

# A rapid decrease in pulmonary arterial pressure by noninvasive positive pressure ventilation in a patient with chronic obstructive pulmonary disease

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## Abstract

The natural history of chronic obstructive pulmonary disease (COPD) is characterized by progressive decrements in expiratory airflow, increments in end-expired pulmonary volume, hypoxaemia, hypercapnia and the progression of pulmonary arterial hypertension (PAH). Noninvasive positive pressure ventilation (NPPV) treatment is increasingly used for the treatment of acute and chronic respiratory failure in patients with COPD. NPPV can increase PaO<sub>2</sub> and decrease PaCO<sub>2</sub> by correcting the gas exchange in such patients. The acute effect of NPPV on decreasing PAP is seen in patients with respiratory failure, probably due to the effect on cardiac output. Here, a case with COPD whose respiratory acidosis and PAH rapidly improved by NPPV was presented and therefore we suggested to perform an echocardiographic assessment to reveal an improvement of PAH as well as respiratory acidosis, hypercapnia and hypoxemia with that treatment.

**Key words:** Noninvasive positive pressure ventilation, chronic obstructive pulmonary disease, respiratory failure, pulmonary arterial pressure, echocardiography

## Introduction

Pulmonary arterial hypertension (PAH) is the primary cardiovascular complication encountered in chronic obstructive pulmonary disease (COPD). The natural history of COPD is characterized by progressive decrements in expiratory airflow, increments in end-expired pulmonary volume, hypoxemia, hypercapnia and the progression of PAH.<sup>[1,2]</sup> In patients with COPD, unfavorable prognostic factors include airway obstruction, ventilatory capacity, hypercapnia and PAH.<sup>[3]</sup> In particular, PAH in COPD leads to the development of right ventricular hypertrophy, dilation and failure, the so-

called cor pulmonale, which indicates an advanced stage of respiratory disease.<sup>[1,2]</sup> Furthermore, PAH can be a predictive factor for hospitalization for acute exacerbations in COPD patients.<sup>[4]</sup> Doppler echocardiography which is an inexpensive, easy and reproducible method, is commonly used to estimate mean and systolic pulmonary arterial pressure (PAP) and to diagnose PAH in patients with COPD.

The goals of therapy consist of attenuation of PAH, enhancement of right ventricular function, alleviation of clinical symptoms and improvement in survival. The agents that have been most extensively evaluated for these purposes include oxygen, vasodilators, theophylline and inotropic medications. Currently, noninvasive positive pressure ventilation (NPPV) treatment is increasingly used for the treatment of acute and chronic respiratory failure in patients with COPD.<sup>[5]</sup>

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Here, a case with COPD whose respiratory acidosis and PAH rapidly improved by NPPV was presented.

### Case Report

A 69-year-old man admitted with severe dyspnea, cough, expectoration of purulent sputum, cyanosis and tendency to sleep. He had a diagnosis of COPD and using NPPV and long-term oxygen therapy together with his medical treatment since the last three years. He had a history of 40 packs-year smoking and was an ex-smoker for 20 years. His medical history revealed systemic hypertension and usage of antihypertensive medication for 10 years.

Initial physical examination revealed that, he was semi-conscious, obese with barrel chest, dyspneic and using accessory respiratory muscles with supraclavicular, suprasternal and intercostal retractions heaves and had flapping tremor, chemosis in his conjunctivas and cyanosis in his nails and mucosa. His physical examination was as follows: body temperature 36.8°C, arterial blood pressure 154/72 mmHg, heart rate 96 beats/min, respiratory rate 40 breaths/min. Pulmonary auscultation revealed silent chest in both hemithoraces and inspiratory crackles in both basal areas. There were no heart murmurs, no peripheral edema, no jugular vein distention. The liver and spleen were not palpable. The abdomen was soft and flat without tenderness or rigidity. His hemogram, blood chemistries and hemostasis tests were within normal limits. Arterial blood gas (ABG) analysis in room air was as the following pH:7.23, PCO<sub>2</sub>:79 mmHg, PO<sub>2</sub>:39 mmHg, O<sub>2</sub>sat%:73 % and HCO<sub>3</sub>:34 mEq/L [Table 1]. Chest radiography revealed bilateral hyperlucency, increased bronchovascular images of the lung fields and enlargement of right descending

pulmonary artery. Pulmonary function tests revealed very severe bronchoconstriction; FVC: 560 ml(14%), FEV1: 450 ml (15%), FEV1/FVC: 80%, PEF: 990 ml (13%), FEF 25-75: 420 ml (14%). His ECG revealed sinus rhythm with incomplete right bundle brunch block and left anterior hemiblock. His echocardiography revealed very severe pulmonary hypertension with 60 mmHg mean and 75 mmHg systolic pulmonary arterial pressures by Doppler. He had right atrial (40 mm) and right ventricular (44 mm) enlargement and right ventricular hypertrophy with 10 mm of right ventricular free wall diameter. However, right ventricular ejection fraction of the patient was within normal limits (65%).

Bronchodilator therapy, systemic corticosteroid, intravenous antibiotic treatment and NPPV were initiated with a fraction of inspired oxygen of 2 L, a positive end-expiratory pressure of 4 cm H<sub>2</sub>O and a pressure-support level of 8 cm H<sub>2</sub>O with bilevel positive airway pressure (BIPAP) (Respironics Inc; Murrysville, PA). So, BIPAP treatment with nasal and oro-nasal masks has been continued with the increased pressure supports to 18/5 cmH<sub>2</sub>O. Afterwards, his mental status improved and tendency to sleep gradually diminished, his ABG analysis and mean and systolic PAPs were dramatically improved at the 11<sup>th</sup> day of NPPV treatment [Table 1].

### Discussion

The prevalence of PAH increases as COPD worsens and the development of PAH and cor pulmonale appears to affect survival of patients with COPD. Potential causes proposed to explain the development of PAH in COPD include gas exchange abnormalities, destruction of the pulmonary vascular bed, alterations in respiratory mechanics, changes in intrinsic pulmonary vessel tone and increased blood viscosity. In acute attacks of COPD, NPPV allows time for other conventional therapy to work, thus reversing the progression of respiratory failure and reducing morbidity and mortality and also improvements occur in minute ventilation, respiratory rate and transdiaphragmatic activity.<sup>[4]</sup>

**Table 1: Arterial blood gases and pulmonary arterial pressure by Doppler echocardiography at baseline and at the end of noninvasive positive pressure ventilation treatment in the patient with chronic obstructive pulmonary disease**

	Treatment day	PAP (mmHg)		Arterial blood gases				
		Mean	Systolic	pH	PCO <sub>2</sub> (mmHg)	PO <sub>2</sub> (mmHg)	HCO <sub>3</sub> <sup>-</sup> (mEq/L)	SatO (%)
At baseline	0	60	75	7,23	79,0	39,0	34,0	73,0
At discharge	11	25	40	7,44	40,7	71,0	27,9	94,7

PAP - Pulmonary arterial pressure

NPPV can improve pH relatively rapidly, at one hour after instituting ventilation.<sup>[7]</sup>

Doppler echocardiography is a well-tolerated, efficient evaluation method that can be used at NPPV initiation in acute COPD patients. It is recommended as a useful tool for early detection of hemodynamic alterations due to NPPV application in patients with acute ventilatory failure.<sup>[8]</sup> In our case, BIPAP treatment with nasal mask improved patient's mental status, respiratory acidosis, hypercapnia and hypoxemia. Our patient had compensated cor pulmonale without heart failure but with right atrial and right ventricular enlargement, right ventricular hypertrophy and normal right ventricular ejection fraction. So, an acute decompensation could occur in such a patient. It is very important to decrease PAP in patients with COPD in order to prevent progression to heart failure, the so-called decompensated cor pulmonale. Hence, NPPV is a successful treatment modality to decrease PAP more rapidly compared with medical therapy. In our patient, mean and systolic PAPs were dramatically improved at the 11<sup>th</sup> day of NPPV treatment as well. An echocardiographic assessment of right heart structure and functions is a simple, noninvasive and reproducible method in patients with COPD. The subcostal approach successfully detected adequate Doppler signals in a large proportion of COPD patients.<sup>[9]</sup> Although ultrasound examination of the heart is technically difficult in patients with hyperinflation of the lungs, it could be obtained suitable images in up to 80-97% of patients.<sup>[10]</sup>

As a result, an echocardiographic assessment should be performed to the patients having NPPV treatment in COPD acute attacks, in order to reveal an improvement of PAH as well as respiratory acidosis, hypercapnia and hypoxemia with that treatment. Long-term results of NPPV on PAP are not available and further research in patients with COPD is needed.

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