Case Report

Cerebral gas embolism in a case of Influenza A-associated acute respiratory distress syndrome treated with high-frequency oscillatory ventilation

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Abstract:

A 22-year-old obese asthmatic woman with Influenza A (H1N1)-associated acute respiratory distress syndrome died from cerebral artery gas emboli with massive cerebral infarction while being treated with High-Frequency Oscillatory Ventilation in the absence of a right to left intracardiac shunt. We review and briefly discuss other causes of systemic gas emboli (SGE). We review proposed mechanisms of SGE, their relation to our case, and how improved understanding of the risk factors may help prevent SGE in positive pressure ventilated patients.

Key words:

ARDS, cerebral gas embolism, high frequency oscillatory ventilation, influenza a H1N1, positive pressure ventilation, systemic gas embolism

A case of Influenza A (H1N1)-associated acute respiratory distress Syndrome (ARDS) in 2011 receiving high-frequency oscillatory ventilation (HFOV) complicated by fatal cerebral gas embolism is presented and followed by a discussion of risk factors and proposed pathophysiologic mechanisms for systemic gas emboli (SGE).

Case Report

A 22-year-old obese (BMI, 46 kg/m^2) asthmatic woman developed ARDS from influenza A subtype H1N1. Her hypoxemia was refractory to 100% oxygen delivered at 6 cc/kg ideal body weight tidal volumes with mean airway pressures (mPaw) of 25-30 cm of H_2O on conventional pressure control mechanical ventilation despite the use of neuromuscular blockade and inhaled nitric oxide [Figure 1]. Extracorporeal membrane oxygenation was considered but the patient's morbidly obese body mass index and evolving acute kidney injury proved prohibitive of this strategy. An echocardiogram demonstrated normal cardiac function without intracardiac shunt seen on saline bubble contrast study. Her hospital course was complicated by pneumomediastinum with subcutaneous emphysema [Figure 2]. On hospital day 5, an FIO_2 of 0.9 was required to maintain arterial PaO_2 of > 50 torr.

On hospital day 6, the mode of mechanical ventilation was changed to HFOV targeting mPaw of 35-40 cm H_2O, Power of 90 cm H_2O, Frequency of 5 Hz, Inspiratory time of 33% and FiO_2 of 0.8. The FiO_2 was decreased to 0.60 when the arterial PaO_2 improved to 50-60 torr [Table 1].

Forty-two hours after HFOV initiation (hospital day 8), physical examination revealed bilateral non-reactive pupillary dilation in the patient. Emergent bedside computed tomography of her head revealed diffuse cerebral edema, bi-cortical infarcts, brainstem herniation, and multiple right-sided cortical gas emboli [Figure 3]. Patient expired after institution of comfort measures and withdrawal of mechanical ventilation.

Discussion

Venous gas emboli from vascular access sites are known to travel systemically in the presence of a right to left intracardiac or intrapulmonary shunt such as in persistent foramen ovale, pulmonary arteriovenous fistulas, or anomalous origins of coronary arteries.[1] SGE have also been documented to occur in the setting of blunt and penetrating chest trauma as well as iatrogenically during invasive manipulation of the heart and lungs due to the creation of traumatic fistulae.[2-4] However, little is known about how SGE result secondary to positive pressure ventilation (PPV).

In our patient, the echocardiogram with saline bubble contrast did not visualize a right to left shunt of any kind and there was no surgical

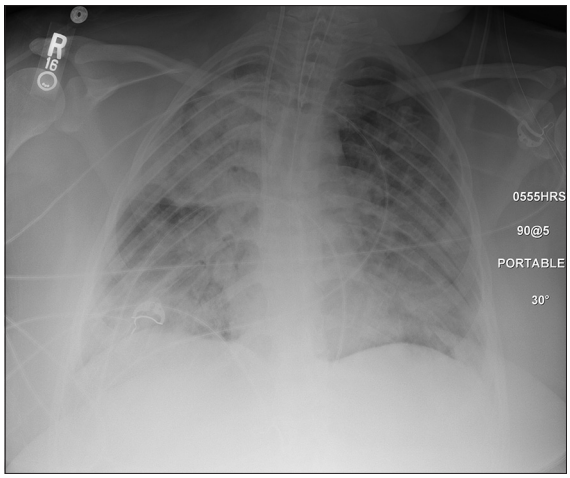

Figure 1: Chest X-ray of a 22-year-old obese asthmatic woman presenting in ARDS

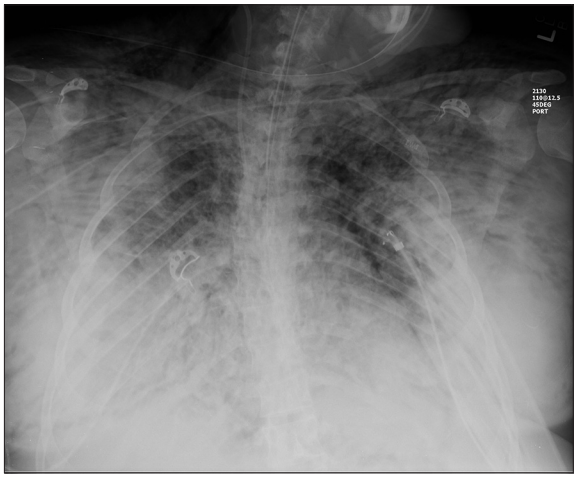

Figure 2: Chest X-ray demonstrating subcutaneous emphysema

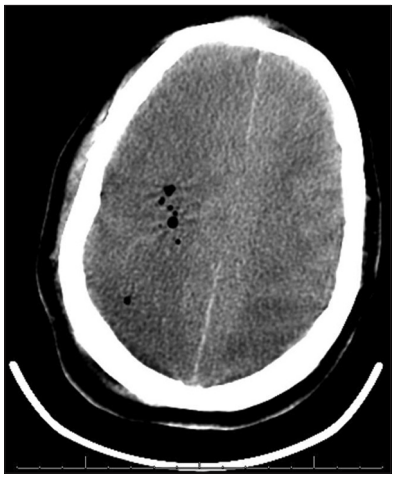

Figure 3: Computed tomography of the head demonstrating cerebral gas emboli

manipulation. Therefore, SGE were likely related to mechanical ventilation and/or the underlying lung injury.

A review of the literature reveals only a handful of case reports

Table 1: Ventilator settings and respective arterial blood gas analysis

Ventilator settings	ABG
PC = 13	pH = 7.20
Peep = 22	pCO_2 = 40 mm Hg
Rate = 23	pO_2 = 53 mm Hg
Paw (mean) = 30	
FIO_2 = 1.0	
Oscillator	pH = 7.31
Paw (mean) = 35	pCO_2 = 52 mm Hg
Frequency = 5 Hz	pO_2 = 58 mm Hg
Power = 90 cm H_2O	
FIO_2 = 0.8	
Oscillator	pH = 7.34
Paw (mean) = 40	pCO_2 = 39 mm Hg
Frequency = 5 Hz	pO_2 = 68 mm Hg
Power = 90 cm H_2O	
FIO_2 0.6	

Mode and settings of mechanical ventilation used and the cooresponding arterial blood gas

presenting non-paradoxical SGE attributed to PPV.[5,6] The mechanisms remain unknown. One explanation is that the high airway pressures that can result in subcutaneous emphysema, pneumomediastinum and/or pneumothorax (i.e., barotrauma) also can damage the capillary integrity of the bronchovascular tree and drive gas emboli into the pulmonary venous return. Ibrahim *et al*. illustrated this mechanism of SGE when they reported that right main-stem intubation and single lung ventilation ameliorated echocardiographic evidence of SGE in the setting of a left-sided pneumothorax. Left atrial and ventricular gas bubbles did not return after left chest tube insertion evacuated the pneumothorax and the endotracheal tube was pulled back to allow dual lung ventilation again.[5] This proposed mechanism is similar to the mechanism of SGE associated with bronchoscopic use of neodymium: yttrium-aluminum garnet (Nd: YAG) laser or Argon-Plasma Coagulation (APC) of endobronchial lesions.[7] APC or the laser compromises capillary or pulmonary venous integrity while airflow or endobronchial pressures are transiently high enough to drive gas into the bronchial venous system and travel systemically. In our patient, mPaw near 40 cm of H_2O were felt to have contributed to her barotrauma and disruption of the alveolar-capillary interface driving air into the mediastinal structures including the pulmonary venous return. However, airway pressures of this magnitude are not rare in HFOV and it has been shown that significant dampening of pressures occur within the tubing circuit and airways such that the lung experiences a lower pressure on HFOV than what is displayed on the ventilator, which is in contrast to conventional ventilation.[8] A conference of clinicians experienced in the use of HFOV produced a protocol for initiation, maintenance, and termination of HFOV published in 2007. This protocol recommends a starting mPaw of 34 cm H_2O and includes a titration table suggesting mPaw over 40 cm H_2O, if needed.[9] Although current thought variably associates "large" tidal volumes, "high" peak, and "high" mPaw with pulmonary barotrauma, published data have not demonstrated a clear and causative relationship. That is, evident pulmonary barotrauma is probably more dependent on the underlying disease processes that put the lung at risk (i.e., asthma, chronic interstitial lung disease, ARDS, and pneumonia).[10] It is

important to point out that our patient had both the significant pre-existing risk factors for barotrauma (i.e. pneumonia, ARDS, and asthma) with subsequent evidence of pulmonary barotrauma (subcutaneous emphysema) in the setting of generally considered high mPaw.

In this case, it is also possible that a patent foramen ovale opened due to high central venous pressures allowing paradoxical emboli from a venous access site to occur that was not visualized on the earlier echocardiogram with bubble evaluation, or that the saline bubble contrast study itself may have contributed to SGE due to mechanisms discussed below. A post-mortem evaluation was not obtained to refute this possibility. However, a patent foramen ovale or other congenital abnormalities do not have to be present for paradoxical gas emboli to occur. Butler and Hills demonstrated in 1985 that transpulmonary passage of gas can occur in the absence of a right to left intracardiac shunt when the filtering capacity of the lung is "overwhelmed" by large amounts of pulmonary arterial gas.[11] Weaver and Morris published a case of venous gas emboli that presumably traveled systemically through this mechanism resulting in fatal cerebral gas emboli.[12]

Conclusion

SGE are a rare complication of PPV. Lung injury is frequently present in most published cases of SGE relating to PPV, although lung injury is not a prerequisite for SGE to occur during PPV (i.e., transpulmonary vasculature passage of gas). Risk factors for pulmonary barotrauma include asthma, chronic interstitial lung disease, ARDS, and pneumonia. It remains to be seen what peak pressures, plateau pressures, or mPaw are considered "safe" or unlikely to cause pulmonary barotrauma. It may be that different disease states affect what pressures are considered "safe." Perhaps, permissive hypoxemia could be a reasonable alternative to normoxia when mPaw reach uncomfortably high levels. Extracorporeal membrane oxygenation should also be considered. This technology allows the use of much lower airway pressures when compared with alternative strategies. Additionally, transpulmonary vasculature passage of gas bubbles from venous sources requires that healthcare providers be vigilant about venous access sites to minimize this catastrophic and potentially preventable event.

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