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Case Report

Acute myopericarditis masquerading as acute myocardial infarction

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Abstract

Patients with abrupt onset of chest pain, ischemic ECG abnormalities and elevated levels of cardiac markers could be given a diagnosis of acute myocardial infarction. However, some other diseases should be taken into consideration in this clinical setting when coronary arteries are proven to be normal. Here we report a case of acute myopericarditis with clinical presentation of myocardial infarction and normal coronary anatomy. The Herpes Simplex Virus II was considered as the organism causing myopericarditis and the patient was recovered by the treatment with valacicloavir. A precise diagnosis is a prerequisite of successful treatment and favorable prognosis.

Key words: myopericarditis, acute myocardial infarction, virus

INTRODUCTION

Patients with abrupt onset of chest pain, ischemic ECG abnormalities and creatine kinase(CK) or troponin I elevations could be given a diagnosis of acute myocardial infarction(AMI). However, some other diseases should be taken into consideration in this clinical setting when coronary arteries are proven to be normal. Here we report a Caucasian case of acute myopericarditis with clinical presentation of myocardial infarction and normal coronary anatomy.

CASE REPORT

General materials

A 26-year-old Caucasian professional athlete suffered from high fever(39.2℃) and thereafter felt severe acute chest pain after he came to China in summer from a South-hemisphere country in winter(big difference of climate) and had routine football training. The body temperature went down temporarily on the next day after he took amoxycillin and paracetamol, but the chest pain lasted for 3 days and relieved gradually. He never felt shortness of breath on exertion or dizziness. He felt only tiny chest pain and malaise when he was admitted to hospital on July 20,2007. He denied history of hypertension, diabetes and family history of heart disease. He is a non-smoker.

On physical examination, his temperature was 37.8℃, the pulse 72 beats per minute(bpm), the blood pressure 120/80 mmHg and the oxygen saturation was 99% while the patient was breathing ambient air. No rash was seen and the remainder of the examination was normal.

The experimental test and special examination

The results of laboratory tests are shown in ***Tab 1***. The electrocardiograph(ECG) obtained after admission presented normal sinus rhythm, ST segment elevation in widespread leads except for lead aVR, especially in the leads of anterior wall(0.7mV on V4)(***Fig 1***). The transthoracic echocardiogram showed mild pericardial effusion(3-5 mm in diastolic period) without significant cardiac structural or left ventricle segmental wall-motion abnormalities, the left ventricular ejection fraction(LVEF) was 61%.

To exclude acute myocardial infarction, a multidetector computer tomography(MDCT) coronary angiography was performed and no significant lesion was found in coronary arteries(***Fig 2***). The SPECT $^{99}Tc^{m}$-MIBI myocardial perfusion scan(***Fig 3***) detected low

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perfusion in basal anterior wall, anterior wall and posterior wall of left ventricle and mild compromised LV systolic function with LVEF 48%.

Diagnosis, treatment and prognosis

Considering that the patient had fever, chest pain, typical ECG pattern of epicardial injury and moderate elevated level of blood cardiac marker, with exclusion of coronary artery disease, a diagnosis of myopericarditis was considered. Absolute rest was recommended. Intravenous ganciclovir(anti-virus drug),creatin phosphate, and oral azithromysin, monopril, bisoprolol, trimetazidine were administered.

Initially, he recovered really well with diminishing serum level of troponin I and cardiac enzymes(***Fig 4***) and ST segment elevation resolution, however, his temperature kept fluctuated from 37℃ to 37.5℃. Ganciclovir and azithromysin were discontinued on day 7 after admission. The level of cardiac markers went up again on day 6 and fluctuated thereafter, accompanied with low fever (up to 38.5℃ on day 9, ***Fig 4***). The serial ECGs showed regular AMI-like evolvement, resolved ST elevation to baseline and inverted T wave(***Fig 1***). Follow-up of Ultrasound found less pericardial effusion and did not find any cardiac structural change or abnormal movement of LV wall.

Tab 1 **Results of laboratory test**

Variable	Value	Reference
WBCs(10^3per mm^3)	10.70	4-10
Neutrophils(%)	73.40	50-70
Hemoglobin(g/dl)	14.30	11-16
Cholesterol(mmol/l)	3.10	2.8-6.5
CK(u/l)	481	30-170
CK-MB(u/l)	27.00	0-25
Troponin I(ng/ml)	11.65	0-0.4
ESR(cmH_2O/hour)	65.00	0-15
CRP(mg/dl)	16.10	0-0.8
U&E, LFT	Normal	
ASO & RF	Negative	
Virus antibodies△	Negative	
CTD-related antibodies	Negative	

*WBCs, white blood cells; CK, creatin kinase; ESR, erythrocyte sedimentation rate; CRP, C-reactive protein; U&E, urea, creatinine and electrolytes; LFT, liver function test; ASO, anti-streptolysin O; RF, rheumatoid factor; CTD, connective tissue disease.

△ The antibodies(Ig M) to followed viruses were tested: Herpes Simplex Virus-I&II, Respiratory Synctial Virus, Coxsackie Virus, Rubella Virus, Cytomegalovirus, Epstein-Barr Virus, Adenovirus, Human Immunodeficiency Virus, Hepatitis A, B&C virus.

After repeated inquiry, the patient reported a 4 years history of genital herpes, i.e., infection of Herpes Simplex Virus(HSV-II), and he was prescribed valacicloavir 50 mg twice daily when necessary. The genital herpes reoccurred several times in the first 3 years, but not in the last year. Valacicloavir 50mg, twice daily, was prescribed again since HSV-II was suspected to be the virus causing myocarditis. Thereafter, the patient's temperature began to decrease and the blood cardiac markers ran down into normal range gradually(***Fig 4***). His heart rate reduced slowly from 70 bpm down to 50 bpm(that being his usual heart rate previously). Thereafter, the beta-blocker was discontinued. However, the ESR was still rapid(60 mmH_2O/hour), and the CRP is a bit higher than normal(1.11 mg/dl). No changes were found by cardiac ultrasound re-evaluation compared with the result of last time. The patient was allowed to go back to his homeland to continue the treatment, considering that the normal level of cardiac marker and he felt completely normal. The patient was followed up by private correspondence for 6 months; he avoids exercising intensely and feels well and healthy.

DISCUSSION

Acute myocardial infarction is a common heart disease with high mortality and morbidity. In the clinical setting of abrupt onset of chest pain, ischemic ECG abnormalities, and elevated level of creatine kinase or troponin I/T, suggestive of myocardial injury, AMI

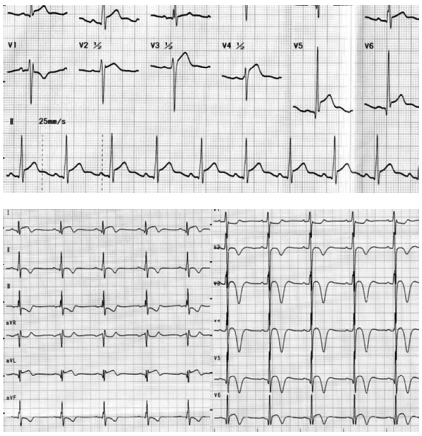

Fig 1 The ECG on admission(upper panel) and day 7(lower panel). The upper ECG showed widespread ST segment elevation with PR segment depression and the ratio of the ST-segment elevation to T-wave amplitude in excess of 0.24 in lead V_6. The lower presented the ECG evolvement, resolution of ST segment elevation with T wave inversion, mimicking the ECG changes in AMI patients

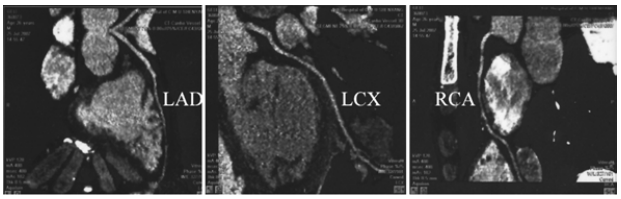

Fig 2 MDCT coronary angiography showed normal coronary arteries, left anterior descending branch(left), left circumflex branch(middle) and right coronary artery(right)

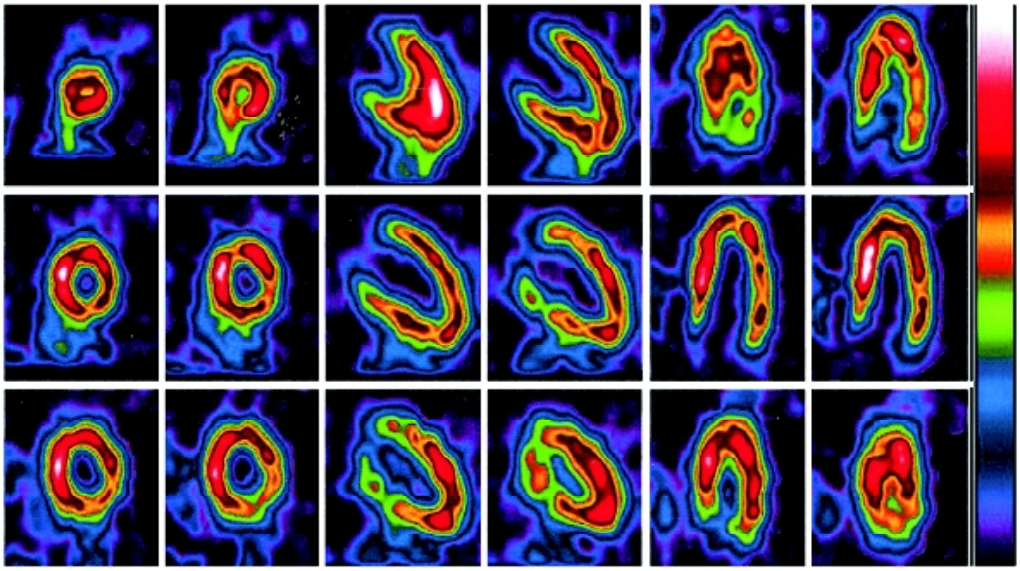

Fig 3 The SPECT $^{99}Tc^{m}$-MIBI myocardial perfusion scan detected low perfusion(yellow color) in basal anterior wall, anterior wall and posterior wall of left ventricle. The brightness of color reflected the extent of myocardial perfusion

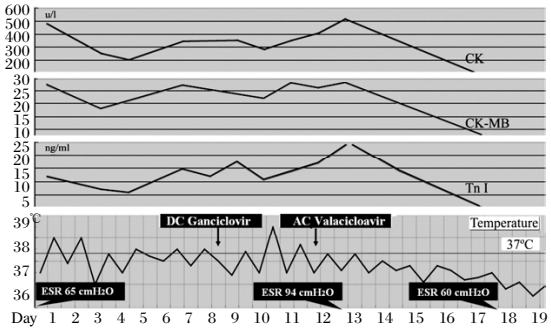

Fig 4 The summarizing graphics presented the changes of cardiac markers, temperature and other condition in hospital according to the chronological order

should be considered as a probable diagnosis regardless of the age and gender. In this case, the young football player does not have any risk factors of atherosclerosis. The ECG change was characterized of widespread ST elevation in precordial and limb leads except in aVR, PR-segment depression and the ratio of the ST-segment elevation to T-wave amplitude in excess of 0.24 in lead V_6, indicating typical ECG features of pericarditis[1], but not AMI; in which the ST eleva-tion is typically convex in shape and usually confined to a single coronary vascular territory[2]. The extent of cardiac markers' elevation is mild to moderate, not consistent with the involved myocardium area reflected by ST elevation in ECG. Negative findings in MDCT angiography helped us definitely exclude the diagnosis of AMI. Thus, after exclusion of coronary artery disease, clinical presentation of the patient led to the consideration of an important differential diagnosis-acute myocarditis.

According to Dallas criteria[3], endomyocardial biopsy is gold standard for the diagnosis of myocarditis and capable of assessment of inflammation and determination of etiology. However, biopsy studies had highly variable results, with the incidence of myocarditis ranging from 0 to 80 percent[4] and viral genome identified only in less than 20 percent of patients with presumed myocarditis[5]. The limited sensitivity for the diagnosis of myocarditis may be explained probably by sampling error because of the focal and transient nature of inflammatory infiltrate[6-8]. Furthermore, the procedure has some risk of death and cardiac perforation. Therefore, endomyocardial biopsy is generally reserved for the patients who present rapidly deteriorated heart failure or arrhythmias and prepare to be treated with immunosuppressive therapy[9]. Biopsy was not performed in this patient.

However, the findings of $^{99}Tc^{m}$-MIBI scan, low perfusion in local area of left ventricle, indicate the decreased cell's viability and the ability to incept the nuclear element, when coronary artery disease was excluded. Combined with the symptom of fever and chest pain, elevated level of cardiac markers, ESR and CRP, the ECG ST-T changes, pericardial effusion detected by echocardiogram and exclusion of other relative systemic disease involving heart muscle, e.g., connective tissue disease, the diagnosis of acute myopericarditis can be established.

It has been generally assumed that bacterial disease is less commonly associated with myocarditis in immunocompetent hosts[10]. Fungal myocarditis is exceedingly rare. Viral infection is the most common cause of myocarditis in developed countries, and the most frequently identified viruses are the enterovirus coxsackievirus B and adenovirus[1, 11,12]. However, HSV

was also found in the biopsy samples of myocarditis patients[11, 13] and listed in the causes of myocarditis[10]. For this patient, the initial experiential treatment with a wide-spectrum anti-virus drug(ganciclovir) failed to optimally control the development of the disease. Other differential diagnose were further considered and excluded, e.g., left ventricle apical ballooning syndrome[14], vasculitis, etc. There was no indication to resort to immunosuppressive agents, since the patient did not present heart failure and arrhythmias at all. After the history of HSV-II infection was acquired, Valacicloavir, a specific anti-HSV drug, was prescribed and then the patient recovered smoothly, inferring that the HSV-II may be the causal virus of perimyocarditis in this patient. Of course, the conclusion cannot be drawn without the support of pathological and virological evidence. However, the detailed inquiry of related history is able to at least provide some clue for the diagnosis and treatment. The level of ESR and CRP was still high before the patient was discharged, although it went down obviously. We considered that he was in the process of recovery that may need a relatively long time and the inflammation response still lasts in this process.

In conclusion, myopericarditis or myocarditis is a common disease mimicking AMI, presenting chest pain, ischemic ECG changes and elevated level of cardiac markers. The differentiation of diagnosis is of great importance. Thrombolytic agents or anticoagulants may rescue AMI patients, but kill the patients with myopericarditis. To exclude coronary artery disease by coronary angiography or MDCT is prerequisite for the final diagnosis of myocarditis masquerading as AMI. The diagnosis of myopericarditis can be established without endomyocardial biopsy. Etiological finding is indispensable for successful treatment.

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