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COVID-19 Recovered Parturient with Severe Pulmonary Hypertension Undergoing Caesarean Section

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ABSTRACT

A 30-year-old COVID-19 recovered pregnant lady presented to us for severe pulmonary hypertension. An emergency caesarean section was performed in view of severe pulmonary hypertension under combined spinal epidural anaesthesia with special precautions including inhaled nitric oxide and extracorporeal membrane oxygenation standby. The surgery was uneventful, and the patient was diagnosed with idiopathic pulmonary hypertension and managed with medical treatment subsequently. This report describes the anaesthetic management of caesarean section in this high-risk patient and reviews the latest evidence regarding the association between COVID -19 infection and pulmonary hypertension.

Keywords: COVID-19; Pulmonary hypertension; Pregnancy; Case Report

INTRODUCTION

Pulmonary arterial hypertension is classified as class IV (extremely high risk) according to the modified World Health organisation (WHO) classification in pregnant women with congenital and acquired heart disease. Management of these high-risk patients for delivery requires expertise and special precautions. There is also increasing evidence showing the relationship between COVID-19 infection and pulmonary hypertension which shall be reviewed in this article.

CASE PRESENTATION

On 23 April 2022, a 30-year-old, previously healthy pregnant woman (Weight: 70.9kg, Height: 152cm, Body Mass Index: 30.7, 32+3 weeks gestation) was referred to the obstetric unit by a private doctor for shortness of breath. She had a previous caesarean section for failed induction 1 year ago, uneventfully with a foetal birth weight of 3kg. She was infected by COVID-19 virus at 25 weeks gestation with her first rapid antigen test result positive on 28th February 2022 with no prior vaccination. She had fever and upper respiratory tract infection (URTI) symptoms and visited COVID-19 designated clinic on day 10 and was prescribed with symptomatic treatment only e.g.,

paracetamol.

She had exertional dyspnoea and orthopnoea with occasional palpitations on presentation but no chest pain or syncope. Cardiac and respiratory examinations were unremarkable with normal blood pressure and oxygen saturation except for a heart rate of 108 bpm. There was no ankle oedema or calf swelling. The foetus was appropriately sized with normal movement. Haemoglobin, liver, renal function, and troponin I levels were unremarkable. The electrocardiogram (ECG) showed sinus tachycardia, a new T wave inversion in V1-3, and an S1Q3T3 pattern, raising the suspicion of pulmonary embolism (PE). An urgent computer tomography scan of the pulmonary artery (CTPA) showed prominent pulmonary trunk but no evidence of PE. Serial ECG, blood gas, and troponin I results were normal. The patient discharged herself against medical advice the same day.

An outpatient echocardiogram performed on 13th May 2022 showed a D-shaped left ventricle, impaired right ventricular systolic function with dilated right ventricle, and right atrium, suggesting severe pulmonary hypertension. The right ventricular systolic pressure was 60-65mmHg (estimated) with moderate to severe tricuspid regurgitation. Left ventricular ejection fraction was normal. The patient was admitted to the labour ward for observation. A repeat CTPA showed a supra-cardiac partial anomalous pulmonary venous return but no evidence of PE. Elevated plasma NT-proBNP (496ng/L) suggested heart failure.

In view of severe pulmonary hypertension and impaired ventricular function, an emergency caesarean section was performed on the 14th May 2022 (Gestation 35+3 weeks) under combined spinal epidural anaesthesia (CSE) by senior anaesthetists, obstetricians and midwives. Intensivists and paediatricians stood by with extracorporeal membrane oxygenation (ECMO), nitric oxide, high flow nasal oxygen (HFNO), vasopressors and inodilators prepared. Large bore peripheral cannula and invasive monitoring were placed under local anaesthesia followed by insertion of a lumbar epidural catheter and intrathecal injection of bupivacaine in dextrose and fentanyl. Noradrenaline infusion and HFNO were used to maintain the patient's blood pressure and oxygen saturation. Delivery was within 6 minutes of incision with a foetal birth weight of 3.09kg. Apgar score was 8 on 1 and 5 minutes after birth. 1g of tranexamic acid was given for haemostasis and oxytocin was omitted to avoid worsening pulmonary hypertension. Sterilisation was performed. The surgery concluded within 1 hour. Estimated blood loss was 340ml. Epidural morphine and levobupivacaine were given at the end of surgery.

She was transferred to the ICU and remained hemodynamically stable. She was discharged to the postnatal ward after three days and the EA catheter was removed. She regained the ability to walk on level ground without assistance and was discharged on post-op day 7.

Further workup showed no family history of cardiovascular or autoimmune disease. There were no signs or symptoms of autoimmune disease, and the autoimmune markers were all negative. Serum HIV marker was negative. There was no suspicious medication history upon query. Her chest X-ray and lung function test was normal. Lung ventilation perfusion scintiscan excluded chronic thromboembolic pulmonary hypertension. Cardiac MRI scan showed no evidence of myocardial infiltration, infarct, or scarring. Right Heart catheterization 1 month post-delivery showed idiopathic precapillary pulmonary hypertension (RAP 19mmHg, PA 57/28mmHg, Mean PA 39mmHg, PCWP 9mmHg, PVR 12WU) and found the supracardiac partial anomalous pulmonary venous drainage to be hemodynamically insignificant. The patient was prescribed sildenafil and eplerenone. Follow up 4 months post-

delivery showed reduced exercise tolerance of 1 flight of stairs with the 6-minute walk test of 337 metres. Fortunately, follow-up right heart catheterization 8 months post-delivery showed good response to medical treatment (RAP 9mmHg, PA 41/11mmHg, Mean PA 26mmHg, PCWP 14mmHg, PVR 2WU) and her exercise tolerance was much improved. She will continue medical treatment and be monitored by the cardiologist for the disease's progress.

DISCUSSION

This is the first case of possible COVID-19 associated pulmonary hypertension in a pregnant woman we are aware of in Hong Kong. In this case, we hypothesise the unexpected rapid development (less than 2 months) and recovery of pulmonary hypertension in an otherwise healthy pregnant woman could be related to COVID-19 infection. The result from right heart catheterization showed evidence of precapillary pulmonary hypertension and common causes were excluded from the workup above. It should be classified as idiopathic pulmonary arterial hypertension, yet there is increasing evidence showing COVID-19 infection can lead to pulmonary hypertension as shown below.

The prevalence of pulmonary hypertension (12%) and right ventricular dysfunction (14.5%) were high among 200 consecutive patients with COVID-19 who were admitted to non-ICU care units in Milan.[1] Another study from Sweden reported 39% of patients (67 patients in total) with COVID-19 had acute pulmonary hypertension.[2] There are also several case reports of pulmonary hypertension in post COVID-19 patients in the literature.[3,4]

On the other hand, an increased prevalence of pulmonary hypertension and right ventricular dysplasia were noted on transthoracic echocardiogram in COVID-19 recovered patients 2 months after discharge.[5] Thus, it is possible for our case to develop pulmonary arterial hypertension after recovery from COVID-19.

Meanwhile, autopsy findings in patients who died of COVID-19 showed pulmonary arterial wall thickening, a characteristic histological finding in pulmonary arterial hypertension. SARS-Cov-2 spike protein mediated cell signalling was proposed as the culprit for arterial hyperplasia.[6] While the exact mechanism of vascular injury by COVID-19 remains unknown, several mechanisms such as virus-cell interaction, silent hypoxemia, inflammation with cytokine storm, and uncontrolled complement activation have been proposed.[7] All of these mechanisms can contribute to pulmonary vascular remodelling leading to increased pulmonary vascular resistance and, therefore, pulmonary hypertension.

The outcome of pulmonary vascular hypertension in pregnancy remains poor, with a mortality rate up to 30% in idiopathic PAH, highest in the first month after delivery.[8] This is because after delivery, autotransfusion of the uteroplacental and reduced aortocaval compression further increase the stroke volume and cardiac output.[9] It will further increase pulmonary blood flow and lead to worsening of pulmonary hypertension and, thus, the mortality rate. In view of this, we have prepared nitric oxide and ECMO standby to prepare for the potential hemodynamic instability.

For prognosis, our patient belongs to class IV in WHO classification in pregnant women with congenital and acquired heart disease. Study has shown the mortality rate of class IV was 3.6%.[10] After delivery, the WHO function class (Table 1) is a helpful tool in guiding the prognosis of pulmonary hypertension patients. Fortunately, our patient's functional status improved with time to class I, which in turn, improved her prognosis as she has a low

risk profile.[11]

Table 1: World Health Organization functional class description of patients with pulmonary hypertension

I	No limitation of usual physical activity; ordinary physical activity does not cause dyspnea, fatigue, chest pain, or presyncope
II	Mild limitation of physical activity; no discomfort at rest; but normal activity causes increased dyspnea, fatigue, chest pain, or presyncope
III	Marked limitation of activity; no discomfort at rest but less than normal physical activity causes increased dyspnea, fatigue, chest pain, or presyncope
IV	Unable to perform physical activity at rest; may have signs of RV failure; symptoms increased by almost any physical activity

There are some limitations regarding this case report. Firstly, the sample size is small as this is a rare condition in Hong Kong during pregnancy. Secondly, there is limited high-quality evidence available. This could be attributed to insufficient awareness of pulmonary hypertension as potential complications of COVID-19 infection in which more studies are required to delineate the possible relationship.

In conclusion, there is increasing evidence that illustrates the possible relationship between COVID-19 infection and pulmonary arterial hypertension in pregnancy. With the ever-growing COVID-19 recovered population, we will encounter more of these patients in the future. Early identification and multidisciplinary management is needed for better outcomes for both the mother and baby due to potential lethal complications.

DATA AVAILABILITY

The data used to support this study are included within the article.

AUTHOR CONTRIBUTIONS

All authors contributed to the concept or design of the study, acquisition of the data, analysis, or interpretation of the data, drafting of the manuscript, and critical revision of the manuscript for important intellectual content. All authors had full access to the data, contributed to the study, approved the final version for publication, and took responsibility for its accuracy and integrity.

CONFLICT OF INTEREST

The authors declare that they have no conflict of interest.

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ETHICS APPROVAL

The patient was treated in accordance with the tenets of the Declaration of Helsinki. The patient provided written informed consent for all treatments and procedures.

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