

# Infant presenting with pulmonary haemorrhage as a sequelae of accidental asphyxiation

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August 4, 2020

## Abstract

Pulmonary haemorrhage in children is rare. Respiratory infections, foreign body aspiration, cystic fibrosis and congenital heart diseases remain the leading causes. We present a 7- week-old male infant, previously fit and well who developed pulmonary haemorrhage while breastfeeding in a sling strapped to mother. We hypothesise that accidental asphyxiation during breast feeding, generated forced inspiratory effort against an obstructed upper airway and led to negative pressure pulmonary oedema (NPPE) and pulmonary haemorrhage. Accidental asphyxiation should be considered in the differential diagnosis of children presenting with pulmonary haemorrhage.

## LETTER TO THE EDITOR

### Infant presenting with pulmonary haemorrhage as a sequelae of accidental asphyxiation

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Conflict of interest: None

Keywords: pulmonary haemorrhage; negative pressure pulmonary oedema; asphyxiation; intrathoracic pressure

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To the Editor,

Pulmonary haemorrhage, though a well-recognised entity is uncommon in paediatric population <sup>1-3</sup>. It can present as an insidious and chronic course or as an acute life-threatening event<sup>1,2</sup>. There is no clear data on the incidence of pulmonary haemorrhage in children <sup>1,2</sup>. Respiratory infections, cystic fibrosis and congenital heart diseases remain the leading causes <sup>1,2</sup>. Less common causes identified are foreign body aspiration,

upper airway bleeding, trauma (accidental, related to tracheostomy), suffocation (intentional or accidental), pulmonary hypertension or embolism or arteriovenous fistula, tumours, congenital lung malformations, primary hematologic bleeding, coeliac disease, Heiner's syndrome (cow's milk sensitivity), vasculitis and associated syndromes, idiopathic pulmonary haemorrhage of infancy, catamenial and factitious hemoptysis<sup>1,2</sup>. Pulmonary haemorrhage is not only rare but poorly understood in infants<sup>3</sup>. Suffocation leading to airway obstruction and in turn, leading to oronasal and pulmonary haemorrhage in infants is poorly studied<sup>3</sup>. Discrepancy and inconsistency in reporting these presentations also contribute to the poor understanding and availability of data<sup>3</sup>. McIntosh et al reported in a 10-year retrospective hospital study that the incidence of suffocation is 0.22/10 000 live births and 40% of them can progress to pulmonary haemorrhage<sup>3</sup>.

We describe a 7-week-old male infant, who presented to Emergency Department after mother noted gurgling noises followed by blood from mouth and floppiness. The incident occurred when the infant, previously fit and well was breastfeeding in a sling strapped to mother while she was walking in a farm with the older siblings. The only relevant history was coryzal symptoms noted two weeks before this episode.

In view of further clinical deterioration with respiratory failure, hypotension and bradycardia, he required a brief period of cardiopulmonary resuscitation, intubation and hypovolemic correction with fluids, red blood cells (haemoglobin of 91 g/L) and plasma. Frank blood was noted at mouth, vocal cords, nostril and through nasogastric tube on insertion. On admission to intensive care, he needed high frequency oscillation ventilation for about 24 hours following which he was switched to conventional ventilation and then extubated after 3 days.

Investigations done included chest radiograph (Figure 1) that showed diffuse ground glass opacification. Echocardiogram, cranial ultrasound, ophthalmology assessment and coagulation studies were normal. CT chest with contrast was performed 6 days after admission and was normal. Vasculitic and septic screenings were negative. Respiratory viral screen was positive for rhinovirus but thought to be of minimal significance in this clinical context.

Safeguarding team were involved in view of the nature of presentation. However following review of events there were nil concerns raised. The patient recovered well with no neurological sequelae and was discharged home after 8 days of hospital stay. He remains well at 12 months follow up.

## Discussion:

Negative Pressure Pulmonary Oedema (NPPE) is a known postoperative and anaesthetic complication and especially in cases of acute upper airway obstruction<sup>4,5</sup>. It was described as early as 1927<sup>5</sup>. Asphyxiation by choking /strangulation are also known causes of NPPE<sup>5</sup>.

We hypothesize that, pulmonary haemorrhage in this case is due to accidental asphyxiation during breast feeding in a sling, generating forced inspiratory effort against an obstructed upper airway and led to NPPE and haemorrhage.

NPPE is triggered when forced inspiratory efforts are generated to overcome the obstructed upper airway which leads to highly negative intra thoracic pressure (ITP). The obstruction also leads to hypoxia and hypercarbia leading to a catecholamine surge. There is an increase in venous return by reducing right atrial pressure due to the transmission of negative ITP and the elevation of the mean systemic pressure due to catecholamine-induced venoconstriction. The right ventricular volume increases leading to shifting of the interventricular septum to the left. The catecholamine surge also causes elevation of the systemic vascular tone leading to an increase in the left ventricular transmural pressure, raising ventricular wall tension which leads to an increase in afterload which then reduces left ventricular ejection fraction. The consequence of these changes is a net shift of blood from systemic to the pulmonary circulation. Pulmonary capillary pressures increase as a result of the increased blood flow and the increased vascular tone while intra alveolar pressures drop, and alveolar-capillary membrane breaks. Fluid and blood move rapidly into interstitial and alveolar spaces leading to NPPE and pulmonary haemorrhage<sup>4,5</sup>.

Approach to pulmonary haemorrhage should start with a careful history and examination to identify aetiology as listed before<sup>1-3</sup>.

Investigations should include full blood count and blood film to look for severity of haemorrhage and differentiate acute vs chronic anaemia<sup>1,2</sup>. Erythrocyte sedimentation rate (ESR) and C reactive protein (CRP) to look for evidence of inflammation or infection, renal function tests in the context of sepsis or systemic vasculitis<sup>1,2</sup>. Coagulation studies to look for any coagulation related disorders<sup>1-3</sup>. Vasculitic screen to rule out systemic vasculitis<sup>1,2</sup>. Respiratory viral screens and cultures to look for any infectious pathology<sup>1-3</sup>.

Chest radiograph can show patchy or diffuse ground glass changes indicating haemorrhage and on occasions may point towards aetiologies such as focal infiltrates or consolidation suggestive of infective process, or unilateral hyperinflation suggestive of foreign body aspiration and other findings such as cavitation/nodules and hilar lymphadenopathy<sup>1,2</sup>.

CT chest with contrast can help identify extent and sequelae of pulmonary haemorrhage and provide further etiological clues<sup>1,2</sup>. Echocardiogram to look for any cardiac disease<sup>1,2</sup>.

Bronchoscopy might be useful in identifying focal active bleeds and for therapeutic interventions<sup>1,2</sup>. Broncho alveolar lavage can help find evidence of hemosiderin laden macrophages as an indication of previous pulmonary haemorrhage<sup>1,2</sup>. Lung biopsy to be considered when diagnosis is not conclusive<sup>1,2</sup>.

Investigations for specific aetiologies when suspected need to be carried out<sup>1-3</sup>. Management should include treatment of acute presentation, respiratory failure and treatment targeted towards underlying cause and its complications when identified<sup>1-3</sup>.

Pulmonary haemorrhage though uncommon can be a life-threatening entity. Accidental asphyxiation by suffocation should be considered in the differential diagnosis of children especially infants presenting with pulmonary haemorrhage.

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Informed consent was obtained from parent/legal guardian of the child

