CASE REPORT

Neonatal Cerebral Air Embolism

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Background: Vascular air embolism (VAE) is rare but potentially lethal condition, and survival is rarely reported in newborn. ***Case Characteristics***: A preterm (27^{+1} weeks) neonate on Continuous positive airway pressure developed sudden cardiac asystole on day 3 of life and required 30 minutes of cardiopulmonary resuscitation. ***Observation***: Infant had air embolism in liver and brain. He survived but developed cystic encephalomalcia requiring extensive neuro-rehabilitation. ***Message***: Air embolism should be considered as differential diagnosis of sudden unexplained cardiac deterioration in well neonate.

Keywords: *Encephalomalacia, Neonatal stroke, Neonatal resuscitation*.

Vascular air embolism (VAE) is rare but potentially lethal accident, and survival is rarely reported in newborn [1,2]. Precise incidence of VAE is not known due to under-recognized and under-reported nature of this almost fatal catastrophe. We report a newborn with cerebral air embolism, its acute management, and neurodevelopment outcome at 4 months.

CASE REPORT

An extreme preterm (27^{+1} weeks), 895 gm, male infant was admitted with respiratory distress syndrome male (RDS) and was commenced on CPAP support (7 cm of water and 40% oxygen). He also required surfactant replacement therapy (two doses within 12 hours of birth). Parenteral nutrition was started through umbilical venous line since admission. Infant's respiratory distress improved gradually and he was started on trophic feeds on day-2 of life; intravenous infusion consisted only of parenteral nutrition and IV Caffeine. There was a sudden episode of cardiac asystole on day-3 of life with mottled and marbled skin. He required extensive cardio-pulmonary resuscitation (CPR) (30 minutes) and 4 doses of IV adrenaline. Blood gas analysis showed severe metabolic acidosis (pH 7.02, HCO_3 7.0 meq/L, PCO_2 30 mmHg) with normal potassium and calcium. Circulation could be restored only after 30 minutes of CPR with return of pulses and auscultable heart sounds. Infant required adrenalin infusion and mechanical ventilation as post resuscitative care. A bedside trans-illumination test ruled out pneumothorax, which was later confirmed with *X*-ray chest. A neuro-sonogram performed within one hour of the event showed presence of multiple echogenic specks with posterior acoustic shadowing in branching distribution suggestive of air bubbles in bilateral parietal and occipital regions within the cerebral parenchyma (***Web Fig*.1**). Abdomen sonogram showed presence of umbilical line in sub-diaphragmatic position, in portal vein with similar echogenic specks suggestive of air bubbles in the portal vein tributaries along the umbilical line. A 2D Echo performed 24 hours later showed normal cardiac function and presence of a PFO. A follow-up neuro-sonography after 2 days showed disappearance of these lesions confirming re-absorption of air specks. A continuous ECG tracing did not show any arrhythmia and QTc interval was normal.

Post-resuscitation, infant developed multiple multi-focal myoclonic seizure that required treatment with Phenobarbital and Levetiracetam. Sensorium improved gradually and infant could again be weaned to CPAP support in another seven days. A term corrected USG brain showed development of multiple parenchymal cystic spaces suggestive of cystic periventricular leucomalacia in parietal and occipital regions bilaterally. An electroencephalogram (EEG) done at 35 weeks was reported to be normal. Infant initially had severe truncal and axial hypotonia, which improved gradually during NICU stay with developmentally supportive care. Infant was discharged at 35 weeks Post Menstrual Age (PMA) on oral feeding at weight of 1920 gm (3^{rd} percentile) and head circumference of 29 cm (9^{th} percentile). Infant was started on early stimulation and intensive physiotherapy after discharge. BERA at 3 months was normal. Cortical visual impairment was diagnosed and visual rehabilitation was started. At corrected 4 months of age,

head circumference was less than 3rd centile, and spasticity of limbs was present with grade 3 neck control.

DISCUSSION

Cerebral air embolism in preterm neonate is mostly fatal with very few survivors reported till date [1,2]. The largest reported newborn case series (25 cases) of air embolism is of pre-surfactant era where very high pressure ventilation was used with almost universal fatal outcome [3]. The barotrauma causes seepage of air from ruptured alveoli into systemic circulation [3]. Other potential sources of VAE in newborn include advance necrotizing enterocolitis, central or peripheral vascular access, neurosurgery, cardiac surgeries and ECMO [4,5]. While smaller amount of air may be broken down in the system and may not cause ill-effects, large amount of air can be fatal. In the right side of circulation, it can cause air lock of the right heart resulting in absent right ventricular output and sudden death. The amounts of air require to cause fatal embolism could be as little as 3-5 mL/kg [4]. Air in cerebral circulation causes not only occlusive infarcts but also cascade of endothelial injury, disruption of blood brain barrier and cerebral edema which further expands the occlusive ischemic infarcts [6].

Cerebral air embolism can also be seen in preterm infant after prolonged cardiopulmonary resuscitation. This is likely to be due to frail pulmonary alveoli which rupture into blood vessels during neonatal CPR [7-9]. The source of air embolism in our case was unclear. USG abdomen showed presence of air in portal vein tributaries. UVC air could also be due to CPR or line handling for emergency medicines. The PFO in our case could be portal for the systemic air embolism (paradoxical air embolism). It is possible that cerebral air embolism in our case could be due to extensive CPR; however, we also believe that introduction of new air embolism in critical unstable newborn would have led to unsuccessful CPR [7]. Nevertheless the neurological devastation caused by air embolism is grave, irrespective of the source of origin.

Trans-oesophageal echocardiography is the most sensitive modality, and can detect 5-10 microns of air bubble. Precordial doppler USG / ECHO is most common used and readily available and can detect 15 microns of air bubble. [5]. Air embolism is reported in 89% of preterm neonates in largest reported series of unsuccessful CPR in post-mortem series [7]. True incidence of air embolism in survivors of CPR is not known.

Management of VAE includes prevention of further release of air (*e.g.*, covering the surgical site with wet mop in neurosurgery), reduction in the size of embolic air, and support of circulation with effective CPR and vasopressor. Administering 100% oxygen not only improves tissue oxygenation but also reduces air bubble volume by eliminating nitrogen. Air lock in the right side of heart may be relieved by partial left decubitus position. Chest compression in CPR will support the hemodynamics and will also break the air bubbles into smaller fragments [5]. Therapeutic hypothermia was found to be beneficial in an adult in improving neurological outcome [10].

Air embolism is a potentially preventable complication and should be kept as differential diagnosis in case of sudden unexplained deterioration in an otherwise stable neonate.

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