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Peripartum Cardiomyopathy: An Atypical Presentation Of COVID-19 Infection

EL Ghali Mohamed Benouna (MD)[1], Amine Ech-chenbouli (MD)[1], Hajar Bendahou (MD)[1] Othmane Benmallem (MD)[1], Salim Arous (MD)[1], Rachida Habbal (MD)[1], Ouissal Aissaoui (MD)[2*], Said Salmi (MD)[2], Chafik kettani (MD)[2], Lahoucin Barrou (MD)[2], Hicham Afif (MD)[3]

[1]Department of Cardiology Ibn Rochd university hospital; Medicine and Pharmacy Faculty - Hassan II university Casablanca Morocco
[2]Department of Anesthesia and Reanimation Ibn Rochd university hospital; Medicine and Pharmacy Faculty - Hassan II university Casablanca Morocco
[3]Department of Pulmonology Ibn Rochd university hospital; Medicine and Pharmacy Faculty - Hassan II university Casablanca Morocco

[*]**Correspondent author:** Ouissal Aissaoui, Department of Anesthesia and Reanimation Ibn Rochd university hospital; Medicine and Pharmacy Faculty - Hassan II university Casablanca Morocco. Email: aissaoui.wissal@gmail.com; ORCID : https://orcid.org/0000-0001-8353-8786

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Abstract

Sars-cov-2 is a pandemic infection by the coronavirus called Covid19. They are more than 3 million of covid 19 positifs patients with more than 200000 deaths related to this virus till this day, all over the world. PPCM refers to an uncommon form of idiopathic heart failure that occurs during pregnancy or until 5 months postpartum, her association with COVID-19 was never reported until this case. We report a case of 19 years old women who experienced a positive test of COVID-19 associated with peripartum cardiomyopathy 6 days after delivering a twin. Her condition being aggravated she was put on artificial respiration and received a prolactin inhibitor with hydroxycholoroquine associated with azithromycin with a good clinical course. The viral origin of peripartum cardiomyopathy was often raised. This case suggests although the SARS-COV-2 virus may be responsible for myocarditis which is associated with a context of peripartum can be extremely severe.

Keywords: *COVID-19, Peripartum cardiomyopathy; hydroxychloroquine.*

Introduction

COVID-19 is a pandemic infection by the coronavirus called Covid19. They are more than 3 million of covid 19 positifs patients with more than 200000 deathsrelated to this virus till this day, all over the world [1]. The classical clinical picture of COVID-19 is that of a flu-like syndrome of mild severity in most cases, but in 15% of cases it is complicated by interstitial pneumonia and a variable degree of respiratory failure [2]. Among COVID-19-positive patients, there are those that consult the emergency department for heart symptoms, such as palpitations or chest pain rather than respiratory problems [3]. Little is known about a possible relationship between COVID-19 and cardiovascular diseases. Periapartum cardiomyopathy (PPCM) was not described until now.

PPCM refers to an uncommon form of idiopathic heart failure that occurs during pregnancy or until 5 months postpartum [4-5]. While some patients experience favorable clinical outcomes, serious cases require assisted circulation and cardiac function can deteriorate to the point that a heart transplant is necessary.

In this paper we report a peripartum cardiomyopathy associated with a SARS-COV-2 proved infection in a young patient of 19 years old; whish is to the best of our knowledge the first case of PPCM associated with SARS-COVID-19.

Case Report

We report the case of a 19-year-old primigravida, with no particular pathological background, or any particular medication, who presented 6 days after vaginal delivery of a twin an acute respiratory distress without chest pain evolving in a febrile context. The clinical examination on admission found a conscious patient, febrile at 38.5, normotensive at 118 / 76cmHg, polypneic, her heart rate was at 120 beats per minute her oxygen saturation was 93% under high concentration mask. On Cardiovascular examination we found a mitral regurgitation murmur, and crackling groans on auscultation. The obstetric

examination was without particularities. The electrocardiogram showed sinus tachycardia without disturbances of ventricular repolarization and without signs of myocardial ischemia. The front chest X-ray visualized cardiomegaly with an interstitial syndrome and signs of pulmonary vascular overloads of the type of acute pulmonary edema supplemented by thoracic CT objectifying infectious lesions with a frosted glass appearance in favor of an infection with COVID- 19. Faced with this aspect at chest CT, a PCR was requested returning positive. Transthoracic echocardiography was performed objectifying an aspect of dilated cardimyopathy with a severe impairment of the left ventricular systolic function estimated at 15%. The telediastolic diameter of the left ventricle was measured at 61mm (Figure 1A). Severe functional mitral insufficiency was associated (Figure 2B). Pulmonary systolic pressure was high at 55 mmhg and the inferior vena cava was dilated at 23mm, there was also a pericardial effusion. The right heart showed a normal systolic function.

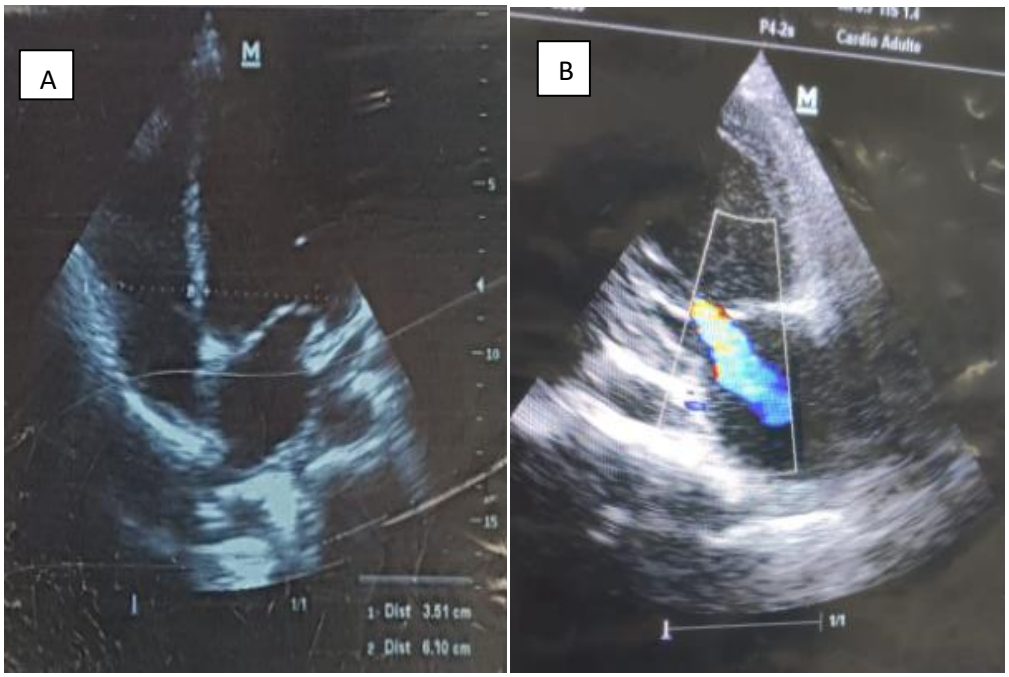

Figure 1: (A) Echocardiography four chamber view showing dilatation of the left ventricle; **(B)** Echocardiography two chamber view showing moderate mitral regurgitation.

The biological balance sheet showed an elevation of cardiac enzymes: troponin:46ng/L, CPK: 473UI/L, BNP: 2168pg/ml, and an inflammatory syndrome (CRP at 115 mg / l and Pct: 0.51), lymphopenia at 815/µl, and D-dimers at 870µg/l. Therefore, we concluded to a severe form of peripartum cardiomyopathy associated with an infection by SARS 2- COVID-19 virus.

After 2 days of hospitalization; invasive ventilation was performed for Respiratory and hemodynamic instability, her blood gas showed a severe respiratory acidosis with hypoxemia and hypercapnia (PH:7.32, Po2:52.3mmhg, Pco2:44mmhg). Treatment consisted of dobutamine and noradrenaline associated with two specific treatments: Dostinex a prolactin inhibitor for her peripartum cardiomyopathy and the Moroccan protocol for treatment of COVID 19 patients associating hydroxycholoroquine 200 mg x3 per day; azithromycine500mg per day, corticotherapy ,zinc and vitamin C.

The evolution was favorable after 48 hours of intubation, normal parameters of oxygen saturation; blood pressure and blood gas pressures were achieved and radiological improvement resulting of the resolution of the lung oedema on the chest x-rays of control. Echocardiographic control showed an improvement in cardiac function with an increase of the left ventricular systolic ejection fraction to 35%, a regression of mitral insufficiency and low filling pressures, with Pulmonary pressure in control was at 25 mmHg, and an inferior Vena cava at 14mm.

Discussion

From January 2020, coronavirus disease (COVID-19) has spread fast from China, mainly to Southwest Asia and Europe, especially to Italy, and it is now found everywhere around the world. The disease is caused by Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-CoV-2), the seventh member of the coronavirus family which infects humans and to which Middle East Respiratory Syndrome Coronavirus (MERS)-CoV and SARS-CoV also belong [6]. Diagnostic criteria for PPCM have been previously proposed [4, 7]. These criteria include left ventricular EF < 45–55%, fractional shortening < 30%, the lack of any previous history of heart disease, and the lack of other potential causes of heart failure. In this way, PPCM diagnoses are dependent on exclusion criteria that preclude other identifiable causes of heart failure in women without any preexisting heart disease [7]. Our case matched all of these criteria.

The differential diagnosis of potential PPCM cases should include ischemic cardiomyopathy, dilated cardiomyopathy, and myocarditis. The careful examination of family and patient histories is therefore particularly important for

PPCM diagnoses. Preeclampsia has been reported to occur in 30% of PPCM cases [8] and should therefore be included as a potential indicator of PPCM as part of the differential diagnostic process. However we couldn't establish the history of pregnancy in our patient since she was not medically followed.

Many pathophysiological hypotheses have been proposed to explain this pathology, namely a poor adaptation to hemodynamic changes in pregnancy linked to an increase in cardiac output, an increase in plasma volume and an alteration in peripheral vascular resistance. The Hilfiker-Kleiner team pointed to the role of a peptide derived from the cleavage of prolactin, the lactation hormone [9]. It has, in fact, been shown that the increase in intramyocardial oxidative stress increases the enzymatic activity of cathepsin D and its release into the blood circulation, responsible for the cleavage of prolactin leading to the release of a peptide of 16 kDa. This peptide has anti-angiogenic and proapoptotic properties, responsible for an inadequacy of myocardial vascularization and contractile dysfunction of cardiomyocytes. Other studies suggest the involvement of myocarditis lesions of viral origin [10]. Wish we think is the most likely etiology in our patient since she had a proved infection of SARS-COV-2.

An early report on 99 patients hospitalised from 1st to 20th January 2020 at Jinyntan Hospital, Wuhan, China, for SARS-CoV-2-related pneumonia, shows that pre-existing cardiovascular disease was present in 40% of them [10]. A second report from the same period of time on 138 patients hospitalised at Zhongnan Hospital of Wuhan University shows that 26% of the patients required cardiologic intensive care. Among these patients, 16.7% developed arrhythmias and 7.2% experienced an acute coronary syndrome in addition to other complications [12]. Furthermore, patients diagnosed with pneumonia due to SARS-CoV-2 infection showed an increase in high-sensitivity cardiac troponin I levels, suggesting myocardial injury [13]. Other published and anecdotal reports indicate the presence of myocarditis, cardiac arrest, and acute heart failure in SARS-CoV-2- infected patients. It is not clear whether these cardiac conditions are provoked by SARS-CoV-2 or are complications typical of any other pathology with higher cardio-metabolic demand, and thus unrelated to the viral infection.

Myocarditis was the first recognized as a cause for PPCM 30 years ago, but the reported incidence rate of myocarditis is high variable over the years. Sanderson showed that 45% PPCM patients could suffer from myocarditis by histological detection [13]. Another study showed supportive evidence on this association [15]. Moreover, a 62% incidence rate of myocarditis was found in PPCM patients [16].

Viral infection is one of common causes of myocarditis. It could also contribute to the pathological changes of PPCM. Similar to the autoimmune attack theory, autoantibodies against hear tissue can develop after episode of viral infection. EpsteinBarr virus (EBV), human cytomegalovirus (CMV), human herpes virus 6 (HHV6) and parvovirus B19's viral genomes have been found in biopsy specimens in 31% PPCM patients [16]. The ejection fraction of PPCM patients can be improved from 50.2 to 58.1% by anti-viral therapy, while the ejection fractions of those with persisted viral titers are decreased from 54.3 to 51.4% [18]. Animal study further confirmed this theory by providing the result that pregnant mice can develop worse myocarditis after experimentally infected with echovirus or coxsachievirus than those non-pregnant mice [16]. Among these known viruses, this case report suggests that SARS-COV-2 may cause similar effects on the heart of pregnant women.

Conclusion

The viral origin of peripartum cardiomyopathy was often raised. This case suggests although the COVID-19 virus may be responsible for myocarditis which is associated with a context of peripartum can be extremely severe. It also shows that in the pandemic context that we are currently experiencing any respiratory or cardiac sign presented by a woman in the peripartum period must push us to make a cardiac investigation simultaneously with a SARS -COV-2 infection screening.

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