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Case Report

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Pulmonary Arterial Hypertension in Covid 19 Patient : Therapeutic and Prognostic Impact

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Introduction

The coronavirus disease of 2019 (COVID-19) caused by the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) first appeared in Wuhan, China. It was officially declared a pandemic by the World Health Organization in March 2020. Patients with underlying chronic health conditions, mainly hypertension and cardiovascular disease, are at increased risk of developing more severe COVID-19 disease. Pulmonary hypertension (PH) is a pulmonary vascular disease characterized by pulmonary arterial remodeling and vasoconstriction leading to elevated pulmonary artery pressure and, ultimately, right heart failure [1]. So far, there have been only a few reported cases of COVID-19 disease in patients with PH. The authors of this article report a case of a Covid-19 infection and pulmonary arterial hypertension emphasizing its therapeutic and prognostic impact.

Case report

76-year-old male, with a history of high blood pressure treated by amlodipine 5 mg, he was diagnosed with a Horton for 9 months under treatment with prednisone, admitted in the ICU for SARS CoV2 pneumonia. The patient had a contact with an infected individual one week before. 5 days later, intense fatigue, dry cough, 39° fever. Following a positive PCR test conducted at a regional hospital, the patient was admitted into our facility after 4 days of treatment for acute respiratory distress. During his admission, the patient was conscious (GCS 15/15), polypneic (27 cycles/min), Oxygen saturation in the arterial blood (SaO2 80 %) in the ambient air and (SaO2 97%) in high concentration oxygen. Hemodynamically stable, cardiac frequency at 64bpm, blood pressure 130/80. temperature of 36.5°C. CT scans showed typical ground glass opacities associated with crazy paving, graded CO-RADS 5. CBC showed a lymphopenia of 540/mm, WBC 7220 el/mm3, Hb at 10.6 g/dL, Platelets of 177000 el/mm3. D-dimers were 1080 µg/L for a normal value (<280), troponins at 1.7 ng/L and BNP at 119 pg/ml. Ferritin at 2884 ng/L and CRP of 37mg/L for a normal value (<5.0), Interleukin 6 at 7.97 pg/ml for a normal value (<7.00). A pre-therapeutic ECG was normal, rhythm of 74 bpm, no repolarization trouble and QTc at 433mm. Echocardiography was realized and showed a moderete tricuspid regurgitation (Tricuspid valve morphology normal) with a pulmonary arterial systolic pressure at 47 mmhg, normal right ventricular systolic function (TAPSE 25 mm) RV/LV = 0.89. Non-dilated and non- enlarged left ventricle, normal left ventricular contraction. Arterial gasometry showed pH 7.45 PCO2 44 mmhg PO2 96 mmhg and paO2/ FiO2 188.

Therapeutic care was based on oxygen therapy and non-invasive ventilation, anti-coagulation, antihypertensive treatment, corticosteroid therapy and the association Hydroxychloroquin + Azythromycin. We did not have to use a specific medication for the pulmonary arterial hypertension in particular endothelin receptor antagonists (ERA), phosphodiesterase-5 (PDE5) inhibitors, inhaled nitric oxide (iNO) and prostacyclins. The patient had a favorable outcome under oxygen therapy and his pulmonary arterial pressure have decreased (30 mmhg).

Discussion

In late March 2020, experts from over 32 U.S. PH Centers responded to a Pulmonary Hypertension Association (PHA) query. Only 13 COVID-19 cases were reported, with one death, prompting us to ask, why have there been so few catastrophic COVID–PAH patient events? At the outset of the pandemic, PAH patients were warned to self-isolate, and that may be the simple answer [2]. Paradoxically, could PH-specific medication protect against some

cardiopulmonary manifestations of COVID-19? Might there be an altered pulmonary endothelial response due to lack of ability to mount a florid inflammatory response [3] ; Could vascular remodeling and altered lymphocyte subsets render the vasculature too "exhausted" to manifest endotheliitis and launch the cytokine release syndrome?
In our experience, hypoxia and systemic inflammatory response syndrome with PAH and COVID-19 infection is difficult to treat. Previous studies have shown the risk of acute respiratory distress syndrome (ARDS) in pulmonary vascular disease suggesting that COVID-19 pneumonia in the context of PAH will more commonly result in ARDS [4,5].

However, patients with PH can deteriorate rapidly with right ventricular (RV) decompensation and failure in the setting of hypoxemia. Intensive care unit (ICU) treatment of PH patients with COVID-19 includes treatment of the underlying viral respiratory infection, supportive measures including ventilator support (if required), meticulous fluid management to optimize RV preload, reduction of RV afterload with pulmonary vasodilators, and an individualized use of inotropes and vasopressors[1].

Conclusion

The COVID-19 pandemic presents many unique challenges when caring for patients with pulmonary hypertension. While the focus of this communication is on patients with PAH, the presence of PH, whether pre-existing or as a direct result of the lung injury that occurs with COVID-19 infection, is likely to be a major contributor to the morbidity and mortality associated with COVID-19 infection.

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