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Management of A COVID-19 Positive Patient with An Apical Hypertrophic Cardiomyopathy: A Challenging Case

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Abstract

COVID-19 is a disease caused by the SARS-COV-2 virus and was recently declared as a pandemic by WHO on March 11, 2020. The clinical presentation varies from an asymptomatic patient to a typical acute respiratory distress syndrome with a cytokines storm. COVID-19 also presents a significant threat to heart health, according to recently published research. We report in this essay a unique case presenting an unexplored apical hypertrophic cardiomyopathy, describing the management of this rare form of hypertrophic cardiomyopathy with all the challenges that can present a COVID-19 patient in the process.

Introduction

The World Health Organisation (WHO) declared on March 11, 2020 the SARS-cov-2 infection as a global pandemic. There are nearly 6,226,409 cases worldwide, including more than 373,883 deaths on June 1st, 2020(1). To date, Morocco has more than 7800 confirmed cases and crossed the limit of 200 deaths out of a total of 221,554 tests (2). The clinical presentations of the disease start from the asymptomatic patient to the acute respiratory distress syndrome COVID-19 (SARS-CoV-2 infection) interacts with the cardiovascular system on multiple levels, increasing morbi-mortality in patients with underlying cardiovascular conditions and provoking myocardial injury and dysfunction.

Apical hypertrophic cardiomyopathy (AHCM) is one of the rare cardiomyopathies, which can also present like an acute myocardial infarction (MI). Its association with COVID19 was never reported until this case. The governmental health department in its fight against COVID-19 has set up a sectional strategy in order to divide patients between the various care centers according to their COVID-19 status and the severity of the clinical picture.

A cardiology team was deployed in the level III Hospital intensive care departments; we manage the most critical patients representing along with the novel coronavirus disease outbreak a real therapeutic challenge. We report a unique case of association of COVID-19 infection and ACHM, emphasizing the therapeutic difficulties encountered.

Presenting concerns

A 47-year-old male patient declared COVID-19 positive after a nasopharyngeal swab (PCR type) was admitted in a secondary center and underwent the national anti-COVID protocol including the association Hydrochloroquine-Azithromycin.

The patient complained about stable angina associated with palpitations 4 months ago. He was a heavy tobacco smoker, no personal or family history of heart disease, no sudden cardiac arrest or heart rhythm disorder were reported, neither a prior treatment to diagnosis.

Three days after his admission the patient experienced precordial pain at rest and was referred the same day, to our tertiary care hospital.

Clinical findings

At admission his Glasgow Coma Scale (GCS) was 15/15, eupneic with an oxygen saturation SpO2 at 98% in ambient air. The patient was hemodynamically stable, his blood pressure (BP) was 140/80 mmHg symmetrical, regular rhythm at 75 beats per minute (bpm), the rest of the clinical examination results were normal.

Diagnostic focus

An electrocardiography (ECG) examination at admission was performed because of patient's cardiovascular risk factors and his chest pain. It revealed normal sinus rhythm and a left ventricular hypertrophy (LVH) with symmetrical deep negative T waves and ST segment depression in leads V3-V6, II, III and aVF (Fig.1). QTc interval was estimated at 447ms.

There were no prior electrocardiograms available for comparison. The early troponin HS level at admission was within the normal range (11,5ng/l).

At that point, two diagnosis were retained: firstly, an acute coronary syndrome considering the symptoms and his smoking status and, secondly, a hypertrophic cardiomyopathy taking into account the cardiac electrical aspect. We kept in mind that the association between the two can't be ruled out.

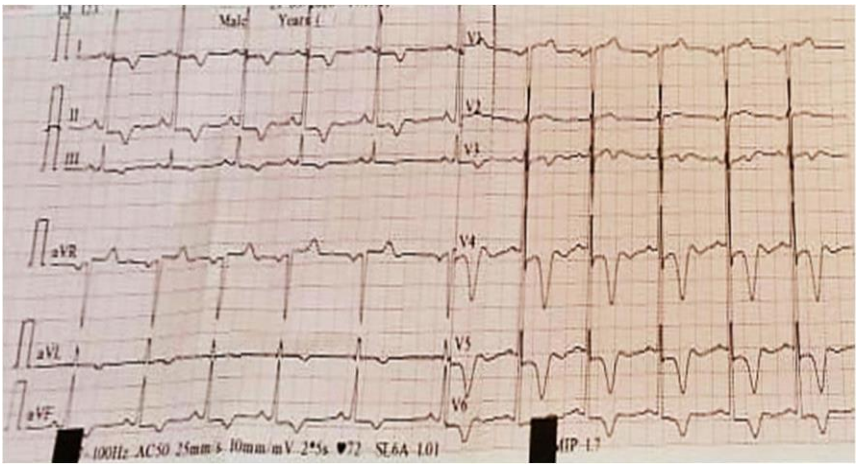

Figure 1: ECG shows normal sinus rhythm, left ventricle hypertrophy and marked T-wave inversions and ST segment depression in leads V3-V6, II, III and aVF.

The echocardiography, within easy reach, showed an asymmetrical concentric hypertrophy that was more prevalent in the apical region with an Ace-of-spades sign in the four-chamber view (Figure 2A, B).

The global ejection fraction remained preserved, with diastolic dysfunction. The mid-cavity gradient was around 32mmHg (Figure 2C).

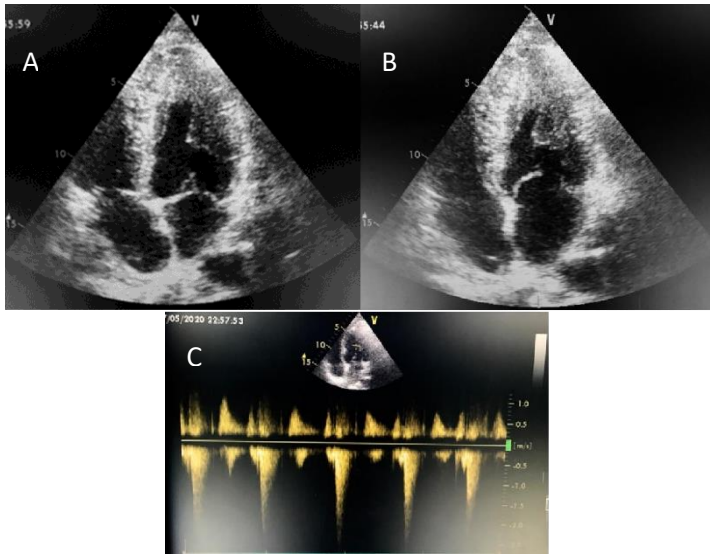

Figure 2: A- Transthoracic echocardiography, four-chamber view. Patient with AHCM. Note the increased thickness of the apex of the left ventricle. B- Hypertrophied papillary muscle narrowing the LVOT. C- Doppler recordings of LVOT gradient.

Therapeutic focus and assessment

In lights of persistence of the symptoms and our patient risk profile, decision was made to take him immediately to cath lab. Coronary angiography via the radial artery using a 5 French diagnostic catheter showed preserved ejection fraction as well as angiographically normal coronary arteries (Fig. 3 A, B, C).

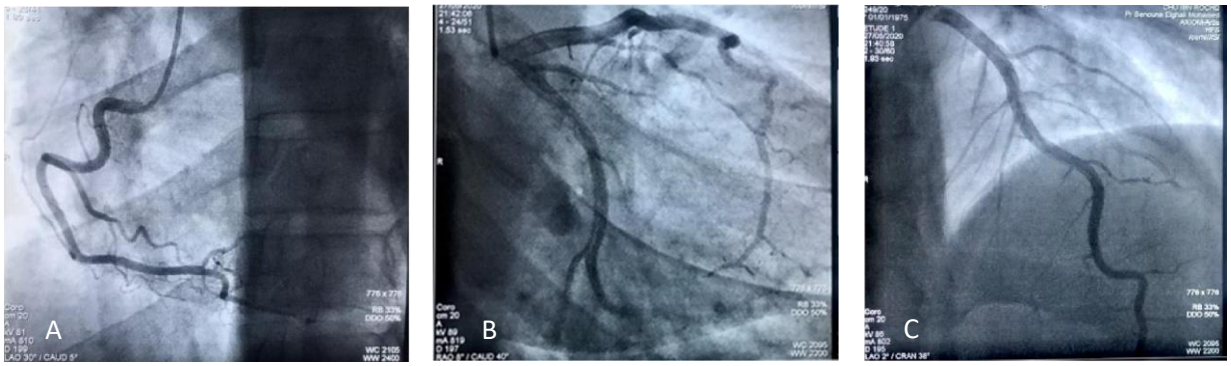

Figure 3: A- Right coronary angiogram. LAO straight. B- Left coronary angiogram. RAO caudal. C- Left coronary angiogram. PA cranial.

The patient was closely monitored in the emergency care unit with proper rehydration and transferred after full pain resolution.

Azithromycin and chloroquine were withheld as well as the beta-blockers due to a low heart rate afterwards (50bpm). An exercise stress echocardiography, to assess intraventricular gradients, will be scheduled after negativation of the COVID-19 PCR-test.

Case Discussion

Chest pain is a leading cause of presentation in the emergency department, even in the context of the Covid- 19 epidemic. In fact, patients who are confirmed or suspect COVID-19 positive and who have cardiovascular risk factors, it is recommended to look for chest pain or equivalent during the examination, to assess the clinical status and to perform an ECG.

In fact, the acute systemic inflammatory reaction caused by the SARS-COV-2 virus associated with parietal biomechanical stress and vasoconstriction are responsible of the destabilization and rupture of atheroma plaques with a higher risk in case of cardiovascular risk factors and previous events.

The severe hypoxia and the cytokine inflammatory storm, encountered in COVID-19 disease promote coagulation and formation of coronary arterial emboli [3].

These two phenomena lead to the occurrence of acute coronary syndromes with or without ST elevation, which explains our medical reasoning and the approach undertaken in this case.

The availability of the transthoracic echocardiography while transferring the patient to the cath lab revealed an underlying structural heart disease.

The apical hypertrophic cardiomyopathy is a rare variant of hypertrophic cardiomyopathy (HCM). Two phenotypes have been described: a “pure form” phenotype (apical segments only) and a more inclusive “mixed form” (hypertrophy extending into “other” segments) which carries a worsened prognosis.

Patients can present with symptoms of angina and dyspnea with a predominant exertional component. Myocardial ischemia could happen in the presence of normal coronaries in angiogram like our case.

Although the exact mechanism is unknown, the underlying pathophysiology is a mismatch of supply and demand secondary to the decreased vasodilator capacity, delayed relaxation of myocardium, decreased capillary-to-myocardial fiber ratio and reduced coronary perfusion pressure [4,5].

It is also possible that the infarction is related, at least in part, to the abnormal intramural coronary arteries. These abnormal vessels have thickened walls due to intimal proliferation or medial hypertrophy, or both and narrowed lumens [6,7].

Apical hypertrophic cardiomyopathy is often first detected by transthoracic echocardiography. Typical echocardiographic examination signs include increased thickness of the left ventricular wall in the apical region. The characteristic spade-like configuration of the left ventricle in the four-chamber view. Assessment of the extent of left ventricular hypertrophy is a very important part of the examination and allows types of AHCM to be differentiated.

The Doppler assessment of possible interventricular gradients is of clinical importance and should be performed in every case. Detection of the diastolic gradient between the apex and the cavity of the left ventricle also plays an important prognostic role. It was found to increase thromboembolic risk, ventricular arrhythmias or perfusion abnormalities. In most cases of AHCM global ejection fraction remains preserved, with significantly reduced end-diastolic volumes and signs of diastolic dysfunction. Some patients might actually develop an apical aneurysm that can also be detected, particularly with echocardiography contrast agents [4,8,9].

If echocardiographic images are inadequate, cardiac magnetic resonance imaging may be used to diagnose apical hypertrophic cardiomyopathy.

Cardiac MRI studies using delayed gadolinium myocardial enhancement indicative of myocardial fibrosis showed apical subendocardial scar and intramural fibrosis in nonthickened segments in many patients who had minimal symptoms and a pure apical phenotype. Left ventricular apical "outpouchings" in AHCM are not uncommon and might or might not be related to subendocardial ischemia from pressure overload or other coronary perfusion and metabolic abnormalities. In some patients, a true apical transmural scar can develop in the absence of epicardial coronary artery disease, which can increase the risk of stroke or ventricular arrhythmia [10,11].

The medical treatment of patients with apical form of HCM is similar to therapy used in the typical form of HCM. The use of a β-blocker or calcium channel blockers plays a beneficial role in patients with preserved ejection fraction and diastolic dysfunction by prolonging the diastole.

For special forms of hypertrophic cardiomyopathy such as AHCM a unique procedure performed in a few centers has been described. The aim of the surgical procedure is to increase the end-diastolic volume of the left ventricle by performing a transapical myectomy [8].

A less invasive technique has been proposed as an alternative to surgery for patients who fail to respond to pharmacologic therapy. Alcohol septal ablation in mid-ventricular obstructive hypertrophic cardiomyopathy and AHCM is not classical and wide accepted treatment in contrast to the subaortic type obstruction.

The most important problem is the identification of the target vessel, therefore a detailed evaluation of the distribution of the first septal branch is necessary [12].

In our case, the association of Hydrochloroquine and Azithromycin had to be terminated due to high risk of cardiac arrhythmia, conduction disorders and other heart injuries (3,13,14). Such life-threatening abnormal heart rhythms are a common finding in patients with HCM.

The risk of sudden cardiac death is lower in AHCM as compared to typical phenotype of HCM especially with left ventricular outflow tract obstruction. Therefore, implantation of an implantable cardioverter defibrillator (ICD) is only recommended for high risk patients with a previous cardiac arrest, a family history of sudden cardiac death, syncope, asymptomatic non-sustained ventricular tachycardia (NSVT) on 24h ECG monitoring, an abnormal blood pressure response to exercise, as well as left ventricular wall thickness > 30 mm measured in the best available imaging method [8,9].

Conclusion

Management of acute chest pain with underlying structural heart disease in light of the COVID-19 global pandemic is a real challenge. When formulating a differential diagnosis for chest pain, it is important to include apical and other variants of hypertrophic cardiomyopathy as part of that differential.

In this case report, the national anti-COVID protocol including the association Hydrochloroquine- Azithromycin as well as the beta-blockers were suspended for safety reasons.

Disclosure

The authors declare that they have no conflicts of interest.

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