Multiple Organ Dysfunction Following Aspiration of Maternal Blood

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Abstract

We describe the case of a term newborn with multiple organs dysfunction, including respiratory distress, acute renal failure, and hypotension following aspiration of maternal blood around the time of birth. We also discuss potential mechanisms for local (pulmonary) and systemic effects of blood aspiration.

INTRODUCTION

Case reports of respiratory distress that follow maternal blood aspiration rarely described other organs function. Generally, that distress was attributed to an “aspiration” phenomenon without supplementing details on how is that aspiration cause pulmonary dysfunction.[1, 2] We describe a case of multiple organ dysfunction (MOD) and maternal blood aspiration and we offer potential mechanism for this under reported phenomena.

CASE

A female infant was born at 38 2/7 weeks’ gestation weighing 3050 grams (appropriate for gestational age) to a 20 year-old gravida 3, para 2 woman. Pregnancy was complicated by anemia. Maternal blood tests revealed blood type O-positive and anti-M antibody (non-IgG. Maternal medications included iron and multivitamins. The mother was admitted to the hospital 2.5 hours prior to delivery after spontaneous onset of labor. Cesarean section was performed because of a non-reassuring fetal heart tracing. At the time of uterine incision the amniotic fluid was noted to be frankly bloody with a 50 percent placental abruption. Maternal hematocrit was 21%. Cord blood gases; arterial: pH 7.2, pCO2 51.4 mmHg, base excess -6.3; venous: pH 7.3, pCO2 38.0 mmHg, base excess -6.7.

At birth the newborn was noted to be vigorous with Apgar scores of 7 and 8 at 1 and 5 minutes, respectively. She was noted to have blood-stained secretions from her nose and mouth, tachypnea, subcostal retractions, and an intermittent cough. She was supported initially with high flow nasal cannula at 5 L/minute advancing to mechanical ventilation at 4 hours of life. Bloody fluid was suctioned from the endotracheal tube immediately following intubation. A chest radiograph revealed low lung volumes and bilateral scattered airspace opacities (Figure 1a). A chest radiograph obtained one day later showed improved lung volumes and persistence of the scattered airspace opacities (Figure 1b).

The infant was initially managed with pressure control, (assist/control), with peak inspiratory pressure (PIP) 20 cm H_2O, positive end-expiratory pressure (PEEP) 6 cm H_2O, FiO_2 1.0. Venous blood gas an hour after intubation was pH 7.29, pCO_2 47 mmHg, HCO_3 22 mmol/L, S_pO_2 91%. Oxygenation improved gradually and FiO2 and ventilatory pressures were weaned accordingly. She was extubated at 2.7 days of life to nasal cannula support that was discontinued day later.

She was treated empirically with a seven day course of ampicillin and tobramycin for presumed pneumonia.

In the delivery room, the infant was transfused with uncross-matched O-negative packed red blood cells. At 0.5 hours of life a CBC showed hematocrit 39%, white blood cells 21.7 K/mM^3, platelets 348 K/mM^3 and a 12 nucleated RBC/100 WBC. The infant had a rapid rise in total bilirubin from 5.7 mg/dL at 4 hours of life to 16.8 (15.8 indirect, 1.0 direct) at 25 hours of life. She was treated with phototherapy initiated at 8 hours of life. She was also treated with intravenous immunoglobulin (IVIG) at 25 hours of life as serum bilirubin was approaching an exchange transfusion level. This patient’s blood group was O-positive and her serum antibody screen was negative. Peripheral blood smear at one day of life showed microcytic anemia with polychromasia

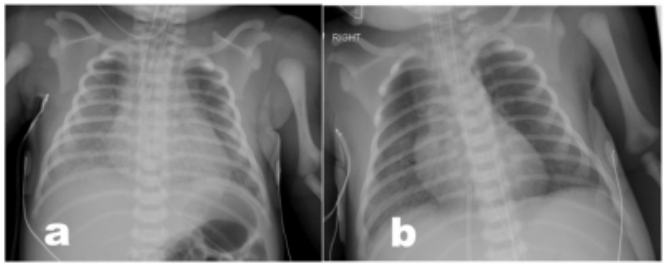
and mild anisopoikilocytosis, including spherocytes, suggestive of a hemolytic process.

Analysis of urine obtained at 8 hours showed a red and cloudy appearance with elevated protein and blood. However, microscopic urine examination showed no red blood cells. At 24 hours of life serum sodium was 135 mmol/L, potassium 2.9 mmol/L , creatinine 1.2 mg/dL ,urea nitrogen 29 mg/dL, glucose 110 mg/dL , and calcium 7.8 mg/dL . Serum creatinine dropped to 0.7 mg/dL the next day, and by the third day all these studies were within the normal range. Urine output was decreased over the first day of life and improved after a 20 mL/kg intravenous fluid bolus and treatment with dopamine at 20 hours of life. Dopamine dose reached a maximum of 10 mcg/kg/min and was discontinued by day of life 4. A renal ultrasound at approximately 36 hours of life demonstrated increased renal vascular resistance that was absent on a repeat ultrasound at 80 hours of life. An echocardiogram on the second day of life estimated right ventricle and pulmonary artery systolic pressures to be systemic to mildly supersystemic with a small-to-moderate left-to-right shunt across the atrial septum.

This infant also had melena noticed shortly after birth that resolved over two days. Abdominal radiographs did not show pathologic findings. Feeding was started on day of life 5 and was tolerated well. She was discharged at day 11 day of life. At the two month outpatient follow-up visit, there were no apparent sequelae found.

Figure 1

Figure 1. Chest radiograph, a) shortly after endotracheal intubation, b) one day later.



DISCUSSION

Aspiration of maternal blood along with other amniotic fluid debris, has previously been implicated as a main cause of respiratory distress in term and late preterm newborns.[1, 2] Over the last three decades there has been further understanding of the mechanisms of how impaired pulmonary fluid clearance contributes to respiratory distress and transient tachypnea of the newborn (TTNB).[3, 4]

Elements in blood such as proteins and lipids cause surfactant dysfunction through several mechanisms that include competitive binding to the alveolar space, a step needed for appropriate surfactant function.[5-7] This surfactant dysfunction can be an important factor causing respiratory distress following blood aspiration.

The early onset of respiratory distress, which lasted less than a week seen in our case is similar to other reported cases of respiratory distress following maternal blood aspiration.[2, 8-10]

Our findings add to that of Saia et al, who reported a case of a newborn with MOD consisting of respiratory failure, hyperbilirubinemia, and renal dysfunction. [8] Intrauterine distress can be associated with fetal gasping and aspiration of maternal blood, and with hypoxemic injury to other organs. However, the cord blood gases and prompt response to minimal neonatal resuscitation disputes the significance of this element. We postulate that the MOD could be the result of disseminated inflammation which accompanies mechanism of ventilator induced lung injury. [11] MOD has been better described in acute respiratory distress syndrome, another condition with a predominantly inflammatory pulmonary process. [12]

Respiratory signs caused by aspiration of maternal blood can be difficult to distinguish from TTN and aspiration of other substances such as meconium, vernix, or purulent amniotic fluid. Initial chest radiographic evaluations also are not particularly sensitive.[13]

This infant had unconjugated hyperbilirubinemia with no identified isoimmunization. There is no existing literature describing the renal effects from maternal blood aspiration. However, Saia et al reported a case of a neonate with macroscopic hemoglobinuria detected shortly after birth in which hemoglobin was determined to be adult type by electrophoresis.[8] Renal failure, in this case, could have been caused by consequences of a not well-defined hemolytic process contributing to elevated free serum hemoglobin. This, in turn, antagonized endothelial nitric oxide, resulting in oxidant injury and impaired renal microcirculation. It is possible that serum free hemoglobin precipitated in the ultrafiltrate forming casts, causing the urine to appear dark (as we observed) and obstructing the proximal tubule.[14] Each of these potential mechanisms could decrease glomerular filtration and acutely increase

serum creatinine.

This infant received intravenous immunoglobulin (IVIG) therapy. Renal failure is also a reported complication of IVIG therapy.[15] However, this complication has not been substantiated in the neonatal population.

Clinician taking care for infants with maternal blood aspiration need to be mindful of the multiple organ dysfunction which we describe in this case. More observations and investigations are needed to further define and explain this phenomenon.

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