Short reports

Air embolism in ventilated very low birthweight infants

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SUMMARY Five cases of air embolism in ventilated very low birthweight infants are reported. In all cases the outcome was fatal with the babies dying at about 15 hours of age.

Pulmonary air leaks are well recognised complications of ventilatory support in the newborn period.¹ Most of these leaks are confined to the tissue spaces within the thoracic cavity but in 1970 Gregory and Tooley reported a case of air embolism diagnosed on an x ray film taken at necropsy.² We report five cases of this uncommon complication seen in this hospital in the last six years and draw attention to the clinical features of this uniformly fatal condition.

Case reports

Case 1. A girl weighing 960 g was born after spontaneous onset of labour at 26 weeks' gestation. Only minimal resuscitation was required (Apgar scores 8 and 10) but she rapidly developed increasingly severe respiratory distress necessitating ventilation, and severe generalised pulmonary interstitial emphysema was noticed on chest radiography. Her condition deteriorated and she died at 19 hours of age. Just before death a sample from the umbilical artery catheter was seen to contain bubbles of air and bubbles of gas were seen to appear from a heel prick performed to sustain the diagnosis of air embolism; this was confirmed by an x ray film taken before necropsy.

Case 2. A boy weighing 1175 g was born by elective caesarean section at 28 weeks' gestation because of an antepartum haemorrhage. An earlier premature onset of labour at 24 weeks' gestation had been halted with intravenous salbutamol. He required ventilation from birth but quickly developed pulmonary interstitial emphysema and it proved impossible to maintain his oxygenation. Just before his death, at 14 hours of age, sampling of the umbilical

artery catheter showed air in the vessel and air embolus was confirmed on an x ray film taken immediately after death.

Case 3. A boy weighing 1170 g was delivered by emergency caesarean section at 28 weeks' gestation after a prolonged liquor leak after the mother's membranes had ruptured at 24 weeks. He was intubated from birth and it was difficult to maintain adequate ventilation. At 14 hours of age the baby suddenly deteriorated; the legs and abdomen became white and air was seen in the blood sample drawn from the umbilical artery catheter. The transcutaneous oxygen electrode was reading 18 kPa at the time. A chest x ray film taken before death showed a right pneumothorax, pneumomediastinum, and air in the ventricular cavity and outlining the major vessels.

Case 4. A boy weighing 880 g, the second of twins, was born by a breech delivery at 28 weeks' gestation after spontaneous rupture of the membranes 48 hours before. He was intubated from birth and his condition remained stable until at 15 hours of age he became visibly white with no cardiac output. The cardiac monitor registered a supraventricular tachycardia. The air entry on the right side was reduced and the insertion of a chest drain resulted in spontaneous resolution of the supraventricular tachycardia to sinus rhythm. A pneumothorax occurred on the left side almost simultaneously. There was still no cardiac output and because the indwelling umbilical artery catheter oxygen electrode was reading >20 kPa blood was withdrawn from the umbilical artery catheter and this contained air bubbles. A chest x ray film taken just before death showed bilateral pneumothoraces, pneumomediastinum, and air in the ventricles and outlining the major vessels (figure).

Case 5. A boy weighing 630 g was delivered at 24 weeks' gestation by an emergency caesarean section four days after an amniocentesis for polyhydram-

Figure Chest x ray film taken just before death in case 4. (a) This shows the bilateral pneumothoraces and a pneumomediastinum (1); air in the ventricle can be seen (2). (b) The area surrounding the left humerus has been enlarged to show air in the brachial vein (3). (The structure lying across the arm is the lead from the transcutaneous oxygen electrode.)

nios. The mother had been treated with antibiotics before delivery because of a fever and the infant received antibiotics from birth. The baby required ventilation from birth because of severe respiratory distress that appeared on chest x radiography to be due to surfactant deficiency. By 18 hours of age his respiratory condition had worsened despite the use of tolazoline and volume support and he required very high pressures to maintain his oxygenation. At 19 hours of age he suddenly deteriorated becoming mottled and blue despite the indwelling oxygen

electrode reading 18 kPa. 'Squelchy' heart sounds were heard on auscultation and a chest x ray film showed air in the chambers of the heart and great vessels and support was withdrawn.

The clinical details of the five infants are shown in the table.

Discussion

All five of these babies suffered massive air embolism with no evidence of accidental injection of air

Table Summary of clinical details of the five infants

	Case 1	Case 2	Case 3	Case 4	Case 5
Gestation (weeks)	26	28	28	26	24
Weight (g) [Centile]	960 [50%]	1175 [60%]	1170 [50%]	880 [50%]	630 [50%]
Prolonged rupture of membranes	—	—	+	+	—
Antepartum haemorrhage	—	+	—	—	—
Start of intermittent positive pressure ventilation	4 hours	Birth	Birth	Birth	Birth
Maximum positive inspiratory pressure (cm H ₂ O)	25	30	37	28	47
Maximum mean airways pressure (cm H ₂ O)	18	17	21	18	44
Lowest mean blood pressure (mm Hg)	21	26	28	31	17
Time of death (hours)	19	14	15	15	19
Last continuous PO ₂ reading (kPa)	18	3.8	2.5	20	18
Other Air Leaks					
Pneumothorax	—	—	—	+	—
Pneumomediastinum	—	—	+	+	—
Pneumopericardium	—	—	—	—	—
Pulmonary interstitial emphysema	+	+	—	+	—

via syringes or infusion sets. Death occurred rapidly after the event but in three patients antemortem chest *x* radiography confirmed the diagnosis and lead to immediate cessation of attempts at resuscitation. Clinical features which alerted us to the diagnosis in the three last patients were the pallor of the infant,² the discrepancy between the state of the baby and the high readings of the continuous oxygen monitor, and finally the presence of air in the samples drawn from the umbilical artery catheter. The presence of supraventricular tachycardia in the fourth case is interesting and to our knowledge air embolism has not previously been recorded as a precipitating cause for this arrhythmia.

In the early reports of this complication the babies tended to be of longer gestation (range 25–34 weeks) and emphasis had been placed on the higher pressures (range 28–90 cm H₂O) that had been used.^{2,3} It is noteworthy that the pressures in the first four infants in this study (like those in a similar infant reported by Rudd and Wrigglesworth⁴) were lower (25–37 cm H₂O) although they were obviously much higher than we would like to use. Three of the babies had pulmonary interstitial emphysema in which it is thought high peak airways pressure used during ventilation for hyaline membrane disease plays a causative role.⁵ Four out of the five infants had proved difficult to oxygenate and the use of this high pressure was determined by the evidence of atelectasis on the chest *x* ray film.

Four of the five infants had an additional form of air leak to their air embolism and it seems likely that the air enters the circulation after rupturing out of the pulmonary air spaces. Whether this occurs at the site of weakness in the pericardial reflection near the ostia of the pulmonary veins is not clear.⁶ Two of the subjects had prolonged rupture of the membranes

(in one case four weeks) but there was no evidence that either pulmonary hypoplasia or infection played a role in the pathogenesis of their embolism, although congenital infection may have played a role in the fifth subject. There was no evidence of meconium aspiration or inappropriate resuscitation at birth that might have predisposed these infants to this disaster nor was there any evidence of accidental introduction of air through the peripheral or central cavities. All infants were appropriate for gestational age and there was no evidence of any underlying congenital disorder.

It is of interest that in the patients reported here death occurred at about 15 hours of age. In our experience this parallels the time course of the peak severity in the respiratory distress experienced by these very low birthweight immature babies. In our unit 42% of ventilated babies, <26 weeks' gestation, develop air leaks. It seems likely that air embolism represents the extreme end of the range of air leaks that occur in very immature lungs.

References

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Accepted 1 December 1987

Clostridium difficile and acute enterocolitis

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SUMMARY *Clostridium difficile* belonging to groups not normally detected in infancy was the only potential pathogen detected in the stools of two infants with severe enterocolitis. Further information regarding the virulence of this organism was obtained by use of a recently introduced typing scheme.

Clostridium difficile is not usually considered to be of clinical importance in stool specimens from infants because this organism can also be found as part of their normal gut flora.¹ In adults, *C. difficile* is rarely isolated from normal faecal specimens and its overgrowth with production of toxin, secondary to antibiotic treatment, can result in the development of pseudomembranous colitis. In infancy there are only occasional reports of this condition.^{2,3} The