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Case Report

Acute pulmonary edema secondary to hyperbaric oxygen therapy

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Hyperbaric oxygen therapy (HBOT) has been shown to be effective in the treatment of diabetic ulcers, air embolism, carbon monoxide poisoning and gas gangrene with minimal adverse effects. Very few cases of HBOT causing acute pulmonary edema (PE) has been described; with a study on dogs suggesting that a complication of this therapy could be PE. We describe the case of an 80-year-old man with a history of stable systolic heart failure and diabetes mellitus presenting with acute PE following treatment with HBOT for diabetic foot.

INTRODUCTION

Hyperbaric oxygen therapy (HBOT) has been shown to be effective in the treatment of diabetic ulcers, air embolism, carbon monoxide poisoning, and gas gangrene [1]. It is usually well tolerated with very few side effects. A study on dogs suggested that a rare complication of HBOT is pulmonary edema (PE) with an estimated incidence of 1 per 1000 [2]. In divers who received recompression treatment for dysbarism, the incidence of pulmonary toxicity was 5 per 100 recompressions [3]. We describe a case of acute PE precipitated by HBOT.

CASE REPORT

An 80-year-old male with ischemic cardiomyopathy (ejection fraction 25%), noninsulin-dependent diabetes mellitus (NIDDM) and peripheral vascular disease (PVD) was admitted because of sudden onset of dyspnea. He was getting hyperbaric oxygen treatment for a non-healing plantar ulcer at our outpatient clinic. He was not on antibiotic therapy at the time as wound was not infected. His pre- HBOT heart rate (HR) was 80 beats/min, respiratory rate (RR) was 15 breaths/min and blood pressure (BP) was 134/80 mmHg. After 60 min of breathing 100% oxygen at 2.4 atms, he developed rapidly worsening dyspnea. He was brought to the hospital on 100% oxygen via non-rebreather mask. Physical examination revealed HR 110 beats/min, RR 30 breaths/min, BP 138/74 mmHg, and was significant for diffuse inspiratory and expiratory crackles. Owing to worsening respiratory distress and decline in cognition, he required emergent intubation and mechanical ventilation. Arterial blood gas values were as follows: pH 7.27; $PaCO_2$ 58 mmHg; PaO_2 117 mmHg; and arterial oxygen saturation was 0.85. EKG did not show any ischemic changes. Cardiac biomarkers were negative, but his BNP was significantly elevated at 1568 pg/ml. There were pink frothy secretions in the endotracheal tube and chest radiography showed bilateral alveolar and perivascular infiltrates (Fig. 1). Diagnosis of acute respiratory failure secondary to PE was made, and he was admitted to the cardiac ICU. He received intravenous diuretics, and was successfully extubated 3 days later (Fig. 2).

DISCUSSION

HBOT has been shown to improve the rate of healing of diabetic foot ulcers. Suggested mechanisms include improved wound tissue hypoxia, enhanced perfusion and down-regulation of inflammatory cytokines [4]. Some side effects of HBOT that are described include otic barotrauma, visual changes and possible CNS oxygen toxicity. Very few cases of PE due to HBOT have been described. Weaver *et al*. [5] described three cases in 2001—all of them had pre-existing cardiac disease, and two of them were diabetic. An increased risk of PE in persons with low cardiac ejection fractions has been reported [6]; however, details of this study are not available.

Proposed mechanisms for this include HBO-induced hyperoxia leading to increased peripheral vasoconstriction and

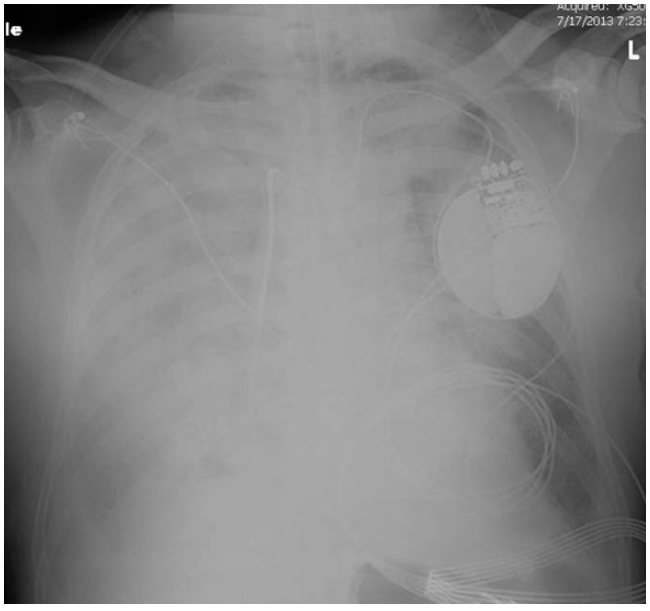

Figure 1: Chest X-ray after emergent intubation on presentation.

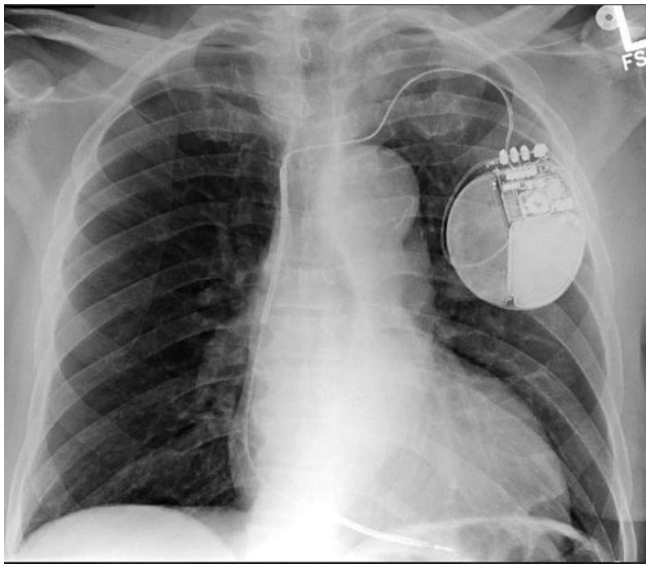

Figure 2: Chest X-ray on Day 3 after extubation.

thus cardiac afterload, increased oxidative myocardial stress, decreased LV compliance by oxygen radical-mediated reduction in nitric oxide, right and left ventricular imbalance and increased pulmonary capillary permeability [2]. HBOT has also been shown to increase N-terminal pro-B-type natriuretic peptide (NT pro-BNP) levels in diabetics by a mean of 100 pg/ml and causing considerable ventricular wall stress [7]. Any of these can precipitate acute PE in a patient with pre-existing heart disease or a diabetic, but further research is necessary.

Treatment remains primarily supportive with diuretics, supplemental oxygen and occasionally ventilatory support.

Our patient had several comorbidities including ischemic cardiomyopathy, NIDDM and PVD, but he was functional and had no overt manifestation of heart failure prior to HBOT. However, his overall hypoxic predisposition might have served as a risk factor for acute PE.

CONCLUSION

Acute PE seems to be a rare but serious side effect of HBOT in patients with pre-existing heart disease. Thus caution should be observed in treating patients with prior heart disease and low LVEF with hyperbaric oxygen.

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