Radiological Case of the Month

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A MALE INFANT of 35 weeks' gestational age had mild respiratory distress shortly after birth. He weighed 2640 g after a pregnancy complicated by breech presentation and pregnancy-induced hypertension, resulting in a cesarean delivery with clear amniotic fluid and no risk factors for infection. Apgar scores were 7 and 8 at 1 and 5 minutes, respectively.

During transport to the newborn nursery, the infant became dusky, developed sternal retractions, and showed 81% oxygen saturation by pulse oximetry. He was afebrile, and findings from physical examination revealed tachypnea, subcostal retractions, and decreased breath sounds present on the left. Therapy was begun with delivery of supplemental oxygen by hood at a fraction of inspired oxygen of 35%. Levels for serial C-reactive proteins and complete blood cell count were normal as were findings from blood culture. Antibiotic therapy was started on admission to the intermediate care nursery. The arterial blood gas level 2 hours after birth showed a pH of 7.22; PaO_2, 60 mm Hg; and $PaCO_2$, 58 mm Hg. A chest radiograph was obtained and repeated 6 hours later (**Figure 1** and **Figure 2**). Within 6 hours, after management by positioning in the left lateral decubitus position and delivery of oxygen by nasal cannula, the respiratory distress, hypercarbia, and acidosis had improved. The infant was discharged following 10 days of antibiotic therapy for presumed pneumonia.

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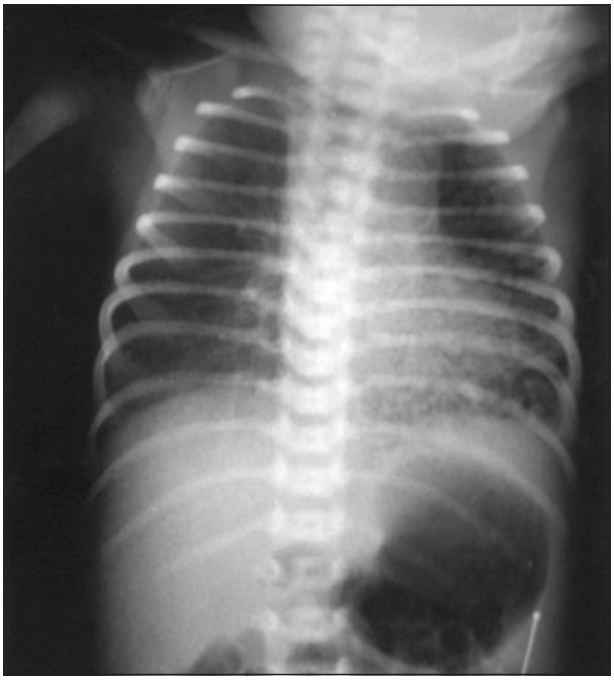

Figure 1.

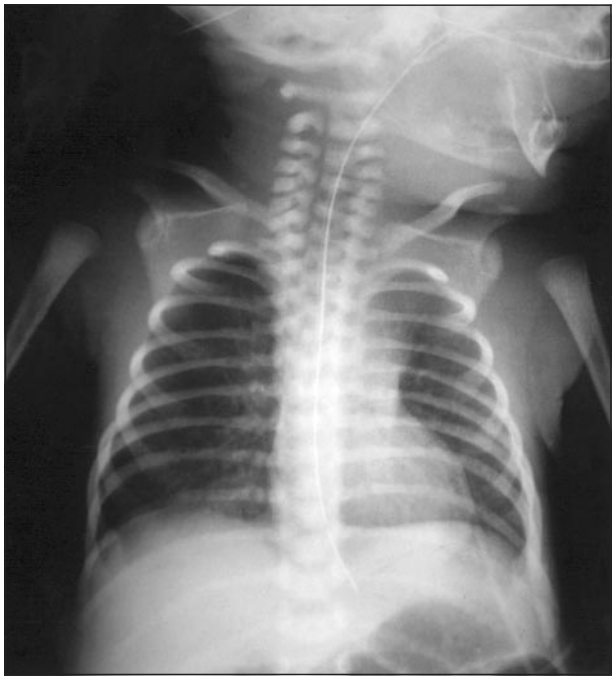

Figure 2.

Denouement and Discussion

Pulmonary Interstitial Emphysema in a Nonventilated Preterm Infant

Figure 1. *Chest radiograph (anterior-posterior view) showing left lower lobe consolidation with multicystic and linear lucencies of pulmonary interstitial emphysema.*

Figure 2. *Repeated chest radiograph (anterior-posterior view) 6 hours later showing almost complete resolution of pulmonary interstitial emphysema.*

Respiratory distress is a frequent problem encountered in newborn infants. In the preterm infant, respiratory distress syndrome occurs resulting from decreased production of surfactant. Signs of tachypnea, sternal retractions, and hypoxia in the near- or full-term infant can be caused by transient tachypnea of the newborn, associated with delayed resorption of fetal lung fluid. In both the preterm and full-term child, respiratory distress may indicate pneumonia, sepsis, congenital heart disease, anemia, central hypoventilation associated with asphyxia, or intrinsic pulmonary mass.

Pulmonary interstitial emphysema (PIE) is encountered in very low-birth-weight infants in the first 48 to 96 hours of life. The infants receive mechanical ventilatory assistance because of poorly compliant or immature lungs. Resultant volutrauma[1] leads to rupture of overdistended alveoli and resultant dissection of air into the interstitium—the bronchovascular bundles, interlobular septa, and lymphatic channels.[2] The diagnosis is based on radiography. Dissection of extra-alveolar air into the pleural leads to air-leak syndromes such as pneumothorax, pneumomediastinum, and pneumopericardium.

Pulmonary intersitial emphysema is considered an air "trap/leak" syndrome because alveolar air enters the interstitium and is also trapped by a check-valve phenomenon either at the site of dissection or from partial airway obstruction.[3,4] Partial bronchiolar obstruction results from aspiration of foreign material, endobronchial plug, or fluid, resulting in uneven expansion of the lung and abnormally high intra-alveolar pressures,[5,6] which may lead to PIE. Pulmonary interstitial emphysema occurred more frequently prior to the use of positive pressure ventilation (PPV). Localized PIE in an infant who has not received PPV may be mistaken for a different cause of respiratory distress such as lung cysts, congenital lobar emphysema, or cystic adenomatoid malformation.

Pulmonary interstitial emphysema can occur in up to one third of infants receiving conventional mechanical ventilation who weigh less than 1500 g with the incidence inversely proportional to decreasing weight. A review of 210 neonates mechanically ventilated for respiratory distress syndrome identified 41 with PIE. The most notable causative factor was high-peak inspiratory pressure.[3,7] Based on clinical, radiological, and pathological criteria, PIE is categorized as acute pulmonary interstitial emphysema, localized persistent interstitial emphysema, and diffuse persistent pulmonary interstitial emphysema. The persistent type is associated with the presence of giant cells, and when diffuse, it is associated with poor outcome.[2]

Treatment of a nonventilated infant with acute localized pulmonary emphysema should be conservative, including positioning with the affected side down, inhaled oxygen therapy by nasal cannula or oxygen hood and frequent monitoring of blood gases and chest radiographs. In seriously ill infants, extracorporeal membrane oxygenation, steroids, surgical resection, and high-frequency ventilation have been used.[8]

Pulmonary interstitial emphysema is a well-recognized complication of PPV for low-birth-weight infants but is an unusual occurrence in mature infants. The cause of the interstitial leak in this patient is thought to be secondary to partial obstruction of the lower lobe airways.

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