An abrupt cardiogenic shock in a female at the era of the COVID 19 pandemic lockdown

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Abstract

We reported the challenging diagnosis of Takotsubo syndrome TTS in a female, admitted to the intensive care unit, with cardiogenic shock, and we highlighted the impact of Levosimendan use in the salvage of this patient.

INTRODUCTION

Takotsubo cardiomyopathy or Takotsubo syndrome (TTS), also known as stress cardiomyopathy, is a type of non-ischemic cardiomyopathy in which there is a sudden temporary weakening of the muscular portion of the heart (1). It usually appears after significant physical or emotional stressors (2). We reported the case of TTS complicated by Cardiogenic shock during the COVID-19 pandemic lockdown

CASE REPORT

We report the case of a 73-year-old woman with a history of Hypertension treated by lercanidipine/ enalapril 20/20, Paroxysmal atrial fibrillation under bisoprolol 10mg+ flecainide (lp) 100mg/d, and acenocoumarin and Hypothyroidism under Levothyroxine 50ug/d, presented to the emergency unit because of sudden onset of progressive dyspnea without chest pain. On clinical examination, she didn't present any shock signs nor fever. The heart auscultation revealed regular rhythm without murmurs. The pulmonary exam under oxygen therapy with facemask showed tachypnea, Oxygen saturation at 78%, and diffuse crepitations.

On the electrocardiogram, the rhythm was sinus, with a significant ST-elevation in D1 AVL leads, a poor R-wave progression and a normal QT interval 440ms. Rapidly the patient developed a cardiogenic shock.

A Mechanical ventilation and inotropes were initiated, and the patient was transferred to the Cath lab. The coronary angiography revealed a 40% atheromatic

plaque in the distal left anterior descending artery. The laboratory tests showed a slight elevation of Troponin US (first point=0.065 ng/ml, second point 6 hours later was 0.089 ng/ml), an acute kidney injury with a creatinine of 176 µmol/l and a normal CRP.

The Chest X-ray showed a diffuse pulmonary edema.

The transthoracic echocardiography (TTE) showed signs of hypertensive cardiopathy, with low left ventricular ejection fraction (LVEF) at 15% and akinesia of mid-apical myocardial segments associated with hyperkinetic basal segments (apical ballooning) (figure 1). There wasn't a left ventricular outflow tract obstruction (LVOTO) or a systolic anterior motion of the mitral leaflet. There was no pericardial effusion. The left ventricular twisting on 2D speckle-tracking imaging was reduced with diminished left ventricular longitudinal strain (GLS -10%) (Figure 2 A).

Twelve hours later, there was a T-wave inversion in the anterior leads with a prolonged QTc interval (501ms) (Figure 3). Given these electrical modifications, the echocardiographic findings, the low and stable troponin elevation contrasting with severe LV injuries, the Takutsubo syndrome was the most likely diagnosis (The Inter TAK score was calculated at 75 points >70 points).

ECMO wasn't available in our hospital. The decision was to switch to the Ca2+-sensitizer levosimendan as an alternative to the catecholamines. Few hours later, we obtained hemodynamic stabilization. And after 4 days, the LVEF has remarkably improved (LVEF 50%) (figure 2B)

At 1-month follow-up, the patient demonstrated remarkable recovery with New York Heart Association (NYHA) Class I symptoms. She continued with outpatient psychologic rehabilitation. At that time, TTE demonstrated normalization of cardiac function with LVEF of 60% and complete recovery of the wall motion abnormality.

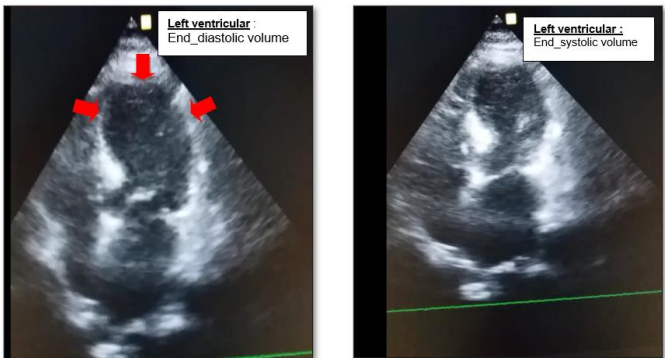

Figure 1: *The echocardiography*

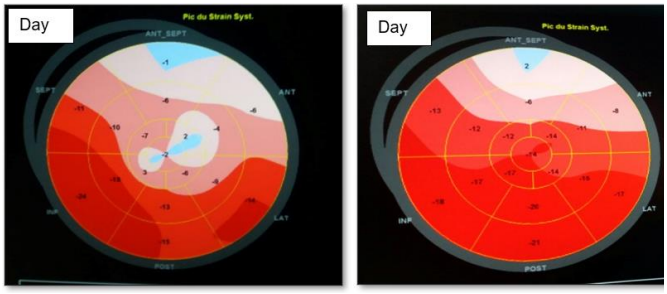

Figure 2: *Left ventricular twisting on 2D speckle-tracking imaging (Day 3-Day 7)*

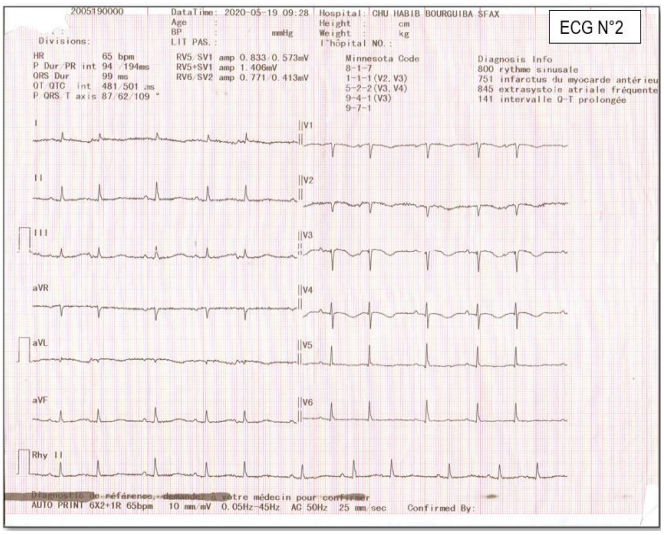

Figure3: *The electrocardiogram*

DISCUSSION

Many cardiovascular consequences of COVID-19 have been described in literature, including myocardial injury, myocarditis, acute coronary syndromes, pulmonary embolism, stroke, arrhythmias as well as heart failure. (1)

This case report illustrated an unusual cause of cardiogenic shock during the first wave of COVID-19, due to the psychological impact of the lockdown.

Takotsubo syndrome is a working diagnosis, and we need many arguments to establish the diagnosis (2).

Initially, we found ST elevation, so it was necessary to practice an emergent coronary angiography to exclude acute myocardial infarction (AMI), and this exploration didn't reveal significant coronary artery stenosis. Few hours later, we noticed progressive T-wave inversion and QT interval prolongation reaching 501ms, which was very suggestive of TTS. The transthoracic echocardiography was very contributive to retain the diagnosis, it showed an Apical ballooning with akinesia of mid-apical myocardial segments associated with hyperkinetic basal segments which extend beyond the distribution of a single coronary artery territory.

Given the findings of coronary angiogram, electrocardiogram, and echocardiography, and in the absence of troponin elevation, TTS was the most likely diagnosis and confirmed after follow-up echocardiography.

In this case, there weren't <red flags> of acute infectious myocarditis (Signs and/or symptoms of viral infections - Elevated

CRP and Pericardial effusion) that's way, cardiac magnetic resonance (CMR) wasn't performed (3). The second challenge in this case report was the treatment.

How did we manage this severe cardiogenic shock?

With Catecholamine (dobutamine and noradrenaline) infusion, the cornerstone in pharmacologic treatment for cardiogenic shock, we didn't notice any amelioration for our patient who presented pulmonary oedema and a refractory shock. Thus, according to the last International Expert Consensus Document on Takotsubo Syndrome, catecholamine should be avoided in TTC, levosimendan is recommended as a safe and effective non-catecholamine inotrope in managing TTS complicated with heart failure.

In our case, levosimendan infusion was well tolerated; and we noted a good clinical improvement.

Levosimendan is a molecule with both inotropic and vasodilator action, with low rates of adverse events. The main mechanism of action is the increase in the troponin C affinity for Ca2+ and the stabilization of troponin C conformation.

The main mechanism of increasing myocardial contractility is based on the increased sensitivity of cardiac troponin C towards intra-cytoplasmic calcium.

The vasodilatory properties lead to a dramatic increase in cardiac output with a concomitant reduction in cardiac filling pressures in the failing heart enabling it to generate more efficient systolic and diastolic functions.

CONCLUSION

Cardiac involvement while the Covid-19 pandemic may manifest atypically. We report the diagnostic challenges with severe cardiogenic shock secondary to the psychological impact of the lockdown on our patient.

This case report highlights the beneficial role of levosimendan in managing TTS with cardiogenic shock as an alternative to mechanical support.

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