Acute myocarditis mimicking ST-elevation myocardial infarction: A diagnostic challenge for frontline clinicians

Kai Ping Ang, MRCP, Chu Zhen Quek, MRCP, Chuey Yan Lee, MRCP, Hou Tee Lu, FRCP

Department of Cardiology, Hospital Sultanah Aminah, Johor Bahru, Johor, Malaysia

SUMMARY

The clinical presentation of acute myocarditis is highly variable ranging from no symptoms to cardiogenic shock. Despite considerable progress, it remains a challenge for frontline physicians to discriminate between acute myocarditis and myocardial infarction, especially in the early phase. Our case serves as a reminder that acute presentation of myocarditis could resemble ST elevation myocardial infarction potentially misdirecting the therapeutic decision. The clinical presentation, electrocardiographic and laboratory findings of the patient are not specific enough to distinguish acute myocarditis from myocardial infarction. The gold standard tests such coronary angiography and cardiovascular magnetic resonance (CMR) can reliably differentiate the two entities.

INTRODUCTION

Acute myocarditis is an inflammatory process involving the myocardium. There are many causes of myocarditis such as viral or bacterial infection, medications, toxins and autoimmune diseases. Acute myocarditis mimics acute myocardial infarction (AMI) has been reported earlier. The clinical presentation of acute myocarditis is highly variable. Despite considerable progress, it remains a challenge for frontline physicians to discriminate between acute myocarditis and myocardial infarction, especially in the early phase.[1-3] We report a young Chinese male with acute myocarditis presented with chest pain, electrocardiographic (ECG) abnormalities and elevated biomarkers, fulfilled the diagnostic criteria of ST elevation myocardial infarction (STEMI), posing a diagnostic challenge and misdirected the therapeutic decision.

CASE REPORT

Case presentation

A 16-year-old Chinese male presented at his general practitioner at midnight with worsening retrosternal chest pain radiating to left arm for two days. He described that the chest pain was so severe that it woke him up from sleep. The chest pain was associated with difficulty in breathing and diaphoresis. He had flu-like symptoms (fever and cough) two days prior to admission. He was a secondary school student and had no past history of hypertension, diabetes or family history of premature coronary artery disease. He does not smoke nor drink alcohol, and did not use illicit drugs. He was immediately referred to a nearest district hospital for treatment. On admission, his heart rate and blood pressure were 92/min and 122/81mmHg. The temperature was normal. There were no audible murmurs, rubs or gallops during the cardiovascular examinations. There were no signs of jaundice, bleeding, rashes or congenital hypercholesterolemia. The other physical examinations were normal. His cardiac enzymes were elevated; creatinine kinase: 1134 (normal range, NR: 20-215u/L) and aspartate aminotransferase: 123 (NR: 10-40u/L) and lactate dehydrogenase: 45 (NR: 45-901u/L). The Troponin T was markedly elevated 4143 (NR: 2.5-34ng/L). Other test included: white blood count: 6.2 (NR: 3.5-12x1000/uL), haemoglobin: 15.5 (NR: 14-18 g/dl), platelet: 239 (NR: 150-400x1000/uL), urea: 3.7 (2.9-8.2 mmol/l), creatinine: 55 (NR: 50-110µmol/l), total cholesterol: 5.2 (NR: <5.2 mmol/l), low density lipoprotein: 3.1 (NR: <3.4mmol/l), high density lipoprotein: 1.7 (NR: > 0.9mmol/l), triglycerides: 1.0 (NR: 0.4 -1.7mmol/l) and random blood sugar: 5.3 (NR: 3.9-6.1mmol/l. The ECG showed hyperacute changes; ST-segment elevations at lead I, II, aVL, aVF and V4 toV6 with reciprocal changes at lead aVR and V1 to V2; indicating a diagnosis of acute inferior-lateral STEMI (Figure 1A). The chest X ray showed a normal heart size, clear lung fields with no widened mediastinum.

Based on the clinical presentation of chest pain, ECG abnormalities and elevated biomarkers, a diagnosis of acute inferior-lateral STEMI was made in accordance with the current Clinical Practice Guideline (CPG).[4] Patient was admitted to a district hospital, immediate transfer to a tertiary hospital was not possible at that time. In line with the STEMI CPG, if primary PCI cannot be performed within 120 minutes from STEMI diagnosis, fibrinolytic therapy is recommended within 12 hour of symptom onset.[4] Under the circumstances, the decision was to treat the patient with intravenous (IV) thrombolysis (IV streptokinase 1.5MU), antiplatelets and statin. After given streptokinase, the ECG showed more than 50% resolution of ST segment elevation (Figure 1B). Patient's chest pain was partially resolved after treatment and no was bleeding reported. On the next day, patient was transferred to coronary care unit in our centre. The coronary angiogram showed normal coronary arteries with no evidence of thrombus or dissection (Figure 2 A, B). Patient remained well throughout hospitalisation. The cardiac enzymes levels were declining.

A cardiac magnetic resonance (CMR) imaging was done on third day of hospitalisation. T2-weighted triple-inversion-recovery (T2STIR) CMR sequences was used to calculate the oedema ratio, T1-weighted sequences (before and after

This article was accepted: 19 August 2019
Corresponding Author: Dr. Hou Tee Lu
Email: luhoutee@gmail.com

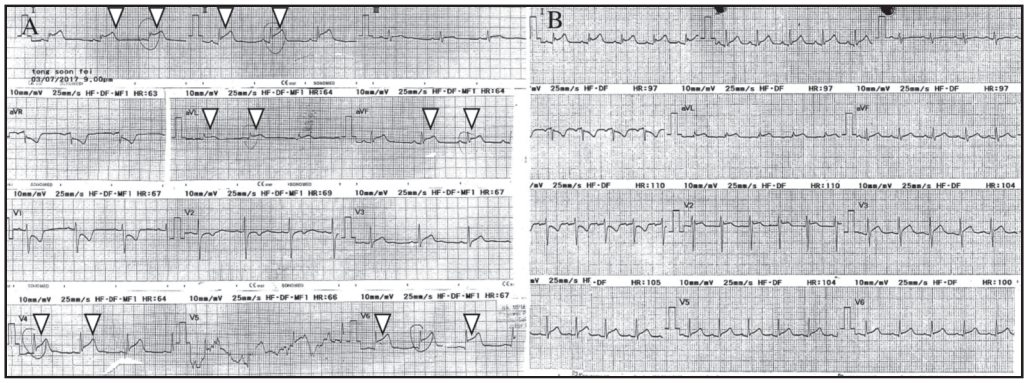

Fig. 1: (A) First ECG showing hyperacute ST-segment elevations at lead I, II, aVL, aVF and V4 toV6 (arrow), with reciprocal changes at lead aVR and V1 to V2. (B) Resolution of ST segment changes.

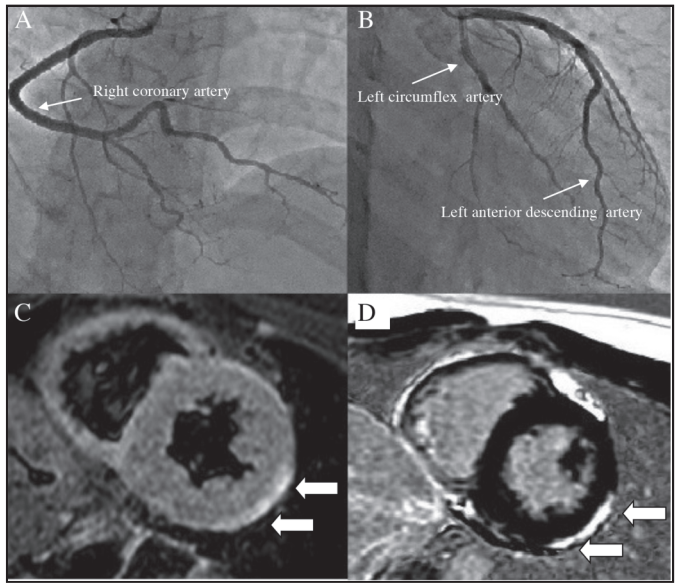

Fig. 2: (A, B) Coronary angiogram showing normal coronary arteries. (C) Cardiac magnetic resonance T2STIR sequence, showing increased signal intensity at subepicardial inferolateral territory (white arrow). (D) late gadolinium enhancement (LGE) sequence, showing gadolinium enhancement at the subepicardial inferolateral territory sparing the subendocardium (white arrow). T2STIR, T2-weighted triple-inversion-recovery; LGE, late gadolinium enhancement.

contrast agent administration) was applied to calculate the myocardial global relative enhancement (gRE). The inversion-recovery gradient-echo imaging was performed to evaluate areas of late gadolinium enhancement (LGE). As a result, subepicardial delayed enhancement was present at infero-lateral territory and T2STIR sequences demonstrated increased signal intensity with oedema. The subendocardial area was normal (Figure 2 C, D). The findings were consistent with a diagnosis of acute myocarditis. There was no pericardial enhancement on gadolinium-enhanced CMR study to indicate pericardial inflammation. Patient was treated conservatively. The antiplatelet and statin were discontinued. He was discharged after four days of hospitalization. Patient was doing well at one and six month follow up visit.

DISCUSSION

Acute myocarditis has protean presentations that span the spectrum from no symptom to cardiogenic shock. Myocarditis can present with symptoms and signs of heart failure or resemble an acute coronary syndrome (ACS), even a STEMI.[1] Our case highlights the diagnostic challenge of acute myocarditis. The presenting history of the patient and the results of conventional diagnostic tests such as ECG, biomarkers were indistinguishable from acute STEMI, misdirecting the diagnosis and treatment decision.

When making a diagnosis, the frontline clinicians seldom have immediate access to the reference or gold standard tests such as coronary angiogram. Moreover, the treatment decision is often at the discretion of the attending doctor based on clinical assessment and the available diagnostic tests. Nonetheless, a misdiagnosis of AMI or delayed treatment can result in complications or death. An integrated assessment and evaluation including medical histories, clinical presentation and results of other auxiliary tests, are necessary for the accurate diagnosis to guide treatment accordingly.

In the opinion of the authors, a careful clinical evaluation is paramount important in making a decision on treatment. A younger patient with a history of preceding viral syndrome supported a diagnosis of myocarditis. Absence of atherosclerotic coronary risk factors (smoking, diabetes mellitus, hyperlipidaemia, family history of premature coronary artery disease) is another important clue that favours a diagnosis of myocarditis. On the other hand, an older patient with severe substernal chest pain (although it is also seen in myocarditis), or a long history of typical angina preceding the acute presentation, and the presence of significant risk factors for atherosclerotic coronary artery disease suggests AMI. However, we should also bear in mind that the absence of these clinical features does not exclude either diagnoses. The clinicians should be prudent to consider fibrinolytic therapy in a young patient with no coronary risk factors and low probability of AMI because of serious bleeding side effects.

Selective coronary angiography is recommended in patients with suspected myocarditis presenting with ACS.[2] Another test for the diagnosis of acute myocarditis is endomyocardial biopsy.[5] However, endomyocardial biopsy is not readily available and its invasive character restricts its generalised application to all patients. Furthermore, endomyocardial biopsy has limited sensitivity (43-64%) with an overall complication rate of 6% and a 0.4% risk of death due to perforation.[3] Current guidelines reserve the test of endomyocardial biopsy to certain clinical scenarios such as life threatening myocarditis.

CMR permits optimal differentiation between normal and diseased myocardium with the use of gadolinium based contrast agents and special magnetic resonance pulse sequences. Imaging is performed 10-20 minutes after contrast agent application to produce late LGE images which depict diseased myocardium with excellent reproducibility, and has been suggested as a noninvasive alternative for diagnosing myocarditis.[5] The most typical finding is subepicardial delayed enhancement sparing the subendocardium, seen in non-segmental distribution in our patient. In AMI, LGE typically shows a subendocardial or transmural enhancement and the edema is typically localised to the territory of the culprit vessel. In myocarditis, edema may be either segmental or diffused. In our case, the finding of LGE sequence gadolinium enhancement at the subepicardial inferolateral territory sparing the subendocardium confirmed the diagnosis of myocarditis. However, CMR is rarely available or practical during acute presentation, and may potentially delay the time-to-treatment of ACS.

CONCLUSION

Our case serves as reminder that acute presentation of myocarditis could resemble STEMI and potentially misdirects the therapeutic decision. The gold standard tests such coronary angiography and CMR can reliably differentiate the two entities but these facilities might not be readily available at first medical contact. In retrospect, the clinical manifestation, including fever with temperature fluctuations and young age with no coronary risk factors favor a diagnosis of acute myocarditis.

CONFLICT OF INTERESTS

All the authors declare no conflict of interest.

CONFLICT OF INTERESTS

Written informed consent was obtained from the patient for publication of this manuscript and accompanying images.

FUNDING

This research received no specific grant from the government or any other funding agency in the public, commercial or not-for-profit sectors.

REFERENCES

1. Tao Z, Miao W, Wang S, Wei M, Su G, Li Z. Acute myocarditis mimicking ST-elevation myocardial infarction: A case report and review of the literature. Exp Ther Med 2015;10: 459-64.
2. Yildirim E, Bugan B, Koklu M, Gormel S, Celik M. Acute myocarditis mimicking myocardial infarction can misdirect the diagnostic approach. International Journal of the Cardiovascular Academy 2016; 2(1): 12-5.
3. Testani JM, Kolansky DM, Litt H, Gerstenfeld EP. Focal myocarditis mimicking acute ST-elevation myocardial infarction. Tex Heart Inst J 2006; 33(2): 256-9.
4. Ibanez B, James S, Agewall S, Antunes MJ, Bucciarelli-Ducci C, Bueno H, et al. 2017 ESC Guidelines for the management of acute myocardial infarction in patients presenting with ST-segment elevation: The Task Force for the management of acute myocardial infarction in patients presenting with ST-segment elevation of the European Society of Cardiology (ESC). Eur Heart J 2018; 39(2): 119-77.
5. Vöhringer M, Mahrholdt H, Yilmaz A, Sechtem U. Significance of late gadolinium enhancement in cardiovascular magnetic resonance imaging (CMR). Herz 2007; 32(2): 129-37.