CASE REPORTS

HYPERTHERMIA - A CAUSE OF HEMORRHAGIC PULMONARY EDEMA IN NEWBORN

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Abstract

A full term, vaginally delivered baby with normal apgar scores presented at 12 hours of age with respiratory distress, hyperthermia, capillary filling time > 3 seconds and X-Ray chest showing bilaterally white lung fields. Chest examination revealed bilateral crepts & endotracheal suctioning revealed small amount of blood. Investigations revealed no evidence of sepsis & coagulation profile was normal. A provisional diagnosis of pulmonary edema or congenital pneumonia was considered. Patient was put on ventilatory support with high peak end-expiratory pressure [PEEP] along with standard resuscitative measures. There was marked improvement within 48 hours and chest X-ray showed clearing of bilateral lung fields. Considering the initial X-ray picture, its rapid clearance, clinical course and other investigations, a final diagnosis of pulmonary edema with hemorrhage was made, hyperthermia being the causative factor. Unphysiological extremes of temperature can disrupt cellular integrity, and when it occurs at alveolar - endothelial level, it may cause pulmonary edema and hemorrhage.

Keywords: Pulmonary edema, hyperthermia, newborn.

Introduction

Pulmonary edema is the abnormal accumulation of water and solute in the interstitial and alveolar spaces of the lung. Normally, fluid is filtered from capillaries in the alveolar septa into the alveolar interstitium and then siphoned into the low pressure extra-alveolar interstitium, from where it is drained by the pulmonary lymphatics. (1) Pulmonary edema occurs when the rate of fluid filtration exceeds the rate of lymphatic removal. This can occur by one of the three mechanisms: (a) increased filtration pressure, (b) increased vascular permeability or (c) decreased lymphatic drainage. (2) An extreme form of the high permeability pulmonary edema may precipitate pulmonary hemorrhage- as evidenced by the fact that the hematocrit in lung effluents obtained from patients with pulmonary hemorrhage is relatively low compared to whole blood [15-20% less]. (3) Extensive search on Pubmed and Sciencedirect did not reveal any previously reported case in children.

Case Report

The patient is a male infant who was born by normal vaginal route at 39 weeks gestation to a 23 year old multipara mother with normal antenatal period. Apgar scores were 8, 9 and 9 at 1, 5, and 10 minutes, respectively. He was appropriately grown for his age, weighing 2954 grams, head circumferance of 34.5 cm and length 53 cm. The baby presented to us with increased work of breathing at 12 hours of age and was transferred to the neonatal intensive care unit (NICU). Detailed family and social history revealed that his father was a watchman and the family lived in a slum area in a one room dwelling. On examination, he was found to have respiratory distress, hyperthermia (skin temperature: 39.2o Celsius), moderate amount of dehydration and prolonged capillary filling time (> 3 seconds). Chest examination showed bilateral crepitations and endotracheal suctioning revealed small amount of blood. Per-abdominal examination revealed normal shape and contour with no organomegaly. X-ray chest showed bilateral white lungs (Fig. 1). A provisional diagnosis of pulmonary edema with hemorrhage or congenital pneumonia was considered. The baby was intubated and mechanically ventilated using high positive end-expiratory pressure (PEEP) levels. Investigations revealed hemoglobin 13.2 gm%, white blood count 8560 cell/cumm with 52% neutrophils. Band cells were 10%. C- reactive protein was 6mg/L. Blood culture revealed no growth. Coagulation profile in form of platelet count; prothrombin count; partial thromboplastin time; international normalized ratio; fibrinogen levels were within normal range. Blood urea and serum creatinine levels were also normal. Echocardiography was normal with no evidence of patent ductus arteriosus. The infant showed marked clinical improvement during the ensuing 48 hours and was successively weaned off mechanical ventilation, nasal continuous airway pressure, and was breathing room air by 72 hours of age. X-ray chest at 60 hours of life showed bilateral clearing of lung fields (Fig. 2). He was observed for further 24 hours before being discharged. Considering the entire course of events, a final diagnosis of pulmonary edema with hemorrhage was made for which the only apparent cause was hyperthermia.

FIGURE 1: X-ray chest on admission showing bilateral white lung fields

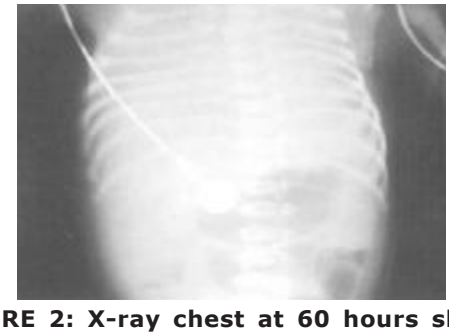
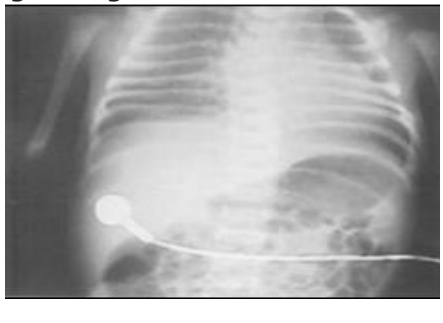


FIGURE 2: X-ray chest at 60 hours showing clearing of lung fields.



Discussion

Differential diagnoses of white-out lung in newborn include hyaline membrane disease, congenital pneumonia, surfactant protein B deficiency, acute left ventricular failure, pulmonary edema and hemorrhage. (4) Pulmonary hemorrhage is defined as the presence of hemorrhagic fluid in the trachea accompanied by a respiratory decompensation requiring increased respiratory support or intubation within 60 minutes of appearance of the fluid. In pulmonary hemorrhage, the clinical spectrum varies from blood-tinged tracheal or pharyngeal secretion to massive intractable bleeding into the lungs. Factors that alter the integrity of the epithelial-endothelial barrier in the alveolus or that change the filtration pressure across these membranes could predispose infants to pulmonary hemorrhage. (3-7) Known associations of pulmonary hemorrhage include intrapartum asphyxia, prematurity, intra-uterine growth retardation, respiratory distress syndrome, infection, hemodynamically significant patent ductus arteriosus, maternal blood aspiration, diffuse pulmonary emboli, hypothermia, coagulation abnormalities and urea cycle defects associated with hyperammonemia. (4) Iatrogenic causes include over hydration, direct trauma by endotracheal tube, instillation of surfactant bolus, ventilation in high FiO2 and extracorporeal membrane oxygenation. (4) None of these factors were present in this case. However, hyperthermia was present. Hyperpyrexia in adults is known to cause pulmonary edema. (8) Unphysiological extremes of temperatures can disrupt cellular integrity & when it occurs at alveolar-endothelial level, it can cause pulmonary hemorrhage. (9) It may be responsible for some of the sudden infant deaths in neonatal age group. (10) The cause of hyperthemia in this case was probably the hot summers in this region during the month of May when day temperatures range between 40-45o C coupled with inadequate cooling facilities and the relative delay in effective feeding in exclusively breastfed babies.

Prompt management of hyperthermia can be life saving & can alleviate the risk of pulmonary hemorrhage in neonates. In our country, with high ambient temperatures in summers and many of the dwellings not having adequate cooling facilities, this condition must be more rampant than reported. Hence the message to parents is to keep babies cool in summers; the doctors should also keep this possibility in mind where relevant.

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