Case Report

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Platypnea-Orthodeoxia Syndrome Associated with COVID-19 Pneumonia and Prolonged Treatment Due to Tamsulosin Use: A Case Report

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Abstract

Platypnea-orthodeoxia syndrome (POS) is an extremely rare condition. There are no other cases in the literature where tamsulosin has been reported to prolong POS treatment. A 67-year-old male patient was hospitalized due to COVID-19. He was followed up in the ward after being in the intensive care unit for ten days. There was a significant decrease in saturation (SaO2) when sitting and standing compared to lying. He was diagnosed with POS. However, there was no significant improvement in POS with exercises. After stopping the tamsulosin he was using, there was a dramatic improvement. He was discharged on the 72nd day of his hospitalization. Due to COVID-19, POS is more likely to appear than before. It is a condition that clinicians should recognize. The pathophysiology of POS has not been fully elucidated. The case we present suggests that alpha-blockers may also be related to pathophysiology.

Keywords: COVID-19; Platypnoea-orthodeoxia syndrome; SARS-CoV-2.

Introduction

Platypnea-orthodeoxia syndrome (POS) is a clinical entity characterized by positional dyspnea (platypnea) and arterial desaturation (orthodeoxia) that occurs when sitting or standing up and usually resolves when lying down. The decrease in oxygen saturation is considered significant when the PaO2 falls more than 4 mmHg, or the SaO2 falls more than 5% from the supine position to the upright position (1). POS is a rare finding, and its true prevalence is unknown (1).

It was first described in 1949 and was first called "orthostatic cyanosis" (2). The terms "platypnea" and "orthopnea" were used to describe shortness of breath and arterial desaturation that worsened in the upright position and improved in the supine position in 1969 and 1976, respectively (3,4).

We present a case of POS that developed due to COVID-19 and whose treatment we think has been prolonged due to tamsulosin use.

Case Report

A 67-year-old man was admitted to hospital with cough, fatigue and low oxygen saturations. He tested positive for SARS-CoV-2. SaO2 at 80% with FiO2 60%. He was taken to the intensive care unit due to the need for high-flow oxygen. He had previous diagnoses of chronic kidney disease (never on dialysis) and benign prostatic hypertrophy. The drugs he uses constantly are tamsulosin and allopurinol 150 mg. Tamsulosin was discontinued because a urinary catheter was inserted, and allopurinol was continued. Pulse 500 mg/day methylprednisolone was given for three days in the intensive care unit, 250 mg/day for three days, 120 mg/day for three days, and 120 mg/day when he came to the ward. Empirical antibiotic therapy (ceftriaxone and clarithromycin) and antithrombotic prophylaxis with enoxaparin (4,000 IU/day) were administered. He was not intubated while in the intensive care unit. He did not need a non-invasive mechanical ventilator and was followed up with high-flow oxygen. He was taken to the ward after ten days of intensive care hospitalization. SaO2 was 94% in the supine position with nasal O2. Sitting SaO2 was 85%, standing SaO2 was 78%. Breathing exercise was taught by physical therapy with the Triflow ball exercise. He practiced exercises harmoniously and regularly. The urinary catheter was removed, and tamsulosin was started once a day due to difficulty and pain in urination. With nasal oxygen in the supine position, with 4 lt/min, his saturation was 94%, at a 45-degree angle, his shortness of breath started, and although oxygen was provided in the

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sitting position, his saturation decreased to 65% within one minute, and he had severe shortness of breath, hypotensive complaints, and blood pressure was measured 70/40 mm/Hg. When the patient was supine, SaO2 rose above 92%, and his blood pressure rose to 100/70 mm/Hg within two to three minutes. Echocardiography was performed to look for the etiology of a cardiac shunt, atrial myxoma, atrial thrombus with stalk or platypnea, and orthostatic hypotension as he had significant platypnea. The ejection fraction was 60%, and heart cavities were normal, and there was no finding to explain platypnea. Contrast-enhanced thorax computed tomography angiography was not performed due to renal failure. On the 52nd day of the patient's hospitalization, non-contrast thorax computed tomography was performed, and fibrotic areas were observed compared with the first tomography (Figure 1). The patient was clinically evaluated as POS. The angle of the daily bed was increased to make it steeper, and short-term sitting exercises were done. There was no significant improvement after exercises such as intermittent sitting and straightening the bed angle applied for six weeks. Noradrenaline infusion

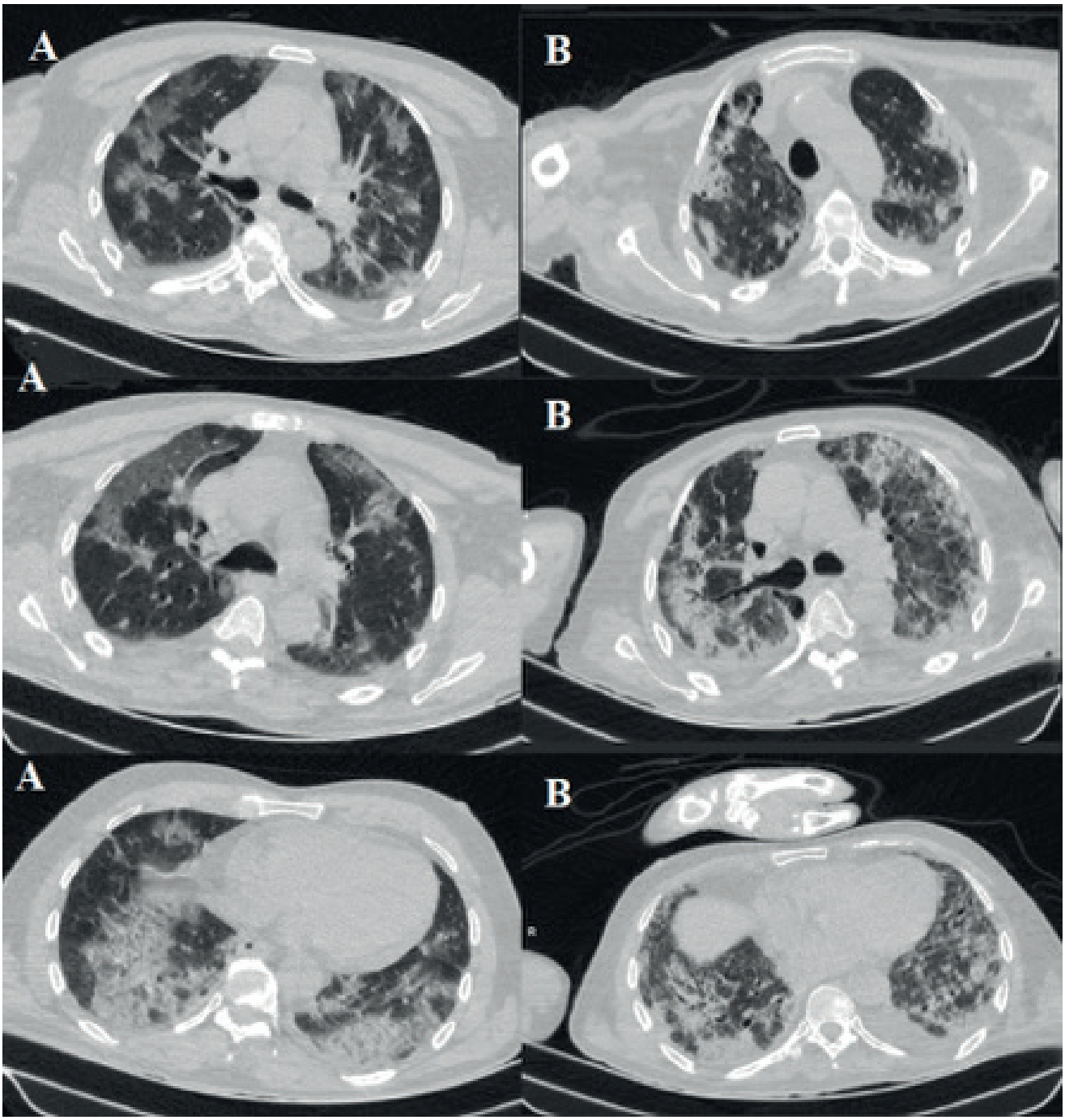

Figure 1. A: Thorax CT taken on the first day of hospitalization, diffuse ground-glass opacities, B: Thorax CT taken on the 52nd day of hospitalization, fibrotic tissues.

at a 0.11 microgram/kg/minute dose was given twice as 12 hours/day. While receiving the infusion, the patient's SaO2 did not decrease below 80% in the sitting position (it decreased to 65% when noradrenaline was not infused), and the patient did not feel shortness of breath clinically as before. When he stood up, he showed hypotensive symptoms for about 30 seconds, and then his blood pressure was around 100/70 mm/Hg. However, even though he did not take a step while standing, his SaO2 fell below 70%. Tamsulosin was discontinued and a urinary catheter was inserted. Approximately 24 hours after the last dose of tamsulosin, the patient was dramatically better. His saturation in sitting position was 93%, and he could sit for minutes. He still had hypotensive symptoms for the first 20-30 seconds when he stood up, but his saturation did not fall below 88%. Walks with 2-3 meters intervals were carried out with support. SaO2 did not fall below 89% in room air without oxygen support in the sitting position. Nasal oxygen support was needed while standing and exerting. He had difficulty walking due to the decrease in muscle strength since he had been lying down for a long time. We discharged the patient with an oxygen concentrator on the 77th day of hospitalization, six days after discontinuing tamsulosin. Information was received by phone three days after discharge, and he was walking 3-4 meters without support. On the 15th day of discharge, he could walk at home without support.

Discussion

A case of POS with orthostatic hypotension, which developed due to COVID-19 and whose treatment we think was prolonged using tamsulosin, was shared. POS is a rare condition, and its true prevalence is unknown.[1] We think that its possible prevalence is higher than known. According to our research in the literature, there are 366 publications in Pubmed when "platypnea orthodeoxia syndrome" is written, and 16 as of 18.12.2021 when "covid and platypnea orthodeoxia syndrome" is written. The feature that distinguishes our case is the prolongation of the treatment due to tamsulosin. We also did not find a case with a previously reported association between tamsulosin and POS. The POS, which did not improve for about two months, showed dramatically positive results about one day after the discontinued tamsulosin. Also, when our case was in a sitting or standing position, SaO2 decreased from 92% to 65%, much more than the cases in the literatüre (5-8).

In the retrospective analysis, we found that the increase in this decrease was more significant after using tamsulosin. It has been reported that the possible pathophysiology of POS may be dilatation and the development of physiological shunt in alveolar capillaries, as in hepatopulmonary syndrome (9). We think that tamsulosin, an alpha-blocker, may have triggered dilatation of alveolar capillaries and delayed treatment. There may also be a different pathophysiological relationship between POS and tamsulosin. It has been stated that physical therapy, albumin, norepinephrine, and indomethacin may benefit treatment. We received a clinically effective response to norepinephrine, and we did not start indomethacin because he had CRF. However, we saw more effective improvement after discontinuing tamsulosin. The pathophysiology of POS is not known exactly (10).

Conclusion

We think that our report of a case worsening with tamsulosin may inspire future research on the relationship between alpha receptors and POS to elucidate the pathophysiology of POS.

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