CASE REPORT

Severe respiratory insufficiency in a patient with chronic obstructive pulmonary disease following carbogen inhalation

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Summary
The case history is presented of a patient with chronic obstructive pulmonary disease in whom severe respiratory compromise developed following carbogen inhalation. Clinicians should be aware of this potentially lethal adverse effect of carbogen in this particular group of patients.

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Introduction

Sudden sensorineural unilateral hearing loss has no known specific etiologic factor. It has been proposed that inhaled carbogen, a mixture of 95 percent oxygen and 5 percent carbon dioxide, will improve oxygenation of inner ear perilymph and thus improve the likelihood of recovery.1

Case report

A 66-year-old man with a 35-pack-year smoking history, presented with sudden right ear hearing loss. The patient had chronic obstructive pulmonary disease (COPD) for 25 years. Spirometry performed several months earlier showed a FVC ratio (FEV1/FVC) and FEV1 of 39% and 52% of predicted, respectively. Upon admission the patients was in no respiratory distress and denied any respiratory symptoms and arterial blood gas levels on room air were the following: pH, 7.45; PCO2, 45 mm Hg; and PO2, 82 mm Hg.

The patient was given carbogen inhalation for 30 min, six times per day. The following day the patient complained of severe shortness of breath. On examination he appeared somnolent with severe respiratory distress, his respiratory rate was 30 breaths/min and his oxygen saturation was 80% on ambient air. His chest-X-ray was normal. Arterial blood gas levels on room air were the following: pH, 7.22; PCO2, 45 mm Hg; and PO2, 82 mm Hg. Carbogen inhalation was immediately stopped and the patient was treated by non-invasive-positive-pressure-ventilation bi-level device improves within 3-h and required no oro-tracheal intubation and invasive mechanical ventilation.

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The patient was weaned from non-invasive ventilatory support within 24 h and was discharged in good clinical condition.

Discussion

Elevation of end-tidal carbon dioxide concentration (up to 6.9%) and increased arterial oxygen tension have been reported during carbogen breathing, although no clinical respiratory compromise has been reported to date in patients with no prior pulmonary disorders.\(^2\)

Carbogen inhalation creates a hyperoxic—hypercapnic condition that is poorly tolerated by COPD patients for at least two reasons. First, supplemental oxygen given to COPD patients with CO\(_2\) retention may result in worsening respiratory acidosis, an effect that is usually attributed to the loss of their hypoxic stimulus to breathe.\(^3\)

Second, the degree of dyspnea in patients with severe COPD during carbogen inhalation correlates with their response to increased central drive CO\(_2\) stimuli.\(^4,5\)

Hence carbogen imposes both an increased hypercapnic motoneural output to the respiratory system with the subjective feeling of shortness of breath and hyperoxic suppression of ventilatory drive that further worsens CO\(_2\) retention thus creating a viscous cycle.

Carbogen inhalation should be used very cautiously in COPD patients if at all and monitoring of end-tidal PCO\(_2\) should carefully monitor with serial arterial blood gas measurements, or its administration.

Conflict of interest statement

The authors have no conflict of interest.

References