European Journal of Case Reports in Internal Medicine

Severe Pneumonia Caused by SARS-CoV-2: A Novel Cause of Platypnoea-Orthodeoxia Syndrome

José Abreu Fernandes[1], João Santos Faria[2], Andreia Fernandes[3], Ana Rita Ramalho[1], Adriana Girão[1], Maja Petrova[1]

[1] Serviço de Medicina Interna, Centro Hospitalar e Universitário de Coimbra EPE, Coimbra, Portugal

[2] Serviço de Medicina Física e Reabilitação, Centro Hospitalar e Universitário de Coimbra, Coimbra, Portugal

[3] Serviço de Endocrinologia, Instituto Português de Oncologia de Coimbra Francisco Gentil, Coimbra, Portuga

Doi: 10.12890/2022_003385 - European Journal of Case Reports in Internal Medicine - © EFIM 2022

Received: 09/05/2022

Accepted: 25/05/2022

Published: 19/08/2022

How to cite this article: Abreu Fernandes J, Santos Faria J, Fernandes A, Ramalho AR, Girão A, Petrova M. Severe pneumonia caused by SARS-CoV-2: A novel causa of platypnoea-orthodeoxia syndrome. *EJCRIM* 2022;**9**: doi:10.12890/2022_003385.

Conflicts of Interests: The authors declare there are no competing interests.

ABSTRACT

Platypnoea-orthodeoxia syndrome (POS) is a condition characterized by dyspnoea and hypoxaemia while sitting or standing, which improves during decubitus. It is usually caused by intracardiac right-to-left shunting through a patent foramen ovale but may also occur due to pulmonary ventilation-perfusion mismatch of other aetiologies. A new cause of POS was recently described: SARS-CoV-2 pneumonia. We report the case of a 62-year-old man admitted for SARS-CoV-2 pneumonia with respiratory failure. Chest computed tomography angiography showed pulmonary thromboembolism and parenchymal lung changes compatible with COVID-19. He had worsening dyspnoea in a sitting position, relieved by assuming the dorsal position. He was diagnosed with POS after other causes were excluded. POS is an underdiagnosed complication of COVID-19 and is manageable with respiratory rehabilitation.

LEARNING POINTS

- Platypnoea-orthodeoxia syndrome is an under-recognized condition presenting as a complication of a structural shunt.
- We describe SARS-CoV-2 pneumonia as a novel cause for this syndrome.
- It is a reversible syndrome provided there is early diagnosis and initiation of pulmonary rehabilitation.

KEYWORDS

Platypnoea-orthodeoxia syndrome, COVID-19, severe pneumonia, rehabilitation

INTRODUCTION

Platypnoea-orthodeoxia syndrome (POS) is a rare condition that is defined by a decline in peripheral oxygen saturation (SpO2) >5% and/or oxygen partial pressure >4 mmHg during orthostatism or sedestation, that improves during dorsal decubitus [1]. It is recognized as a complication of a structural or functional shunt, although its pathophysiology is unclear [1, 2]. Frequently, this shunt has an intracardiac origin, patent foramen ovale being the leading cause, followed by atrial septum defects and aneurysm of the interatrial septum.

Pulmonary conditions such as arteriovenous malformations, structural pulmonary diseases, and hepatopulmonary syndrome are less frequent aetiologies [1–3]. A novel cause of POS was described last year: severe pneumonia caused by SARS-CoV-2.

European Journal of Case Reports in Internal Medicine

CASE DESCRIPTION

We present the case of a 62-year-old man with a medical history of arterial hypertension and dyslipidaemia. The patient attended the emergency department complaining of dyspnoea and exercise intolerance on the 8th day of SARS-CoV-2 infection. Vital signs were normal and SpO2 was 92%. Chest teleradiography showed bilateral opacities. He was admitted for pneumonia caused by SARS-CoV-2 and partial respiratory insufficiency, and dexamethasone and oxygen therapy were initiated. While hospitalized, the patient's clinical condition deteriorated, and the fraction of inspired oxygen (FiO2) was progressively increased. Non-invasive mechanical ventilation (NIMV) was initiated and the patient was transferred to the intermediate care unit (ICU) on the 9th day. He remained on NIMV for 5 days and on high-flow nasal cannula therapy for 7 days. Due to persistent polypnoea, he underwent chest computed tomography with angiography that showed pulmonary thromboembolism (PTE) and pulmonary ground-glass opacities with reticulation. A transthoracic echocardiogram excluded right ventricle dysfunction.

On the 24th day of hospitalization, declining SpO2 was noted during sedestation. A transoesophageal echocardiogram with an agitated serum test was performed to check for right–left shunt with a pressure gradient, which was excluded.

Due to a favourable clinical evolution, the patient was transferred to the Internal Medicine ward. During his stay there, polypnoea with diminishing SpO2 while sitting was recorded, with a 14% decline in arterial oxygen saturation. This was assumed to be due to post-COVID-19 POS, and Physical Medicine and Rehabilitation collaboration was requested.

The patient began verticalization training with oxygen therapy support, then progressed to ambulation in the medical ward. He also started a respiratory rehabilitation program including respiratory therapy and aerobic training.

After a few days of rehabilitation, the patient started assisted ambulation. On the 39th day of hospitalization, he was discharged as he was capable of autonomous locomotion without the need for supplemental oxygen.

Three months later, the patient had returned to his usual daily routines, and was continuing his ambulatory rehabilitation.

DISCUSSION

POS is an under-recognized complication of severe SARS-CoV-2 pneumonia. In a retrospective study, Tan et al. studied all cases of COVID-19 with acute respiratory distress syndrome (ARDS) admitted to an Intensive Care Unit [4] and hypothesized that the mechanisms underlying POS associated with ARDS caused by COVID-19 were complex and multifactorial [4]. The primary mechanism appears to be exacerbation of the intrapulmonary physiological shunt during orthostatism as blood flow is redistributed from the apex to the pulmonary bases [4]. As SARS-CoV-2 infection usually affects the lower lobes with fibrotic changes, the blood delivered to this region is not adequately oxygenated, with a significant change in the ventilation/perfusion ratio [4]. Additionally, SARS-CoV-2 has a particular tropism for the endothelium with consequent endothelial activation, causing microthrombosis and microangiopathy phenomena, while decreasing perfusion and increasing alveolar dead space [4]. The respiratory weakness secondary to intensive care-related myopathy makes a considerable contribution to alveolar hypoventilation [4].

Our patient presented fibrotic alterations in his lungs, particularly in the lower lobes, secondary to COVID-19, supporting the above-mentioned hypothesis. It is unclear why POS does not occur in all cases of interstitial lung disease, so vascular changes in thromboembolic phenomena are probably an important, if not an essential cofactor for developing hypoxaemia in orthostatism [5]. It is crucial to exclude other causes of worsening hypoxaemia such as bacterial superinfection, pneumomediastinum, pneumothorax, or PTE [6]. Clinical manifestations such as hypoxaemia and dyspnoea in orthostatism associated with evidence of desaturation with positional changes allow the diagnosis of POS. Patent foramen ovale is the most frequent cause [1–3], so its exclusion is essential, namely through an echocardiogram with agitated serum.

POS is often recognized during motor rehabilitation, with positional changes evidencing the underlying intrapulmonary shunt [4]. Early rehabilitation in dorsal decubitus is essential to maintain muscle and joint activity while the patient readapts to orthostatism [7]. Although there are no established protocols yet for this specific pathology, these patients benefit from verticalization training with oxygen therapy and a respiratory rehabilitation program which includes learning breathing techniques, resistance training, and physical reconditioning. Initial exercises should be of low to moderate intensity in order not to exacerbate the areas of ventilation-perfusion mismatch [8].

Despite causing apprehension in patients and in their health professionals, it is a reversible condition, with symptomatic resolution expected in 6–39 days [4].

In conclusion, POS is an underdiagnosed complication of severe pneumonia due to SARS-CoV-2. It is crucial to raise awareness of its existence and possible reversibility to allow for the early initiation of rehabilitation.

European Journal
of Case Reports in
Internal Medicine

REFERENCES

1. Agrawal A, Palkar A, Talwar A. The multiple dimensions of platypnea-orthodeoxia syndrome: a review. *Respir Med* 2017;**129**:31–38. doi: 10.1016/j.rmed.2017.05.016.
2. De Vecchis R, Baldi C, Ariano C, Giasi A, Cioppa C. Platypnea-orthodeoxia syndrome: orthostatic dyspnea and possible pathophysiological substrates. *Herz* 2017;**42**(4):384–389. doi: 10.1007/s00059-016-4479-4.
3. De Vecchis R, Baldi C, Ariano C. Platypnea-orthodeoxia syndrome: multiple pathophysiological interpretations of a clinical picture primarily consisting of orthostatic dyspnea. *J Clin Med* 2016;**5**(10):85. doi: 10.3390/jcm5100085.
4. Tan GP, Ho S, Fan BE, Chotirmall SH, Tan CH, Lew SJW, et al. Reversible platypnea-orthodeoxia in COVID-19 acute respiratory distress syndrome survivors. *Respir Physiol Neurobiol* 2020;**282**:103515. doi: 10.1016/j.resp.2020.103515.
5. Longo C, Ruffini L, Zanoni N, Longo F, Accogli R, Graziani T, et al. Platypnea-orthodeoxia after fibrotic evolution of SARS-CoV-2 interstitial pneumonia. A case report. *Acta Biomed* 2020; **91**(3):ahead of print. doi: 10.23750/abm.v91i3.10386.
6. Souza T, Dantas F, Loureiro C, Serra J, Marinho J. Ampliando o diagnóstico diferencial: infecção pelo SARS-CoV-2 associada a pneumomediastino e síndrome platipneia-ortodeoxia. *Revista Científica Hospital Santa Izabel* 2021;**4**:159–163. doi: 10.35753/rchsi.v4i3-4.185.
7. Tham SL, Ong PL, Lee AJY, Tay MRJ. Rehabilitation of patients with platypnea-orthodeoxia syndrome in COVID-19 pneumonia: two case reports. *J Rehabil Med Clin Commun* 2020;3:1000044. doi: 10.2340/20030711-1000044.
8. Tedjasaputra V, Sá RC, Anderson KM, Prisk GK, Hopkins SR. Heavy upright exercise increases ventilation-perfusion mismatch in the basal lung: indirect evidence for interstitial pulmonary edema. *J Appl Physiol* (1985) 2019;**127**(2):473–481. doi: 10.1152/japplphysiol.00056.2019.