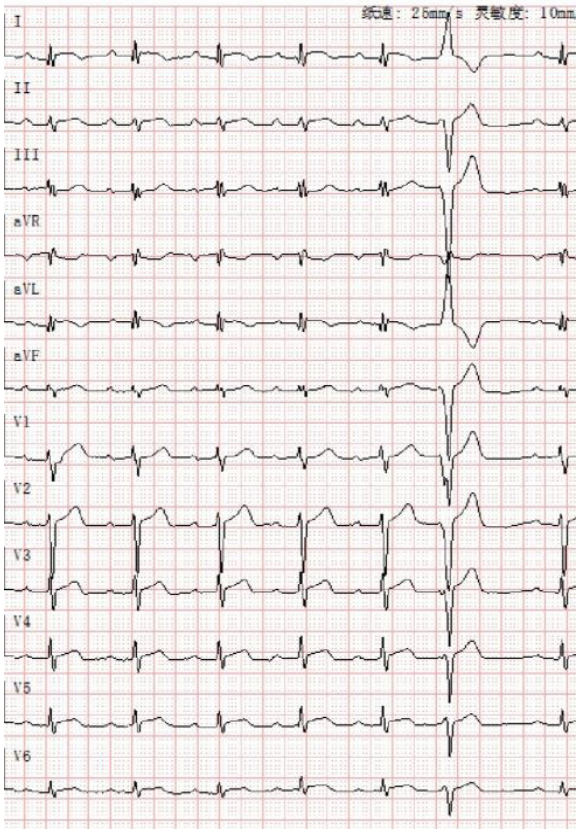

Figure 2. Electrocardiogram of case 1 one day after admission.

2. Case 2

The patient is a 47-year-old female admitted to the hospital with a fever for three days. The patient started to have a fever three days ago without obvious triggers, with a maximum temperature of 39.5 ℃, accompanied by a sore throat and chest tightness. Examination: T 37 ℃, P104 beats/min, R24 beats/min, BP 76/59 mmHg. heart rate 104 beats/min, rhythmic. Auxiliary examination: Electrocardiogram showed (**Figure 3**): (1) Sinus tachycardia, heart rate 114 beats/min; (2) Complete right bundle branch block with qR pattern in lead V1, widening of S-wave in leads I, avL, V5, V6, S-wave > 0.04 s, QRS wave > 0.12 s; (3) ST-T changes, not excepting acute extensive anterior wall myocardial infarction, with obvious bow-back elevation of ST-segment in leads V1–V5, amplitude of elevation at around 0.10–0.70 mv, and fusion of T-wave and ST-segment in a single T-wave and ST-segment fusion in a single curve. Emergency series: K 3.47 mmol/L, Na 129 mmol/L, Ca 2.08 mmol/L, creatine kinase 1061 IU/L, creatine kinase isoenzyme 69.08 U/L; coagulation function: prothrombin time 14.20 s; Blood routine: WBC 10.47×10^9/L, neutrophil 76.40%; lymphocyte 13.40%; neutrophil 8.40%. Neutrophil count 8.00×10^9/L; monocyte count 0.79×10^9/L. Cardiac ultrasound showed: Atrial orthotropy, normal size of each atrium. The left ventricular myocardium was thickened, the left ventricular myocardial motion was diffusely weakened at rest, the left ventricular ejection fraction and short-axis shortening rate were reduced, and the EF was 48%. The morphology of each valve was still active, and a small amount of regurgitant signals could be detected in the mitral valve and the tricuspid valve. The internal diameters of the main artery and the pulmonary artery were normal. The electrocardiogram was reviewed four hours after admission (**Figure 4**): (1) Ventricular tachycardia, P wave disappeared, QRS wave was wide and distorted; (2) ST-T changes, ST segment bow-back elevation in leads V1–V6 was greater than 0.1 mv. The

preliminary clinical diagnosis was fulminant cardiomyopathies (the diagnosis was confirmed after emergency PCI), acute decompensated heart failure, cardiogenic shock and cardiac function class IV. After admission, emergency surgery was performed: Percutaneous selective arterial cannulation, intracoronary local drug release therapy and percutaneous super-selective arteriography. Coronary angiography confirmed: The coronary artery was right dominant, the left main trunk did not show significant stenosis, the anterior descending branch did not show significant stenosis, the diagonal branch did not show significant stenosis, and the distal flow TIMI (Grade 3); The echogenic branch did not show significant stenosis, and the distal flow TIMI (Grade 3); The obtuse marginal branch did not show significant stenosis, and the distal flow TIMI (Grade 3). Diagnostic imaging: Normal coronary artery. Three days after admission, the ECG was reviewed (**Figure 5**): (1) Sinus rhythm, heart rate 65 beats/min; (2) Complete right bundle branch block, with a qR pattern in the V1 lead, widening of the S wave in the I, avL, V5 and V6 leads, with an S wave of > 0.04 seconds and a QRS wave of > 0.12 seconds; (3) ST-T changes, with significant ST-segment regression in the II, III, avF, and V1–V5 leads, only the V1–V4 leads showing a ST-segment regression, and the V1–V5 leads showing a ST segment regression. V1–V4 leads showed horizontal elevation of 0.05–0.1 mv, and T-wave inversion in I, avL and V1-V6 leads. The patient was discharged with stable condition 12 days after PCI.

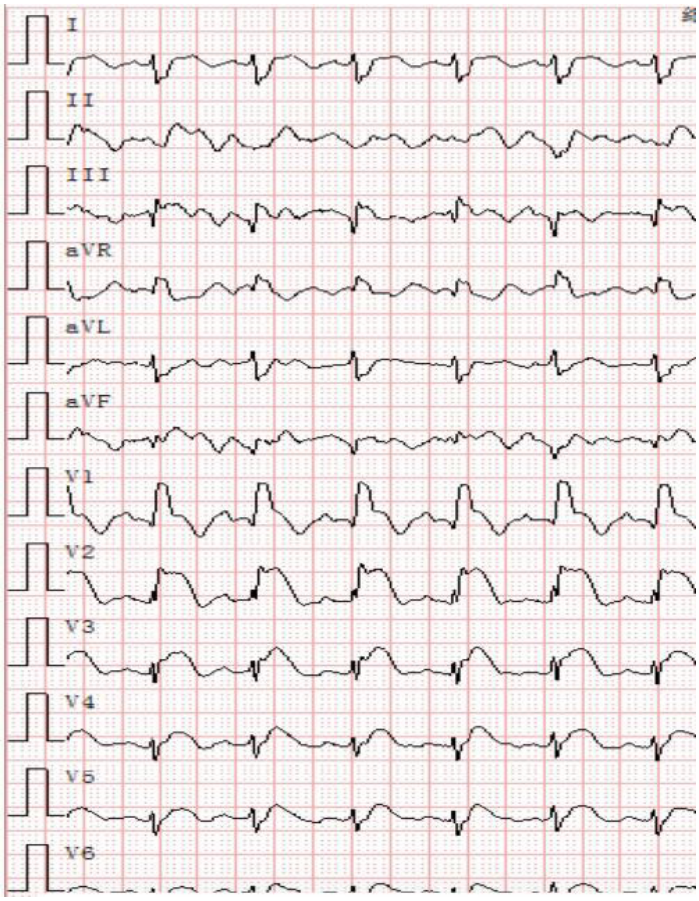

Figure 3. Admission electrocardiogram of case 2.

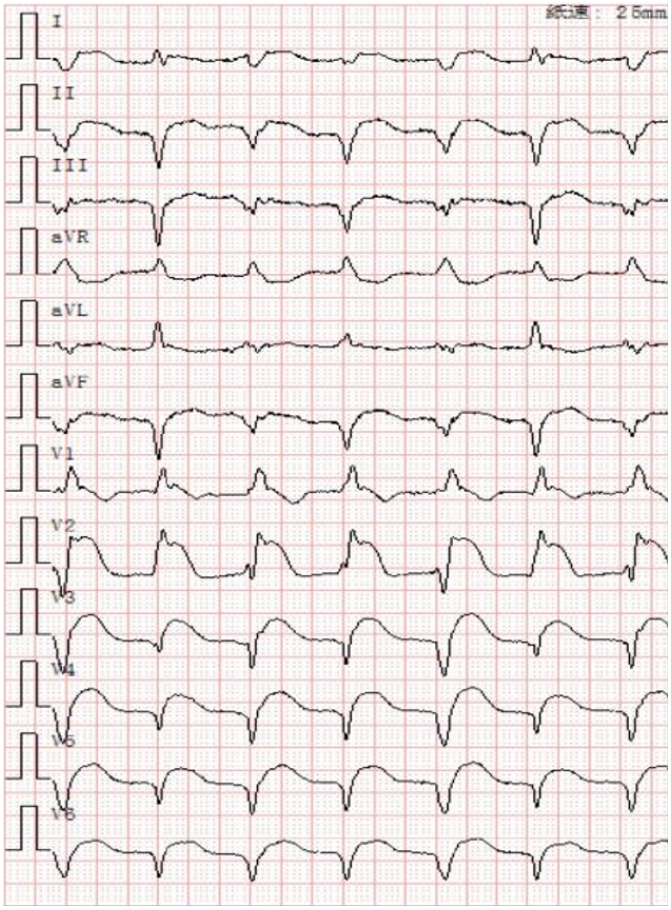

Figure 4. Review of electrocardiogram 4 hours after admission of case 2.

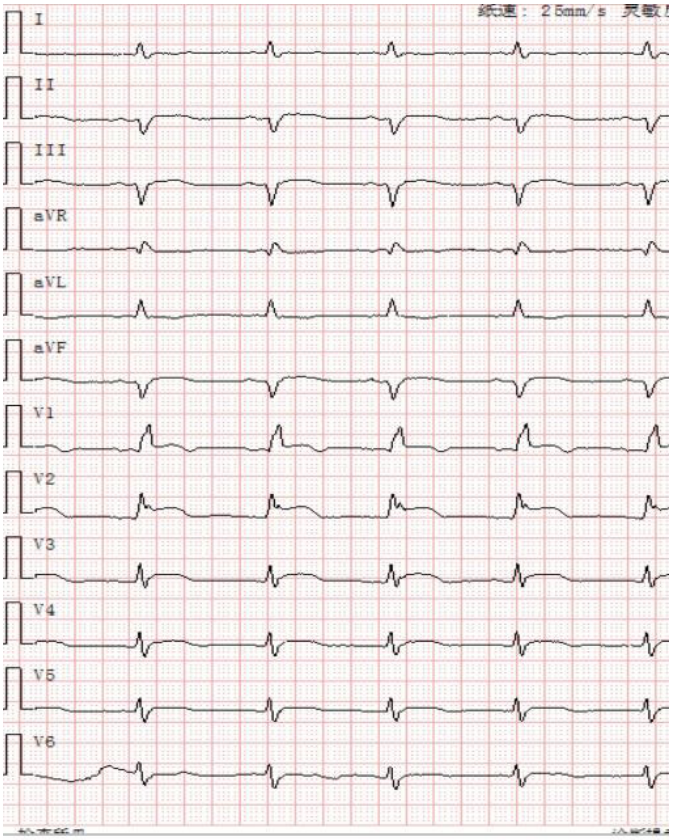

Figure 5. Repeat ECG of case 2 after being admitted to hospital 3 days later.

3. Discussion

Myocarditis refers to limited and diffuse inflammation of the myocardium and its interstitium caused by various reasons, including viral myocarditis, toxic myocarditis (bacterial infection or drug poisoning) and rheumatic myocarditis. Electrocardiographic changes vary due to the nature of the lesion and the type and degree of pathological changes [1]. An electrocardiogram (ECG) is a commonly used clinical examination in patients with acute myocarditis, and about 85% of patients may present with different ECG manifestations [2]. The myocardium is metabolically active and sensitive to hypoxia and toxins [3], both of which can cause limited and/or diffuse myocardial lesions that replicate within myocardial cells, leading to myocardial lysis and a series of myocardial autoimmune responses [4], such as myocardial parenchymal and interstitial inflammation, resulting in myocardial cell lysis, necrosis, denaturation, swelling and infiltration of inflammatory cells into the perivascular connective tissue between the myofibers, followed by a series of myocardial autoimmune reactions [5]. Inflammatory cell infiltration is followed by the formation of scar tissue, myocardial injury, myocardial ischemia, etc., which are the basis for the formation of abnormal myocardial electrophysiological activity.

The diagnosis of myocarditis is usually exclusive and the clinical symptoms of myocarditis are almost indistinguishable from those of acute coronary syndromes, cardiomyopathy, valvular disease, and pericarditis, which may be accompanied by chest pain, elevated levels of markers of myocardial injury and abnormalities on the electrocardiogram (including depression or elevation of the ST-segment, inversion of the T-wave and disease-induced Q-wave formation). In a recent observational study of myocarditis confirmed by endomyocardial biopsy, approximately 92.6% of patients had ECG abnormalities, the most common being ST-T changes in 59.0%. This was followed by bundle branch conduction block in 10.6% [5]. Emergency coronary angiography is feasible to clarify the diagnosis at an early stage and develop a rescue treatment plan early to buy time and emergency angiography does not increase the mortality rate of patients [6], but attention should be paid to reducing the amount of contrast agent to reduce its negative inotropic effect. Acute myocarditis is similar to myocardial infarction, where clinical manifestations of severe myocarditis with ST-segment elevation and elevated troponin levels are mainly seen in young patients with acute viral myocarditis, especially in men [7]. Case 1 was a young man, which may be related to the fact that the onset of myocarditis is predominantly in young adults and that the autoimmune response in men is stronger. Case 2 is a middle-aged woman and the patient has symptoms such as chest tightness, fatigue, poor appetite, significantly elevated myocardial enzyme profile, electrocardiogram shows myocardial ischemic changes as well as new-onset right bundle branch block, recurrent ventricular arrhythmia, which is similar to the sympathetic electrical storm after myocardial infarction. Therefore, it is easy to be misdiagnosed as acute myocardial infarction, which leads to emergency PCI.

Myocarditis has an acute onset and rapid progression and is extremely dangerous, i.e., heart failure, cardiogenic shock, sudden death and other adverse events can occur within a short period of time after the onset of the disease and even combined with malignant arrhythmia, which is hazardous to the patient's life. Therefore, treating myocarditis as early as possible is necessary to prolong the survival period. However, after the occurrence of myocarditis, there are often precursor symptoms of viral infection, such as fever, myalgia, respiratory symptoms or gastrointestinal symptoms [8], and clinically, it is mostly seen in young adults. Therefore, clinical attention should be paid to the early diagnosis and treatment of myocarditis and the pathological features of myocarditis should be analyzed in depth. In addition, myocarditis can also cause an increase in cardiac enzyme profiles and ST-segment damage type elevation in the electrocardiogram, which needs to be differentiated from acute myocardial infarction. On cardiac ultrasound, patients with myocardial infarction show abnormalities of ventricular myocardial wall motion and hypodiastolic and systolic hypoplasia, all of which are higher than in the myocarditis group. Previous studies have found that

ventricular wall motion abnormalities in patients with viral myocarditis are mainly due to a decrease in the amplitude of myocardial contractile motion, which results from myocardial fibrosis caused by inflammatory factors invading the heart [9]. In patients with acute myocardial infarction, segmental ventricular wall motion abnormalities, abnormal ventricular wall thickness, ventricular wall tumors, septal perforation, papillary muscle insufficiency, intraventricular appendage thrombus and decreased cardiac systolic and diastolic function were the main findings [10]. Although both groups of patients showed different ventricular wall motion abnormalities, hypodiastolic and systolic dysfunction, myocardial injury and necrosis in acute myocardial infarction are related to the region of myocardial blood supply innervated by occluded coronary arteries, which is distinctly regional in nature. As a result, acute myocardial infarction is more prone to ventricular wall motion abnormalities, diastolic hypokinesis, and systolic hypokinesis. Other studies have shown that myocarditis patients with clinical manifestations similar to those of acute myocardial infarction are best diagnosed by cardiac magnetic resonance [11]. Cardiac magnetic resonance can not only correctly diagnose both diseases but can also assess the prognosis of both diseases. Cardiac magnetic resonance can detect the surviving myocardium, evaluate myocardial reperfusion and myocardial fibrosis, etc. It can be used to risk stratify patients with myocarditis and acute myocardial infarction, predict adverse cardiovascular events, and thus guide the improvement of clinical treatment programs.

Both patients in Case 1 and Case 2 had a history of upper respiratory tract infection, elevated body temperature and ST-segment elevation on the electrocardiogram, which was similar to that of acute myocardial infarction without the presence of necrotic Q waves. After symptomatic treatment, the ST segment in the electrocardiogram quickly fell back to the baseline level without any obvious electrocardiographic evolution. The myocardial enzyme profile fell back to normal after hospitalization without the enzyme peak evolution pattern of myocardial infarction. All of the above manifestations were helpful in differentiating from acute myocardial infarction. By studying these cases, the staff of the department will have a better understanding of the electrocardiogram in acute myocarditis and will be able to provide rapid and accurate electrocardiogram diagnosis for the clinic.

Disclosure statement

The authors declares no conflict of interest.

References

[1] Chen Q, 2012, Electrocardiography. Shandong Science and Technology Press, Jinan, 1140–1145.

[2] Ammirati E, Frigerio M, Adler ED, et al., 2020, Management of Acute Myocardial and Chronic Inflammatory Cardiomyopathy: An Expert Consensus Document. Circulation: Heart Failure, 13(11): e007405.

[3] Wang S, 2012, Clinical Analysis of 93 Cases of Arrhythmia in Pediatric Viral Myocarditis. Clinical Medicine, 32(9): 92–93.

[4] Xing S, Dong P, Wang H, et al., 2011, Analysis of the Relationship Between Electrocardiogram and Clinical Manifestations in Viral Myocarditis. Journal of Practical Cardiovascular and Pulmonary Vascular Diseases, 19(11): 1881–1882.

[5] Grün S, Schumm J, Greulich S, et al., 2012, Long-term Follow-up of Biopsy-Proven Viral Myocarditis: Predictors of Mortality and Incomplete Recovery. Journal of the American College of Cardiology, 59(18): 1604–1615.

[6] Precision Medicine Group of the Cardiovascular Disease Branch of the Chinese Medical Association, Editorial Board of the Chinese Journal of Cardiovascular Disease, Working Group on Adult Violent Myocarditis, 2017,

Chinese Expert Consensus on the Diagnosis and Treatment of Adult Fulminant Myocarditis. Chinese Journal of Cardiovascular Disease, 45(9): 742–752.

[7] Li Z, 2020, Clinical Application Value of Echocardiography in the Diagnosis of Viral Myocarditis. Modern Medical Imaging, 29(11): 2142–2144.

[8] Liguori C, Farina D, Vaccher F, et al., 2020, Myocarditis: Imaging Up to Date. Official Journal of the Italian Society of Medical and Interventional Radiology, 125(11): 1124–1134.

[9] Geng A, 2020, The Value of Echocardiography in the Diagnosis of Myocardial Infarction. Clinical Medicine, 40(6): 71–73.

[10] Zhou D, Zhuang B, Zhao S, et al., 2019, Progress in Cardiovascular MR Diagnosis of Myocarditis Research: Based on 2018 Revision of CMR Criteria for Diagnosis of Non-Ischemic Myocardial Inflammation Expert Recommendations. China Medical Imaging Technology, 35(10): 1574–1577.

[11] Lurz P, Eitel I, Adam J, et al., 2012, Diagnostic Performance of CMR Imaging Compared with EMB in Patients with Suspected Myocarditis. JACC: Cardiovascular Imaging, 5(5): 513–524.