Cardiol Young 2004; 14: 102–105

ISSN 1047-9511

Brief Report

Echocardiographic characteristics of venous air embolism presenting as reversible pulmonary atresia in a premature neonate

Abhay Divekar,[1] Rebecca Cases,[2] Reeni Soni[1]

[1]Section of Pediatric Cardiology, Variety Children's Heart Center, Children's Hospital; [2]Section of Neonatology, WR 004, Health Sciences Center, Winnipeg, Manitoba, Canada

Abstract Air embolism secondary to mechanical ventilation is a rare but well-described complication in premature infants. We describe the echocardiographic appearance of venous air embolism manifesting as acute obstruction of the right ventricular outflow tract in such a premature infant, and review the pathophysiology of acute obstruction of the right ventricular outflow tract secondary to the "air lock" phenomenon. Awareness of the pathophysiology and echocardiographic appearance of venous air embolism may aid in prompt recognition and potential therapy for this lethal complication of mechanical ventilation.

Keywords: Air embolism; mechanical ventilation; echocardiography

Sudden and significant amounts of venous air embolism are known to produce acute blockade of right ventricular output.[1] The consequent lack of flow of blood to the lungs, and the impaired venous return to the left heart, leads rapidly and progressively to a fatal outcome. The blockage is secondary to development of an "airlock" in the most dependent position of the right ventricle, namely the right ventricular outflow tract.[1] In this report, we discuss unique echocardiographic findings of venous air embolism, showing how survival was dependent on the fortuitous presence of an atrial communication and an arterial duct, part of normal neonatal physiology.

Case report

The patient was born at 27-weeks gestation. The antenatal history was significant for premature rupture of the membranes three weeks prior to delivery. He was delivered in a peripheral hospital, and immediately intubated because of severe hyaline membrane disease. He was placed on assisted mechanical ventilation, with peak inspiratory pressure of 30 cmH_2O, and positive end expiratory pressure of 6 cmH_2O. The rate of respiration was maintained at 55 breaths/min, with an inspiratory time of 0.3 s, and 100% fraction of inspired oxygen. Blood gases sampled post-ductally showed mild respiratory acidosis, and moderate systemic hypoxemia, with a partial pressure of oxygen in the arterial blood gas of 74 torr. Surfactant was administered, the systemic blood pressure was supported with dopamine, and broad-spectrum empiric antibiotic therapy was started for presumed sepsis. After appropriate ventilatory adjustments, he was transported to our intensive care unit by fixed wing air ambulance, maintaining the cabin pressure at ground level. On arrival in our intensive care unit, we noted continuing severe systemic hypoxemia, with a partial pressure of oxygen of 39 torr, for which we started high frequency ventilation. Our oscillator settings were: fraction of inspired oxygen at 100%, frequency of 7 Hz, mean airway pressure of 20 cmH_2O, and pressure change of 35 cmH_2O. He continued to develop progressive severe arterial desaturation, which was unresponsive to 100% oxygen and 40 parts per million of nitric

Correspondence to: Abhay Divekar MD, Assistant Professor, Variety Children's Heart Center, FE-241, 685 William Avenue, Winnipeg, Manitoba R3E 0Z2, Canada. Tel: 204 787 2410; Fax: 204 787 2004; E-mail: adivekar@exchange.hsc.mb.ca

Accepted for publication 3 September 2003

oxide, with a partial pressure of oxygen of 24 torr. Cardiac consultation was sought to rule out the presence of cyanotic congenital heart disease.

Clinical examination was limited to evaluation of vital signs being monitored. Auscultation of the cardiovascular system was not possible secondary to the high frequency ventilation. Echocardiography showed normal systemic and pulmonary venous return, with concordant atrioventricular and ventriculo-arterial connections. There was moderate tricuspid regurgitation, predicting systemic right ventricular systolic pressures. There was predominantly right-to-left shunting across the oval foramen. The arterial duct, however, shunted left-to-right at low velocity. There was a mild decrease in left ventricular systolic function, but the right ventricle was dilated and had poor systolic function. There was no antegrade flow across the normally sized right ventricular outflow tract, although there was evidence of pulmonary insufficiency. Highly reflective echodensities were seen in the right ventricular outflow tract (Fig. 1). Simultaneously, echodensities with characteristics of intracavitary air were visualized in the left atrium, the left ventricle, and the aorta. This was followed by an acute bradycardic episode of less than 10 s that spontaneously resolved prior to initiation of chest compressions. Immediately after the event, the left and right ventricular myocardium was studded with echodensities consistent with air embolism (Fig. 1c). Immediate inspection of the central lines and infusions failed to demonstrate any evidence of inadvertent air embolism.

At this time, we made the diagnosis of pulmonary atresia with an intact ventricular septum, noting the unusual features of a normal sized right ventricle and tricuspid valve, poor right ventricular systolic function, and the presence pulmonary insufficiency, suggesting functional rather than anatomic atresia. The acute bradycardic episode was attributed to air embolism in the coronary arterial bed. Although a lower partial pressure of oxygen in the arterial blood gas would now be acceptable, we anticipated problems with oxygenation secondary to severe hyaline membrane disease. We elected to discontinue high frequency ventilation and use conventional ventilation. Nitric oxide was also discontinued because of lack of response. The patient was started on an infusion of prostaglandin at 0.05 μg/kg/min to maintain patency of the arterial duct. Partial pressure of oxygen in the arterial blood gas stabilized at 39 torr, as did the systemic blood pressure. The fraction of inspired oxygen was to be gradually weaned. After initial stabilization, the patient again progressively deteriorated, with development of severe metabolic acidosis in spite of administration of bicarbonate, systemic hypotension, and progressive severe systemic

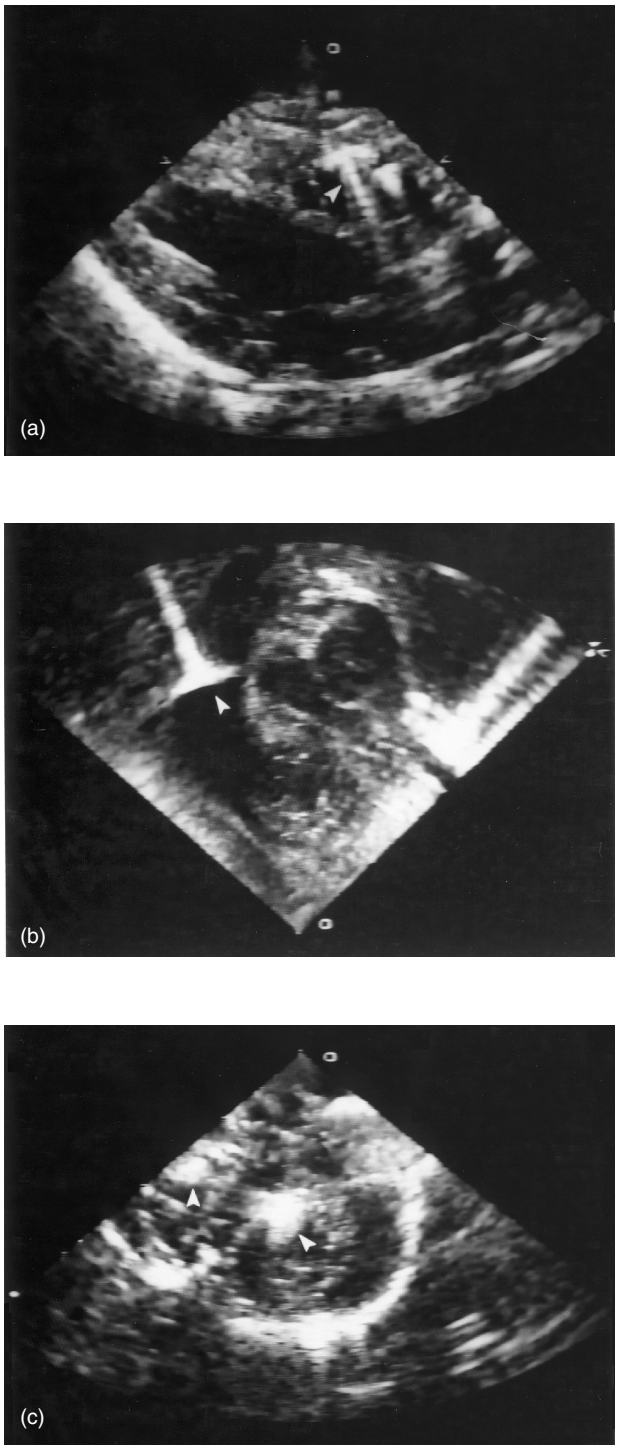

Figure 1.
The echocardiographic parasternal long axis view (a) shows highly reflective echodensities in the right ventricular outflow tract, with the characteristic "ring down" artifact (arrowhead), consistent with intracavitary air. The subxiphoid, sagittal view (b) shows an accumulation of intracavitary echodensities in the anterior portion of the right ventricular outflow tract, once again with characteristic "ring down" artifact (arrowhead), consistent with intracavitary air. The parasternal short axis view (c) shows intramyocardial air in the left and right ventricular myocardium (arrowheads).

https://doi.org/10.1017/S1047951104001210 Published online by Cambridge University Press

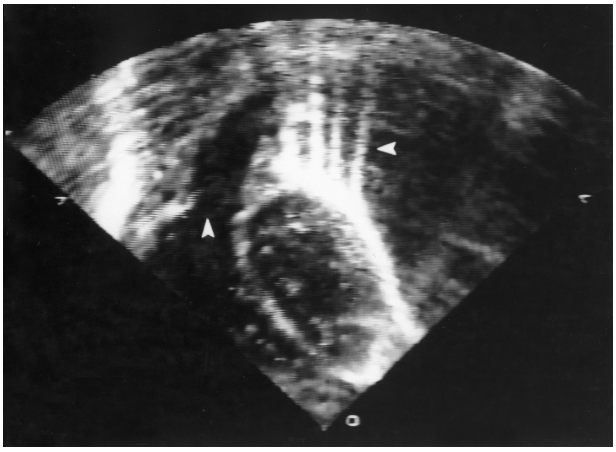

Figure 2.
The echocardiographic subxiphoid coronal view shows a patent right ventricular outflow tract without any intracavitary echodensities (arrowhead in right ventricular outflow tract). Also seen in this still frame is the appearance of air along the pericardial/pleural reflection, showing the characteristic echocardiographic appearance of air with a "ring down" artifact (arrowhead).

hypoxemia while still receiving 100% fraction of inspired oxygen. Increasing doses of inotropic agents were needed to support the arterial blood pressure. Nitric oxide was administered again as a last resort to improve systemic oxygenation.

Surprisingly, there was significant improvement in the saturations of haemoglobin obtained post-ductally, from 60–65% to 100%, and the partial pressure of oxygen increased to 63 torr. Repeated echocardiography now showed a pure left-to-right shunt across both the oval foramen and the arterial duct. There was mild tricuspid insufficiency, and the right ventricular systolic function improved. There was now normal antegrade flow across the right ventricular outflow tract (Fig. 2). The previously seen echodensities in the right ventricular outflow tract had disappeared, as had the echodensities in the right and left ventricular myocardium. There was rapid resolution of the metabolic acidosis, and the fractions of inspired oxygen and nitric oxide could be weaned over the next to 4 to 5 h to 30% and 5 parts per million, respectively. Prostaglandin infusion was discontinued. The remainder of the hospital course was similar to that for any premature infant. At the time of discharge, he was haemodynamically stable, with evidence of mild chronic lung disease but not requiring inhalation of oxygen.

Discussion

To the best of our knowledge, this is the first report describing the echocardiographic manifestations of venous air embolism in a premature infant occurring as a complication of mechanical ventilation. Our experience supports previous descriptions showing that venous air embolism can cause acute obstruction of the right ventricular outflow tract secondary to development of an air lock, and mitigates against a recent publication suggesting that development of an air lock may not be important in the pathophysiology.[2] The haemodynamic effects of air embolism have been extensively studied.[1,3] The effects of systemic air depend predominantly on the site of embolization, with an extremely small amount of air being fatal if the coronary bed is involved.[1] The effects of pulmonary air embolism, on the other hand, depend on the amount as well as the rapidity with which the air is introduced. Small amounts of air, entering the circulation slowly, typically cause peripheral pulmonary embolization, with the clinical manifestations secondary to pulmonary arterial hypertension.[1,3] A larger amount of air introduced suddenly, however, causes acute obstruction of the right ventricular outflow tract, secondary to the development of an airlock.[1] The right ventricular outflow tract is the site of obstruction because it is the most anterior and superior portion of the heart with the infant in the supine position.[1] Total blockage of right ventricular output leads to hypovolemic shock because of impaired venous return to the left heart, while the lack of flow of blood to the lungs results in severe hypoxemia. This combination is typically fatal. The subsequent pulmonary arterial hypertension produced by the distal embolization during dissipation of the "airlock" was now responsive to reuse of nitric oxide in the face of antegrade flow of blood to the lungs.

Systemic air embolism manifesting as an acute coronary event was correctly diagnosed on the basis of characteristic echocardiographic appearance of intracavitary air. The echocardiographic appearance of the myocardium was also correctly attributed to intramyocardial air, as previously described.[4] The echodensities in the right ventricular outflow tract, in contrast, were not correctly identified as the mechanism of obstruction. It was lack of awareness that the venous air embolism could also produce an air lock that prevented us initially recognizing the mechanism of the obstruction in the right ventricular outflow tract.

The source of the air embolism in our patient is speculative, but is believed to be secondary to mechanical ventilation. Decompression sickness and inadvertent introduction of air are unlikely, given that cabin pressure was maintained at ground level, and there was lack of any obvious iatrogenic source. The time course of events further supports this hypothesis. Coincidental discontinuation of high frequency mechanical ventilation probably eliminated any persistent source of air embolism. Pulmonary

and systemic air embolism is a well-documented but rare complication of mechanical ventilation, especially in premature infants.[5–7] This complication has also been reported in full-term infants, and on rare occasions in adults.[6,8] Air embolism in premature infants has a uniformly poor prognosis, with very few survivors reported in world literature.[5]

We attribute the survival in our patient, in spite of complete obstruction of the right ventricular outflow tract, to the normal neonatal physiology occurring in the first days of life. The right-to-left shunt across the oval foramen allowed for decompression of the systemic venous return, while the continued patency of the arterial duct allowed for pulmonary perfusion and systemic oxygenation.

If present, pulmonary insufficiency is held to be a sign of functional rather than anatomic atresia.[9] This feature should, therefore, always be sought during echocardiographic assessment, since it strongly suggests a functional cause. The finding is especially helpful in patients with congenital cardiac disease.

Awareness of the complication, and its echocardiographic features, may have permitted us to attempt manoeuvers that might have dissipated the air lock. A variety of manoeuvers have been described, which include turning the patient into the left lateral position, chest compressions, use of a Swan-Ganz catheter to aspirate air, and transthoracic needle aspiration.[1,10] Change in position, and chest compressions, can be performed easily and rapidly, and may be of clinical use without any potential harm. None of the methods, however, consistently provide relief of obstruction of the right ventricular outflow tract. With the improving survival of extreme premature neonates, and increasing use of aggressive modalities providing mechanical ventilation, it becomes important to recognize this relatively rare complication. Awareness of the complication, coupled with knowledge of the pathophysiology of venous air embolism and the echocardiographic appearances, may aid in the care provided to these fragile infants.

Acknowledgement

We thank Joy McCort and Raymond Saurette, cardiovascular technologists at the Variety Children's Heart Center, whose input was critical in the recognition of the diagnosis.

References

1. Durant TM, Long J, Oppenheimer MJ. Pulmonary (venous) air embolism. Am Heart J 1947; 33: 269–281.
2. Geissler HJ, Allen SJ, Mehlhorn U, Davis KL, Morris WP, Butler BD. Effect of body repositioning after venous air embolism. An echocardiographic study. Anesthesiology 1997; 86: 710–717.
3. Adornato DC, Gildenberg PL, Ferrario CM, Smart J, Frost EA. Pathophysiology of intravenous air embolism in dogs. Anesthesiology 1978; 49: 120–127.
4. Chandraratna A, Ashmeg A, Pasha HC. Detection of intracoronary air embolism by echocardiography. J Am Soc Echocardiogr 2002; 15: 1015–1017.
5. Lau KY, Lam PK. Systemic air embolism: a complication of ventilator therapy in hyaline membrane disease. Clin Radiol 1991; 43: 16–18.
6. Oppermann HC, Wille L, Obladen M, Richter E. Systemic air embolism in the respiratory distress syndrome of the newborn. Pediatr Radiol 1979; 8: 139–145.
7. Tan GH, Sussmane JB. Images in clinical medicine. Massive air embolism in a neonate with pulmonary hypoplasia. N Engl J Med 2002; 346: 751.
8. Weaver LK, Morris A. Venous and arterial gas embolism associated with positive pressure ventilation. Chest 1998; 113: 1132–1134.
9. Smallhorn JF, Izukawa T, Benson L, Freedom RM. Noninvasive recognition of functional pulmonary atresia by echocardiography. Am J Cardiol 1984; 54: 925–926.
10. Alvaran SB, Toung JK, Graff TE, Benson DW. Venous air embolism: comparative merits of external cardiac massage, intracardiac aspiration, and left lateral decubitus position. Anesth Analg 1978; 57: 166–170.