Vascular Air Embolism in Neonates: A Literature Review

Qi Zhou, MD, PhD[1] Shoo K. Lee, MBBS, FRCPC, PhD, DHC, OC[1,2]

[1] Department of Pediatrics, University of Toronto, Toronto, Ontario, Canada
[2] Department of Pediatrics, Mount Sinai Hospital, Toronto, Ontario, Canada

Address for correspondence Shoo K. Lee, MBBS, FRCPC, PhD, DHC, OC, 700 University Avenue, Room 8-500, Toronto, Ontario, Canada, M5G 1Z5 (e-mail: shoo.lee@sinaihealth.ca).

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Abstract

Neonatal vascular air embolism is a rare but often fatal condition. The literature comprises mostly case reports and a few dated systematic reviews. Our objective was to review all case reports of neonatal vascular air embolism to date and provide up-to-date information about patient characteristics, clinical presentations, outcomes, pathogenesis, diagnosis, prevention, treatment, and prognosis. We searched the literature for case reports of neonatal vascular air embolism, using MEDLINE, CINAHL, and EMBASE, and the keywords "neonates" and "vascular air embolism." Results were summarized. There were 117 cases of neonatal vascular air embolism, with a mean gestational age of 30.4 weeks (range: 23–40), mean birth weight of 1,422 g (range 830–3,844), and median age of occurrence of 2 days (range: 1–540) after birth. The majority were preterm (75.2%), male (62.7%), on assisted respiratory support (90.5%), and had air leak syndrome (52.9%). The most common clinical presentation was sudden acute clinical deterioration, sometimes accompanied by crying, cardiac rhythm abnormalities, skin discoloration, and a decrease in end-tidal carbon dioxide concentration. Incidence of mortality and adverse neurological sequelae among survivors was 73.9 and 16.6%, respectively, overall, but significantly ($p < 0.05$) higher among preterm infants (81.8 and 31.2%, respectively) and lower among surgical infants (23.8 and 0%, respectively). Diagnosis included visualizing air in infusion lines or retinal vessels, a decrease in the end-tidal carbon dioxide levels, and radiographic, doppler ultrasound, transesophageal echocardiography, or computed tomography (CT) imaging. The prognosis for neonatal air embolism is poor, especially for preterm infants requiring mechanical ventilation. Prevention is key and treatment is supportive.

Keywords

- vascular air embolism
- neonates
- preterm infants

Key Points

- Vascular air embolism is a rare but often fatal neonatal condition that is often underrecognized.
- Preterm infants on mechanical ventilation and with air leak syndromes are at particular risk.
- Prognosis is poor, prevention is key, and treatment is supportive.

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Vascular air embolism is a rare but often fatal condition in neonates.[1] It occurs commonly, but not exclusively, in preterm infants who are on mechanical ventilation. The literature about neonatal vascular air embolism comprises mainly case reports and does not lend itself to any meaningful estimation of prevalence. There are few comprehensive systematic reviews about neonatal vascular air embolism, and available ones are dated.[1,2] Its rarity and the lack of awareness among clinicians compound the difficulties associated with prompt diagnosis and effective management. The objective of this systematic review was to examine all case reports of neonatal vascular air embolism to date and provide up-to-date information about the patient characteristics, clinical presentations, outcomes, pathogenesis, diagnosis, prevention, treatment, and prognosis of neonatal vascular air embolism. This will increase clinician awareness and understanding about neonatal vascular air embolism, and facilitate early diagnosis and effective treatment to achieve optimum outcomes.

Materials and Methods

Since Lee and Tanswell[1] conducted a literature search for cases prior to 1989, we conducted a literature search from 1986 to 2024, for case reports of neonatal vascular air embolism published in the English language, using MEDLINE, CINAHL, and EMBASE, and the keywords "neonates," "newborns," "newborn infants," "preterm infants," "air embolism," "venous embolism," and "arterial embolism." The search generated 173 results. We excluded six inaccessible articles and duplicate cases. We reviewed all cases, including cases identified by Lee and Tanswell,[1] and tabulated them according to patient characteristics, clinical presentations, and outcomes. We summarized the results and presented them together with a review of the pathophysiology, diagnosis, prevention, and treatment. For statistical analysis, the chi-square test was used to examine differences in incidence rates, using the Social Science Statistics chi-square calculator.[3] Patients or the public were not involved in the design, conduct, reporting, or dissemination plans of our research. This study does not involve human participants.

Patient Characteristics

We identified 117 cases of neonatal vascular air embolism from 63 single case reports and 16 case series (►**Supplementary Material S1** [available in the online version]).[1,2,4–26] The mean gestational age (GA) was 30.4 weeks (range 23–40), mean birth weight (BW) was 1,422 g (range 250–3,400), and a median age of occurrence was 2 days (range 1–540) after birth (►**Table 1**). The majority were preterm (75.2%, $n = 88$) and male (62.7%, $n = 64/102$), among those with documented GA and sex. Most of the vascular air embolism events occurred in the neonatal intensive care unit (82%, $n = 96$), and the remainder occurred during surgery (17.9%, $n = 21$). At the time of vascular air embolism, 90.5% ($n = 106$) of infants were on assisted respiratory support, including hand bagging (1%), continuous positive airway pressure (4.2%), intermittent mechanical ventilation (73.5%), and high-frequency oscillatory ventilation (3.4%). Reasons for assisted respiratory support included respiratory distress syndrome, meconium aspiration, viral pneumonia, amniotic fluid aspiration, congenital alveolar dysplasia, and surgery. Air leak syndrome was present in 52.9% ($n = 62$); these included pulmonary interstitial emphysema (PIE) in 34.1% ($n = 40$), pneumothorax in 27.3% ($n = 32$), pneumomediastinum in 11.1% ($n = 13$), pneumopericardium in 10.2% ($n = 12$), and pneumoperitoneum in 3.4% ($n = 4$). Preterm infants were more likely to have air leak syndromes compared to term infants (64.7 vs. 24.1%).

Clinical Presentation

The cause of vascular air embolism was attributed to lung injury/assisted respiratory support in 70% ($n = 82$), introduction of intravascular air during a surgical procedure in 17.9% ($n = 21$), accidental intravenous (IV) or catheter injection of air in 8.5% ($n = 10$) infants, necrotizing enterocolitis in 1.7% ($n = 2$), cardiopulmonary resuscitation (CPR) in 1% ($n = 1$), and trauma in 1% ($n = 1$; ►**Table 2**). Infants with lung injury/assisted respiratory support were predominantly preterm, low BW, and male, whereas surgical infants were mostly term and equal in sex distribution.

The most common presentation was acute clinical deterioration, with desaturation, bradycardia, hypotension, collapse, and drowsiness. A cry or gasp of short duration was reported in two infants and was likely a response to hypoxia and air hunger.

Cyanosis, pallor, and mottling were commonly associated with non-specific generalized skin discolorations and reflected hypoperfusion and oxygen deprivation resulting from circulatory collapse. Non-specific localized transient skin discolorations, including blanching, blue-black, red, or vivid patches, and migrating areas of pallor in the extremities, were reported in six infants (7.3%) with lung injury/assisted respiratory support and one infant (5%) with surgery, but not among infants with accidental IV air injection or other causes. Non-specific skin discolorations are likely the result of vasospasm and vasodilation of cutaneous blood vessels as they redistribute blood in response to cutaneous hypoperfusion and hypoxia during circulatory collapse. Air bubbles can also cause transient skin discoloration through blood vessel occlusion or spasm induced by irritation of the gas.[1,2] Petechiae were noted in one infant but they appeared before the onset of vascular air embolism and are likely due to other causes.

There are few pathognomonic cutaneous signs of vascular air embolism in infants. Lee's sign[1,4] (pink red blood vessels superimposed on the cyanosed background) is a specific skin discoloration that has only been reported in infants with vascular air embolism and is attributed to direct oxygenation of erythrocytes adjacent to free air in the vascular system, while the tissues continue to be poorly perfused and oxygenated. Liebermeister's sign[5] (sharply defined area of pallor in the tongue) has been described in decompression sickness but has not been reported in neonates with vascular air

Table 1 Characteristics, associated phenomena, and outcomes of infants with air embolism

	All infants ($n = 117$)		Preterm ($n = 88$)		Term ($n = 29$)	
Characteristics	**Value**	**Range/*N***	**Value**	**Range/*N***	**Value**	**Range/*N***
Gestation (mean weeks; *n*)	30.4 ($n = 94$)	23–40	28.8 ($n = 77$)	23–36	39.5 ($n = 14$)	37–40
Birth weight (mean grams; *n*)	1,422 ($n = 91$)	830–3,400	1,214 ($n = 77$)	250–2,660	2,875 ($n = 11$)	1,400–4,150
Male:female ratio (N = male:female)	1.6:1	$N = 64{:}38$	1.8:1	$N = 49{:}26$	1.2:1	$N = 15{:}12$
Embolism age (median days; *n*)	2 ($n = 107$)	1–540	2 ($n = 77$)	1–260	45 ($n = 27$)	1–540
Surgery (*n*; %)	21	17.9%	7	7.9%	14	48.2%
Associated phenomena	*n*	Percentage (%)	*n*	Percentage (%)	*n*	Percentage (%)
Air leak syndrome	64	54.7	57	64.7	7	24.1
Interstitial emphysema	40	34.1	39	44.3	1	3.4
Pneumothorax	32	27.3	27	31.6	5	17.2
Pneumomediastinum	13	11.1	12	13.6	1	3.4
Pneumopericardium	12	10.2	11	12.5	1	3.4
Pneumoperitoneum	4	3.4	3	3.4	1	3.4
Abnormal EKG	7	5.9	5	5.6	2	6.8
Air withdrawal from central catheter	12	10.2	6	6.8	6	20.6
Outcomes						
Death	85/117	73.9	72/88	81.8	14/29	48.2
Adverse neurological sequelae in survivors	5/30	16.6	5/16	31.2	0/14	0

Abbreviation: EKG, electrocardiogram.
Note: *n* denotes infants with documented gestation, birth weight, sex, and embolism age.

embolism. Air in the retinal vessels was reported in one infant.

Cardiac arrhythmia and electrocardiogram abnormalities, including tachycardia, bradycardia, ST elevation, arrhythmia, ventricular fibrillation, and asystole, were reported in 5.9% ($n = 7$). A millwheel murmur and diminished heart sounds were heard in some cases. Air bubbles were withdrawn from a central catheter in 10.2% ($n = 12$). A decrease in end-tidal carbon dioxide concentration ($EtCO_2$) was noted in 9.4% ($n = 11$), reflecting reduced cardiac output and volume-related hypotension. Inappropriate high arterial oxygen concentration was recorded from an intra-aortic oxygen electrode in one case.

Pathogenesis

Neonatal vascular air embolism can be spontaneous or iatrogenic.[1,2] Spontaneous vascular air embolism is mostly arterial in nature and commonly occurs when air is injected into the pulmonary veins by mechanical ventilation,[9] especially in preterm infants with respiratory distress syndrome. Air leak syndromes, including PIE, pneumothorax, pneumomediastinum, pneumopericardium, pneumoperitoneum, and necrotizing enterocolitis, preceded vascular air embolism in 52% of reported cases. Barotrauma from high intrabronchial pressures during mechanical ventilation can cause microscopic alveolar rupture and fistularization distal to the terminal bronchioles,[2] creating a direct communication between the airway, interstitium, and small vascular channels. Air may enter into the intrapleural space and cause air leak syndromes, or into the pulmonary veins and cause vascular air embolism. Air can also gain access to the systemic venous system via the lymphatic ducts.[10] The amounts of air required to cause fatal embolism could be as little as 3 to 5 mL/kg[11] in infants. Air can often be found in both the arterial and venous system because of retrograde flow into the right heart through an incompetent pulmonary valve, and passive retrograde flow of gas bubbles because of their buoyancy.[1]

Iatrogenic vascular air embolism is usually venous in nature. Air can be introduced into the venous circulation through IV lines because of inadequate flushing, infusion sets running "dry," cracks in the tubing, or the use of infusion pumps without pressure or air-sensing technology.[2,4–12] Lung laceration has been reported in 25 to 30% of chest tube insertions and may favor reversal of the intrabronchial pressure pulmonary venous pressure gradient, resulting in pulmonary vascular air embolism.[1,13] During cesarean

Table 2 Clinical presentation and outcomes of vascular air embolism in infants by causative categories

	Lung injury/assisted respiratory support	Surgery	Accidental IV injection of air	NEC/other
Number (*n*)	82	21	10	4
Characteristics				
Gestation (wk)	29.2 (range 23–40)	36.4 (range 32–40)	31 (range 25–40)	36.6 (range 30–40)
Birth weight (g)	1,325 (range 250–4,150)	2,527 (range 1,000–3,400)	1,140 (range 830–1,760)	2,525 (range 2,350–2,700)
Sex	47M/25F	11M/8F	6M/2F	0M/3F
Presentation				
Desaturation	6	7	1	0
Bradycardia/arrhythmia	9	3	1	0
Hypotension	6	4	0	0
Collapse	4	1	2	1
Cry/Gasp	1	0	1	0
Drowsy	0	0	1	0
Skin discoloration				
Nonspecific generalized	15 (18.2%)	1 (5%)	2 (20%)	0
Cyanosis	11	1	1	0
Pallor	4	0	1	0
Mottling	2	0	1	0
Nonspecific localized	6 (7.3%)	1 (4.7%)	0	0
Blanching	2	0	0	0
Blue-black patches	1	1	0	0
Red patches	1	0	0	0
Livid discoloration	1	0	0	0
Migrating areas of pallor in extremities	1	0	0	0
Specific				
Lee's sign	2	0	0	0
Outcome				
Death	70 (85.3%)	5 (23.8%)	7 (70%)	1 (100%)
Neurological sequelae among survivors ($n = 30$)	5 (41.6%)	0	0	0

Abbreviations: F, female; IV, intravenous; M, male; NEC, necrotizing enterocolitis.

section, air can enter the cut lumen of large veins and sinusoids during incision of the placenta, and cause vascular air embolism.[14] CPR may also cause neonatal vascular air embolism.[15] Halbertsma et al[27] reported an 89% incidence of vascular air embolism in a postmortem study of unsuccessful CPR in newborn infants and postulated that frail pulmonary alveoli may rupture into blood vessels during neonatal CPR. Finally, air can enter the vascular system during ECMO or surgical procedures,[28] including repair of congenital heart disease, ventriculostomy, ventriculoperitoneal shunt, cranial remodeling, foramen magnum decompression, removal of brain tumor, duodenal surgery, ileostomy, fundoplication, liver transplantation, peritoneal dialysis, arthrogram, intraosseous infusion, and abscess irrigation.[6,16–26] Venous air embolism typically results in right-sided cardiac air but in neonates, venous air can access the arterial systemic circulation through the patent foramen ovale, especially in the face of raised pulmonary vascular pressures.[1,2]

Injuries resulting from vascular air embolism are mostly due to the mechanical effects of bubbles occluding flow in the circulatory system, together with the spasm of the vessels induced by irritation of the gas.[1,2] Air bubbles in the heart can cause an air lock that disrupts the circulation of blood. Occlusion of blood vessels supplying major organs can cause organ damage, for example, coronary arteries (myocardial ischemia and infarction, arrhythmia) and cerebral arteries (seizures, stroke, hypoxic–ischemic encephalopathy).[2] Vascular air embolism can also induce acute injury to the microvasculature through the release of oxygen free radicals from activated neutrophils, leading to increased permeability and pulmonary edema, the release of thromboxane A2, pulmonary vasoconstriction, and increased vascular

resistance and lung lymph flow.[28–31] Animal studies show that much of the brain dysfunction that follows vascular air embolism may be due to the effects of air on vascular endothelial cells, causing vasoconstriction and reduction in cerebral blood flow, rather than the effects of bubble entrapment.[32]

Outcomes

The overall mortality was 73.9% ($n = 85$) and the incidence of adverse neurological sequelae (cerebral palsy, severe [grade 3 or higher] intraventricular hemorrhage, hypoxic–ischemic encephalopathy) among survivors was 16.6% ($n = 5/30$). However, the outcomes depend on the maturity, cause, and setting of the vascular air embolism event.

Preterm infants compared to term infants had significantly ($p < 0.05$) higher incidence of mortality (81.8 vs. 48.2%) and adverse neurological sequelae among survivors (31.2 vs. 0%; ►Table 1). This is not surprising as preterm infants are more likely to have respiratory distress syndrome, require assisted respiration, and have air leak syndromes which predispose to spontaneous vascular air embolism. Lee and Tanswell[1] observed a correlation between the timing of vascular air embolism and GA or BW, but no relationship with inflation pressure, suggesting that barotrauma is inflicted earlier in the more immature lung and that the development of vascular air embolism is determined as much by the physical characteristics of the lung being inflated as by the characteristics of the inflation.

In contrast, surgical cases compared to non-surgical cases had significantly ($p < 0.05$) lower incidence of mortality (23.8 vs. 84.3%) and adverse neurological sequelae among survivors (0 vs. 35.7%). This may be because surgical patients were less likely to be preterm, vascular air embolism was more likely to be promptly diagnosed and treated in the intensively monitored and equipped environment of the surgical operating room, and some surgical patients were already on extracorporeal membrane oxygenation or cardiopulmonary bypass.

Venous air embolism is potentially less harmful than arterial air embolism because air bubbles are filtered out in the microvasculature of the lung and do not enter the arterial vasculature to cause organ failure. In infants, however, the small lung sizes can be overwhelmed by as little as 3 to 5 ml of air, and air can enter the arterial vasculature through the patent ductus arteriosus. In this cohort, the mortality rate was 85.3% among infants with arterial air embolism (lung injury/assisted respiratory support) and 70% among infants with venous air embolism (accidental IV air injection), while the incidence of neurological sequelae among survivors was 41.6 and 0%, respectively.

Diagnosis

Diagnosis requires awareness and a high index of suspicion.[1,2] In some cases, columns of air or a frothy mixture of blood and air, were withdrawn with blood from the umbilical arterial catheter. An inappropriate high arterial oxygen concentration recorded using a transcutaneous oxygen monitor, or from intra-aortic oxygen electrodes; and an abrupt decrease in the end-tidal carbon dioxide levels, demonstrated by capnometry, have been reported.[1,6] Air bubbles in retinal blood vessels were observed in one case of an infant undergoing eye surgery.[7] Radiography, doppler ultrasonography, transesophageal echocardiography, or computed tomography (CT) imaging are diagnostic, and free air may be seen in the arterial and venous systems, as well as in the heart, brain, or portal system.[1,2] Imaging should be performed antemortem. Postmortem radiographs need to be interpreted with caution as intravascular air may appear as early as 25 minutes after death.[8]

Prevention and Treatment

Nursing protocols to prevent vascular air embolism should be adopted.[33] These include priming of IV infusion sets, use of pressure or air-sensing technology in IV pumps and 0.2-micron in-line filters in IV infusion lines, employing head-down position and the Valsalva maneuver during insertion and removal of infusion lines, ensuring that an injection cap is attached to each lumen of central venous catheters, securely luer-locking all connections, clamping the catheter when opening the IV system, withdrawing any air within the injection site cap/catheter prior to injecting any fluid, avoiding use of scissors to remove IV line dressings, and clamping proximal to the damage if an IV line becomes disconnected or damaged. Avoiding high airway pressures and barotrauma, and minimizing the need for mechanical ventilation, are helpful strategies.[1,2] Using soft rubber catheters, instead of stiff plastic chest tubes, for drainage of pneumothoraces, may be less traumatic to the lung and reduce the risk of vascular air embolism.[1,13]

Awareness is critical for prompt diagnosis and treatment is largely supportive.[1,2] Prompt CPR, volume expander, and vasopressor support should be given as needed. The source of vascular air embolism should be promptly stopped, for example, clamp an IV line that is infusing air. If an umbilical artery or central catheter is present, air should be aspirated if possible. This may remove air bubbles causing airlock in the heart. The infant should be placed in a Trendelenburg and left decubitus position to reduce the risk of air bubbles embolizing the cerebral and coronary arteries and allow the entrapped air in the heart to be stabilized within the apex of the right ventricle, thereby relieving the obstruction of the pulmonary outflow tract. The infant should be given 100% oxygen to correct hypoxemia and increase the diffusion gradient for nitrogen out of the bubbles, causing them to shrink. Hyperbaric oxygen may be of benefit but there is a lack of clinical trials evidence to support this. Head or total body cooling may provide neuroprotection. Corticosteroids and/or barbiturates may also be considered.

Limitations

As this is a review of a small number of case reports, it is not meaningful for estimating prevalence, there is potential for reporting bias, and generalizability is limited. The review does not include case reports published in other languages than English.

Prognosis

The prognosis for neonatal air embolism is poor, especially for preterm infants requiring mechanical ventilation, who have a mortality rate of 81.8% and adverse neurological sequelae among survivors incidence of 31.2%. Prevention is key and treatment is largely supportive.

Authors' Contributions

Q.Z.: Conceptualization, methodology, investigation, data curation, writing—original draft. S.K.L.: Conceptualization, methodology, supervision, writing—review and editing.

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Conflict of Interest

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