CASE REPORT

Myocardial injury mimicking acute myocardial infarction due to coronavirus infection in adults with pre-existing apical hypertrophic cardiomyopathy

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Abstract

Symptoms of apical hypertrophic cardiomyopathy (ApHCM) can mimic acute myocardial infarction (AMI). Following COVID-19 infection, the elevation of troponin in ApHCM might be confusing, due to its similarity with AMI. We report the case of a 64-year-old male patient presenting with exertional dyspnoea and chest discomfort. He had no history of coronary artery disease (CAD), but his swab test was positive for COVID-19. The physical examination was normal. The 12-lead electrocardiogram showed a sinus rhythm of 78 bpm, with deep inverted T waves in leads V2 to V6, I, and aVL, and left ventricular hypertrophy. An Echocardiographic examination showed an 18 mm apical wall thickness of the left ventricle. Laboratory tests revealed elevated hs-Troponin level, but diagnostic coronary angiography was normal. The diagnostic criteria fulfilled apical cardiac hypertrophic cardiomyopathy. Coronavirus can induce atypical cardiovascular symptoms in pre-existing ApHCM. Misdiagnosis and failure to recognize may result in inappropriate therapy and delay in definitive treatment.

Keywords: Troponin, COVID-19, Echocardiography, Electrocardiography, Hypertrophy, Angiography, Dyspnea, Infarction

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Introduction

The SARS-Cov-2 infection has a wide range of manifestations and complications associated with the virus itself or secondary to inflammatory and immune responses.[1] Myocardial damage is reflected in elevated levels of high-sensitivity troponin, which is more common in severe cases and is associated with a higher inflammatory load that can trigger vascular inflammation, myocarditis, and cardiac arrhythmias. Infection with SARS-Cov-2 in patients with hypertrophic cardiomyopathy (HCM) carries a specific risk of exacerbating the patient's clinical condition, especially the typical exacerbation of ischaemic symptoms, resembling AMI.[2]

The prevalence rate in eastern countries is higher than that in western countries (25% vs <10%) and it is more common in males. Most patients develop symptoms in the fourth decade of life.[2] This case report is being presented to give a different perspective on how COVID-19 infection can induce myocardial injury in the setting of pre-existing ApHCM or Yamaguchi Syndrome.

Case Report

A 64-year-old male patient came to the ER of Dr. Soetomo General Hospital, East Java, in April 2021 with typical ischaemic heart symptoms. Initially, he felt chest pain and dyspnoea one week earlier, which was triggered by moderate-strenuous activities, and got relieved by resting. There was no comorbidity or smoking and family history for Ischaemic heart disease was absent. Physical examination revealed no abnormality with normal vital signs.

During the first admission, the patient tested positive for COVID-19. ECG evaluation showed a sinus rhythm with a rate of 78 bpm, CCWR Horizontal Axis, T inversion in leads V2-V6, I, aVL, and LVH. Chest Xray revealed cardiothoracic ratio (CTR) of 54% with a grounded heart apex. HS-Troponin I laboratory was elevated (81.9 ng/L)(N=0-0.04ng/L). He was diagnosed with Acute Myocardial Infarction (AMI) and COVID-19. Conservative treatment was opted due to lack of dedicated cath lab for COVID-19 patients in our hospital, with aspirin loading (325 mg), clopidogrel loading (300 mg), and sublingual nitrate (5mg), and discharged several days later with double antiplatelet, anticoagulant, and statin.

Four days after discharge, he was readmitted with worsening of symptoms. Non-contrast CT Scan showed bilateral pneumonia which supported a recovery phase of COVID-19 infection, while the PCR result was negative. In laboratory examination, hs-Troponin I was still high (72.9 ng/L).

Diagnostic coronary angiography (DCA) showed no

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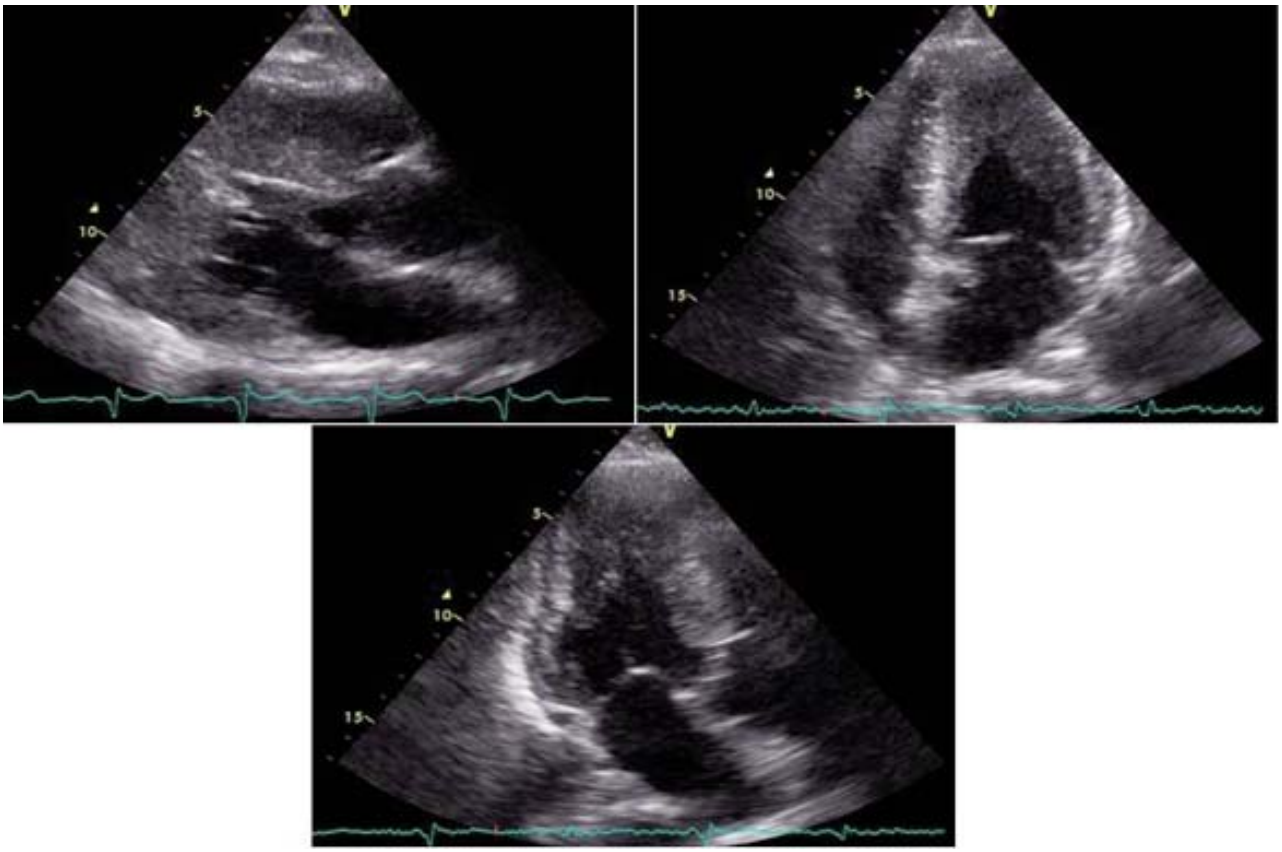

Figure-1: Transthoracal echocardiography demonstrated left ventricular hypertrophy in the apical area with an "Ace-of-spades" configuration in the diastolic phase which is one of the signs of ApHCM seen in view (PLAX; apical 4-chambers; and apical 3 chambers).

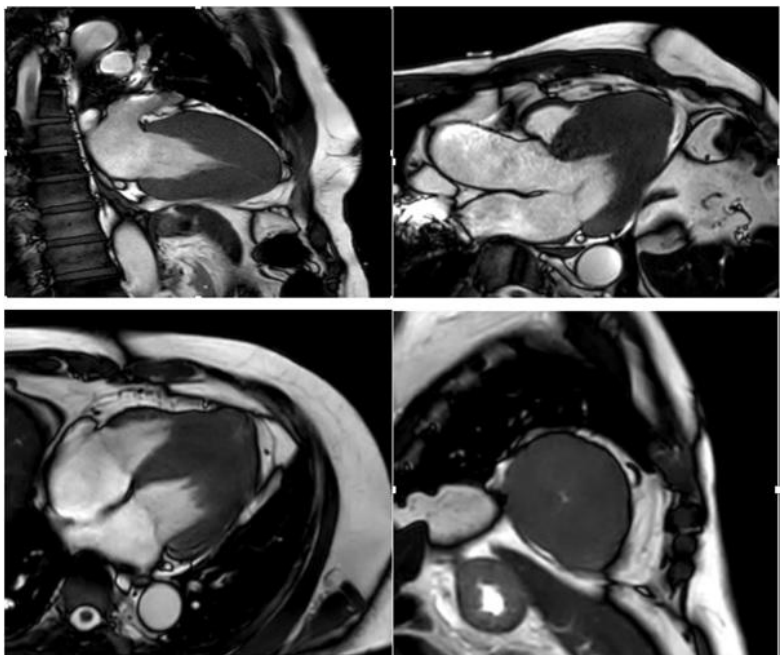

Figure-2: Cardiac MRI. Functional cine images show hypertrophic in the level of mid-apical with a maximal wall thickness of 22 mm during the diastolic phase. Systolic mid-apical obliteration, no apical aneurysm, normal systolic function (EF 62%), LV mass 219 grams, fibrosis mass 7.4 grams, no SAM, normal pericardium

significant stenosis in left main, LAD, LCx, and RCA branch, with the conclusion of a Myocardial Infarction with Nonobstructive Coronary Arteries (MINOCA). Patient received Clopidogrel, Candesartan, Furosemide, Spironolactone, Atorvastatin, and fondaparinux subcutaneous injection.

Transthoracic echocardiography (TTE) examination 1 day later showed thickening of the apical wall of the left ventricle (LV) with a thickness of 18mm, hypokinetic in the apical area with decreased LV systolic function (LV Ejection Fraction by Biplane 54%). Further, an "Ace-of-spades" configuration was also obtained from 4-chamber view (Figure 1). Then, a cardiac MRI (CMR) was performed. Functional cine images showed hypertrophy at the level of mid-apical with a maximal wall thickness of 22 mm during diastolic phase (Figure 2). Early Gadolinium enhancement was normal. Late Gadolinium enhancement showed fibrosis in mid-apical LV mid-wall and subepicardial, not related to coronary artery territory distribution.

Based on the TTE and cardiac MRI findings, the patient was diagnosed as an ApHCM so the antiplatelet administration was stopped. After 4 days of treatment, the patient had no complaints and was discharged with Bisoprolol 2.5 mg o.d, Candesartan 8 mg o.d, Furosemide 20 mg o.d, and Spironolactone 25 mg o.d.

Discussion

Almost all patients with ApHCM are often asymptomatic and identified accidentally whilst performing medical check-ups. Double carotid pulses and S4 from a non-compliant left ventricle may be present.[4]

We can suspect the diagnosis of ApHCM when we found giant T waves inversion associated with high QRS voltage at left precordial leads on ECG, which can change from time to time or can disappear. A previous study[5] involving 105 ApHCM patients revealed that 94% of patients had an abnormal ECG pattern. These included T wave inversion (93%), LVH (65%), and giant negative T waves (47%). Maximal T wave negativity correlated poorly with apical wall thickness, and ECG did not distinguish well between mixed and pure ApHCM variants. TTE can demonstrate apical hypertrophy, distinguish simple from mixed forms, and identify other prognostic features that may influence the outcome, such as the presence of diastolic dysfunction, MVOCO, or apical aneurysm. An increase in apical left ventricular wall thickness of more than 15 mm with a maximum apical-to-posterior wall thickness ratio ≥ 1.5 is considered pathological in apical HCM.[2,5] Systolic function (EF or fractional shortening) of the radial artery is usually normal or increased in patients with HCM. However, EF is a poor indicator of LV contractile performance when hypertrophy is present.[6]

CMR is more accurate at identifying coronary aneurysms in the apical location; it can also identify between 25% and 43% of anomalies missed by echocardiography. A common finding in ApHCM patients is late gadolinium enhancement (LGE). There is a positive correlation between the amount of LGE and hypertrophy severity, with the risk of heart failure and sudden cardiac death. In the absence of concurrent coronary artery disease, the LGE pattern in patients with apical HCM has distinctive features within the subendocardial pattern. This LGE "MI pattern" confirms the hypothesis that apical myocardial ischaemia is key to apical HCM.[5,7]

In COVID-19-infected patients, the disease presentation is often atypical, and might increase the difficulty in distinguishing between ACS and ApHCM leading to inappropriate therapy and delay in definitive treatment. Strict follow-up among patients with ApHCM is crucial.[8,9] Individuals with ApHCM or Yamaguchi Syndrome may be at increased risk of developing a severe illness should they contract the infection, however, the evidence is still lacking.

Conclusion

This case report showed an example of ApHCM mimicking AMI in a COVID-19 patient. The Myocardial injury could be exacerbated by Coronavirus infection which resulted in chest pain and elevated cardiac markers, therefore mimicking AMI. Fortunately, ApHCM with myocardial injury still has a better prognosis than ACS. Our case showed the importance of TTE examination and multimodality imaging in determining the etiology and assessment for this challenging case.

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