Case Report

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A Case of Iatrogenic Vascular Air Embolism Treated with Bipap and Nitroglycerin

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Abstract

Vascular air embolisms are very rare but fatal complication of diving and many procedures, mostly central line catheterizations. Treatment is mostly supportive with 100% oxygen and in severe cases, hyperbaric therapy. Recent therapy has shown that nitric oxide can decrease the rate of bubble formation in animal studies after compression. We describe a case of iatrogenic air embolism treated with Bi-level non-invasive positive pressure ventilation (BiPAP) and IV nitroglycerin. This case illustrated the importance of recognizing this rare but potentially fatal syndrome, and the need for further research on the utility of nitroglycerin in the treatment of this syndrome.

Keywords: Air embolism; Iatrogenic; BiPAP; Nitroglycerin; Photo-pheresis

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Introduction

Vascular air embolism is a potentially fatal event. It has been described in divers, child birth and multiple surgeries and procedures; however it is most commonly an iatrogenic complication of central line placement. Treatment is rapid recognition, intra-venous (IV) fluids, 100% oxygen and in severe cases hyperbaric oxygen therapy [1]. We describe a case of iatrogenic air embolism during photo-pheresis, treated with Bi-level non-invasive positive pressure ventilation (BiPAP) and IV nitroglycerin.

Case

A 42 year old male with a history of bilateral lung transplant due to cystic fibrosis, with resulting bronchiolitis obliterans obstructing pneumonia (BOOP), on home oxygen of 3 L and on twice weekly photo-pheresis, presents from the dermatology photo-pheresis clinic to our emergency department (ED) with intra-hospital critical care transport. History by transport personnel report that the patient was in his usual state of health upon arrival to the clinic, however soon after his port was accessed, he began to complain of flushing sensation, dyspnea, palpitations, right sided numbness and became very agitated. He subsequently developed slurred speech, and respiratory distress, and was then transported to our ED.

On arrival to the ED, his vital signs were temperature 37.5°C, respiratory rate 24/min, heart rate 96 bpm, blood pressure 188/75 and SpO_2% of 87% on room air. The patient was awake but in severe distress and grunting. He was disoriented to time, person and place. Glasgow coma scale was 10 and he was showing signs of right hemiparesis. He was maintaining his airway and had no signs of oropharyngeal swelling. His right chest port was in place with no overlying erythema and his lung sounds were remarkable for mild inspiratory crackles in the bases. His abdomen was soft and lower extremities were not edematous. He had no rash and was warm to touch.

His repeat vital signs few minutes later, were temperature 37.2°C, respiratory rate 30/min, heart rate 110 bpm, blood pressure 206/103 and SpO_2 of 92% on 15 L/min non-rebreather 100% FiO_2. He was quickly given 0.4 mcg of sublingual nitroglycerin, placed on BiPAP (IPAP 10, EPAP 5, 100% FiO_2) and started on a nitroglycerin infusion at 80 mcg/min, after the establishment of two 18G IV catheters.

Complete blood count showed WBC of 20.50 k/cu mm, comprehensive metabolic panel normal and arterial blood gas few minutes after initiation of BiPAP was pH of 7.00, pCO_2 of 127mm Hg, pO_2 of 151 mm Hg and HCO_3 of 30 mmol/L with SpO_2 of 100%. EKG showed sinus tachycardia with no ischemic changes and chest X-ray was unremarkable, showing Mediport tip at junction of superior vena cava and right atrium with no effusions, consolidations or marked vascular congestion.

The patient's mental status and vital signs began to improve, and after 2 h in the ED he was at baseline. He was completely oriented with full return of neurological function. He was weaned

down to his baseline 3 L/min of oxygen by nasal cannula and nitroglycerin infusion was stopped. He was admitted to the medical intensive care unit (MICU) for further monitoring. Vital signs prior to admission were heart rate 118 bpm, respiratory rate 22/min, blood pressure 142/94 and SpO_2 98% on 3 L/min oxygen. Venous blood gas was pH 7.27, pCO_2 67 mm Hg, pO_2 58 mm Hg and HCO_3 30 mmol/L. CT head without intravenous contrast prior to transport to the MICU was unremarkable for any acute intracranial abnormality and CT of the chest without intravenous contrast was remarkable only for chronic changes of BOOP. He was observed for 24 h in the MICU and was subsequently discharged with no neurological sequelae and no new oxygen demand, at baseline clinical status. Venous blood gas on discharge from the hospital was pH 7.38, pCO_2 51 mm Hg, pO_2 89 mm Hg and HCO_3 30 mmol/L.

Discussion

Prior to his transfer to the MICU, the patient was able to recollect clearly the events in clinic. The patient's subcutaneous port is a Vortex port, which is larger than a typical chemotherapy port, in order to facilitate high flow rates during photo-pheresis. Accessing the port is done with a 16 gauge non-coring Angiodynamic needle that resembles a traditional peripheral IV catheter. The patient reports that after the clinic nurse accessed his port and subsequently withdrew the inner core needle, the catheter was left open to air for approximately ten seconds (longer than usual) and he heard a "whooshing" noise. This was followed by an extreme sense of chest tightness and panic with right facial and upper extremity numbness, and subsequently deterioration of his clinical status prompting his quick transfer to the ED.

The acute clinical deterioration of hemodynamic and mental status, with a clear history of open central access to air, along with rapid return to baseline with supportive therapy, argues strongly for a diagnosis of vascular air embolism.

Vascular air embolism is a rare but potentially life threatening event. It has been described in numerous scenarios, including blunt and penetrating trauma, diving and child birth and most frequently iatrogenically from interventional procedures, such as central line catheterization [1, 2]. The effects of a venous air embolism resemble those of a pulmonary embolus, where lodging in the pulmonary vasculature can cause acute hypoxemia and hypercapnia, eventually leading to increased right ventricular pressures and strain and systemic cardiovascular collapse from heart failure if the embolism is massive enough. The severity of the symptoms depends on the amount of air instilled and proximity to the heart, with a lethal dose described approximately to be 3-5 ml/kg [1, 2]. If there is a patent foramen ovale (PFO) or when extra alveolar air from barotrauma ruptures into the pulmonary veins, then the embolism can gain access to the arterial system and eventually migrate to the brain and cause neurological complications.

Treatment is highly supportive; it includes stopping air entry into the system, placing the patient in Trendelenburg and left lateral decubitus, applying 100% FiO_2 and in refractory cases, hyperbaric oxygen therapy [1, 2].

As far as we know, BiPAP has never been an approved modality for the treatment of vascular air embolism. In fact, there have been a few cases documented in the literature where patients undergoing non-invasive positive pressure ventilation, sustained vascular air embolisms from baro-trauma. These patients all had underlying acute lung infections at the time of therapy [3-5]. In our patient, the positive pressure ventilation may have just worked to alleviate work of breathing while supplying 100% FiO_2. Most recently, nitroglycerin given to rats and pigs prior to exposing them to high pressures, simulating diving, has been shown to decrease bubble formation [6-9]. One study showed decreased bubbled formation in human divers who received nitroglycerin prior to diving [10].

As far as we know, there have been no documented cases of vascular air embolism from improper port access in the clinical setting. Vachalova describes a fatal cerebral arterial gas embolism in and 81 year old male whose port that was used for parenteral nutrition was accessed by lay personnel at home [11].

This case highlights the importance of care when accessing subcutaneous ports, especially ones that are larger than the average sized ports, and the importance of early recognition of air embolism by the emergency physician and/or the intensivist, to rapidly begin supportive therapy. It also highlights the potential use of nitroglycerin and BiPAP in situations where hemodynamic collapse is not eminent, as adjuncts to supportive therapy in the treatment of vascular air embolism.

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