PNEUMOTHORAX IN A NEWBORN WITH MECONIUM ASPIRATION SYNDROME : A Case Report

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ABSTRACT

Pneumothorax is the dissection of air into the pleural space. MAS has become 20% cause of pneumothorax. The thick, viscid meconium can result in partial airway obstruction with air-leak syndrome as the complication. We found respiratory distress in this baby who born through meconium-stained amniotic fluid (MSAF). The risk factor of this case was amniotic membrane rupture 3 days before delivery, oligohydramnion and preeclamsic mother . During the observation, the baby became more cyanotic and respiratory distress. Chest X-Ray examination revealed avascular lucent area at right lateral hemithorax, as a sign of pneumothorax. The baby got chest tube insertion and intubated with oxygen support ventilator. The serial thorax x-ray evaluation showed improvement of lung expansion, without air leakage in right pleural. After 7 days, the chest tube was taken off.

INTRODUCTION

Pneumothorax is the dissection of air into the pleural space; thus sufficient accumulation of air can cause tension pneumothorax. Although sometimes asymptomatic, pneumothorax causes acute deterioration of a neonate in a neonatal intensive care.[1] Primary etiology included pneumonia and sepsis (30%), hyaline membrane disease (20%), meconium aspiration syndrome (20%) and congenital diaphragmatic hernia (10%). Pneumothorax was significantly associated with increased mortality in neonates and prolonged length of Neonatal Intensive Care Unit (NICU) stay [2]

Meconium aspiration syndrome (MAS) is defined as respiratory distress in a neonate born through meconium-stained amniotic fluid (MSAF) having characteristic radiological changes whose symptoms cannot be otherwise explained.[3,4] MAS has become 20% cause of pneumothorax.[5] The thick, viscid meconium can result in partial or complete airway obstruction. Partial obstruction causes air trapping and hyper-distension of the distal airway, commonly termed the ball valve effect. The gas that is trapped may rupture into the pleura resulting in air leaks.[6] Frequency of air leak was found to be 24% in neonates having MAS. Mechanical ventilation has been implicated as a risk factor for development of pneumothorax in MAS. [7]

The use of mechanical ventilation and coexisting diseases have been reported to be important risk factors that can affect the prognosis of pneumothorax. Clinical data

suggest that complications such as volutrauma and air-leak syndromes can negatively affect long-term pulmonary and non-pulmonary outcomes during mechanical ventilation. In the present study, 60% of all cases and 90% of fatal cases received mechanical ventilation. [8]

The prognosis of pneumothorax and MAS is poor. Prompt recognition and treatment of this condition is lifesaving. Pneumothorax typically causes worsening of tachypnea, grunting and cyanosis, as well as decreased breath sounds on the affected side.[9] But, by early detection and prompt intervention, a newborn with MAS complicated pneumothorax could be saved with good prognosis.

The objective of this case was to report the case of pneumothorax as complication of meconium aspiration syndrome in a newborn, focusing on the early diagnostic approach and intervention.

CASE REPORT

H, female baby, weighing 2000 grams, was born by emergency caesarean section for premature rupture of membrane, oligohydramnion and preeclamsia. The amniotic membranes were ruptured for 3 days prior to delivery, and the amniotic fluid was meconium stained.

She was born from 30 years old mother, from 3rd pregnancy, with no history of abortion. The mother has history of routine antenatal care by midwife every month. The routine fetal prenatal screening and obstetric ultrasonography during pregnancy were all normal. Her mother had no history of fever or sign of infection. Before pregnancy, her mother also in good nutritional state within normal range body mass index. Her weight during pregnancy raised about 10 kg. Two month before giving birth the baby's mother also developed hypertension. Shortly before delivered the mother developed orthopnea and general edema. The mother was admitted to emergency department of Sumenep Hospital and referred to Dr. Soetomo Hospital for emergency caesarean section.

Although the calculation of gestational age based on the last first day of menstrual cycle was 33-34 weeks, the physical examination for ballard score was equal to 40 weeks of gestational age. The birth weight was 2000 grams, with 40 cm in length, and the head circumference 32 cm.

The infant was not vigorous at birth. The muscle tone and crying were weak. The heart rate was below 80 times per minute. The baby was placed under a radiant warmer so that the resuscitation team has easy access to the baby without causing excessive heat loss. She was positioned on her back (supine), with the head and neck neutral or slightly extended to opens the airway and allows unrestricted air entry. Brief, gentle endotracheal suction was done to remove meconium and clear the airway. We did suction the mouth before the nose to ensure that there is nothing for the newborn to aspirate if she should gasp when the nose is suctioned. After that, the baby was gently dried from any fluid with warm blanket to prevent evaporative heat loss. Positioning, clearing secretion and drying baby usually provide enough stimulation to initiate breathing. But, the baby still did not have adequate respiration. So, we did tactile stimulation by gently rub the newborn's back, trunk, or extremities.

However, after doing initial step of resuscitation, Neonatal Resuscitation was done until the baby got positive pressure ventilation. The baby still did not have adequate breathing and the heart rate still below 100 beats per minute. In a prolonged period of impaired gas exchange, stimulation alone will not work and positive pressure ventilation will be required. We gave the baby positive pressure using T-

piece resuscitator with PIP 30mmH_20 and PEEP 5 mmH_20. Reevaluation showed heart rate of this baby was increased more than 100 beats per minute. The baby was put on nasal CPAP with PEEP 5 mmH_20 before she was transferred to the neonatal intensive care unit (NICU) for further management.

From physical examination at NICU, she was lethargic, with heart rate 130 beats per minute, respiratory rate 70 breaths per minute. The Apgar scores were 3 and 5 at 1 and 5 minutes, respectively. There was noted to have respiratory distress with tachypnea and increased use of accessory muscles. From physical examination revealed dyspnea, with nostril breathing that improved with administration of O_2 CPAP PEEP 7 mmH_20. Chest examination revealed symmetric breaths movement, with same air entry heard on both sides, subcostal and intercostal retraction and fine throughout the lung field. Cardiovascular examination showed normal heart sound, no audible murmur, and normal peripheral pulses. Abdominal examination revealed normal bowel sound, with no liver or spleen enlargement. The extremity was reddish and warm. Laboratory examination was between normal limit.

From anthropometry measuring, revealed weight for age (WAZ) < -2, length for age (LAZ) < -2, weight for length (WLZ) < -2, and head circumference (HC) is median. (Figure 1)

On the first hours, the baby's chest retraction became worse and got desaturated, from SpO_2 96% to 84%. The baby look cyanotic and respiratory distressed with RR 80-90 breath per minute. The baby was intubated again and tubed in ventilator. The ventilator was set to PCV PIP 20 mmH_20, PEEP 5 mmH_20, rate 60, and FiO_2 50%.

The initial laboratory examination showed: Hemoglobin 19.4 mg/dL, WBC 19.160/µL, PCV 59.1%, Plt 287,000µL, Albumin 4.01 g/dL. CRP 1.03mg/dL, pH 7.48, pCO_2 24.6 mmHg, pO_2 103 mmHg, TCO_2 19 mmol/L, HCO_3 18.6 mmol/L, BE -5 mmol/L, SO2 99%.

As further follow up, the baby underwent x-ray examination. Chest X-Ray examination revealed avascular lucent area at right lateral hemithorax, as a sign of pneumothorax She got O_2 support by ventilator with PCV setting PEEP 5 PIP 20 rate 60 FiO_2 50% and increased to FiO_2 100%, and evaluated for possible infection with blood culture, then treated with Ampicillin and Gentamicin injection.

The baby was prepared to get chest tube insertion. Nasogastric tube size 10 was inserted to her right chest into the pleural cavity and the general pressure regulator was set 5 cm H_2O. Chest xray examination was done for evaluation after chest tube insertion. The lung expansion was improved, and ventilator was set PCV setting PEEP 5 PIP 20 rate 60 FiO_2 50%.

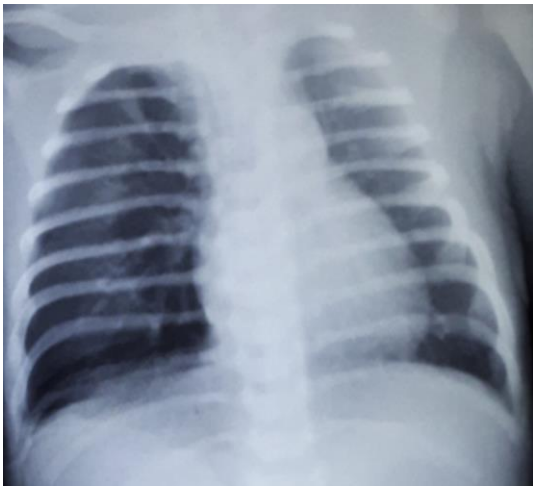

Figure 1. the chest X-Ray of Baby H revealed pneumothorax at right lateral hemithorax

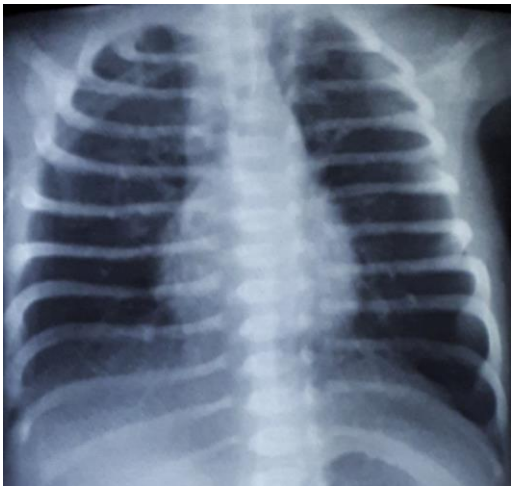

Figure 3. the chest X-Ray evaluation of Baby H showed chest tube inserted to right pleural cavity

On the third day, the baby had general seizure. Hypoxic Ischemic Encephalopaty was suspected as the etiology. The baby was given phenobarbital 40mg as loading dose and continued 5 mg twice daily. Then, she was planned for electroencephalography examination.

On the fourth day, the baby's breathing was improved, with the last FiO2 given was 50%. Echocardioghraphy was performed and revealed severe Tricuspid Regurgitation (maximal PG 63,17) and small secundum Atrial Septal Defect. Pulmonary Hypertension was concluded by that result. Sildenafil 1 mg was given four times daily orally. Inotropic support by dobutamine equal to 5 mcg/kg/minute was also given.

On the fourth day of treatment, fever and dyspnea were subsided, blood culture result was sterile, so both of antibiotic injection were continued. After 7 days antibiotic injection, and insertion of chest tube, the baby got better. The serial thorax x-ray evaluation showed improvement that there was no air leakage in right pleural. After 7 days, the chest tube was taken off.

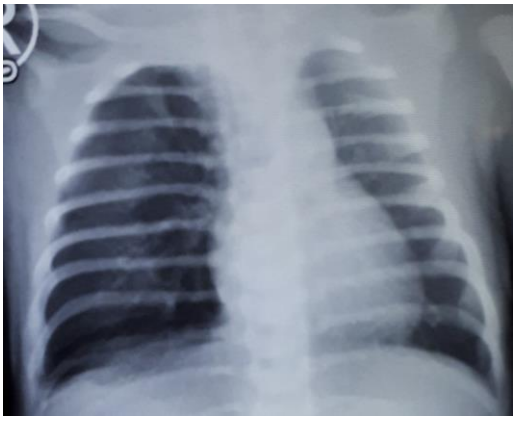

Figure 4. the chest X-Ray evaluation of Baby H showed no pneumothorax

She finally moved to neonatal intermediate ward for further therapy for complication following MAS. She got therapy for Hypoxic Ischemic Encephalopaty, Pulmonary Hypertension and physical rehabilitation for oromotor stimulation. She got phenobarbital 5mg every 12 hours and sildenafil 1mg every 6 hours. But, at the 56th day of observation, the baby was discharged routinely control at neonatology outpatient clinic.

DISCUSSION

Baby H, was born by emergency caesarean section for premature rupture of membrane. The amniotic membranes were ruptured for 3 days prior to delivery, and the amniotic fluid was meconium stained. The baby was not vigorous and still did not have adequate breathing and the heart rate still below 100 beats per minute after doing initial step of resuscitation.

Meconium Aspiration Syndrome can be confirmed as the cause of respiratory distress in this baby as there was history of amniotic membrane rupture 3 days before delivery and meconium stained amniotic fluid at delivery. The other risk factor that caused meconium aspiration in this baby was the history of mother with preeklamsia which lead to intrauterine hypoxia –induced gasping.

Meconium is a complex substance that can be found in the gastrointestinal tracts of fetuses as early as 14–16 weeks of gestation. Although meconium is primarily water (75%), it contains gastric secretions, bile salts, mucus, vernix, lanugo, blood, pancreatic enzymes, free fatty acids, and squamous cells.[10]

In this case, although the assessment of gestational age was 33-34 weeks based on last menstrual period, the physical examination of Ballard score showed the baby is term infant. The presence of meconium in the amniotic fluid at birth is a common event which has been estimated to occur in up to 5% prior to 37 weeks of gestation, 25% of births at term and 23–52% among post-term gestations.[11] Passage of the substance is rare before 37 weeks gestation for several reasons. The terminal cap of the meconium is particularly viscous. Additionally, fetuses do not have strong peristaltic forces that would propel the substance forward. Moreover, the anal sphincter of the fetus is tonically contracted. The passage of meconium may be a response to stresses such as hypoxia, acidemia, or infection. These stresses may cause anal sphincter relaxation and promote intestinal peristalsis. When passage of meconium is noted in premature infants, it is associated with bilious reflux secondary to intestinal obstruction. [10,12]

Meconium aspiration syndrome (MAS) has been defined by clinical criteria: respiratory distress (tachypnoea, retractions or grunting) in a neonate born through meconium-stained amniotic fluid (MSAF); a need for supplemental oxygen to maintain oxygen saturation of haemoglobin (SaO_2) at 92% or more; oxygen requirements starting during the first 2 hours of life and lasting for at least 12 hours and absence of congenital malformations of the airway, lung or heart.[13] In this case, the clinical manifestation of respiratory distress was shown obviously. The radiologic examination also confirm the diagnosis of MAS which revealed patchy infiltrate, and also pneumothorax due to its complication of partial obstruction.

MAS occurs when the fetus passes meconium before birth. Infants born through meconium stained amniotic fluid are at risk for aspiration of meconium in utero or immediately after birth. Aspiration usually occurs in utero as a consequence of hypoxia-induced gasping. Many infants who have MAS are born by cesarean section, indicating that they aspirate meconium before birth. Some aspiration may occur during the second stage of labor, when the shoulders and chest are delivered. [13] MAS is associated with multiple life threatening complications including hypoxic ischemic encephalopathy (HIE) (46%), hypotensive shock (22%), pneumothorax (11.4%), myocardial dysfunction (22%) and pulmonary hypertension (PHN) (17%).[4]

In this case, the baby was suffered from severe MAS based on the condition in need of assisted ventilation more than 48 hours. Newborns exposed to meconium, are more likely to have complications like neonatal sepsis, seizures, neurologic impairment and prolonged NICU stay. The severity of MAS as follows: (1) mild: neonate requires less than 40 % oxygen for less than 48 hour; (2) moderate: neonate requires more than 40 % oxygen for more than 48 hours with no air leak syndromes and (3) severe: neonate requires assisted ventilation for more than 48 hours.[14]

In this case, we did endotracheal suction because the baby was found not vigorous after birth. Endotracheal intubation and suction were performed to remove the meconium in the upper airway before it migrates to the lower airway before multiple breathing. Since 2005, the American Heart Association (AHA) and the

Neonatal Resuscitation Program (NRP) have recommended tracheal suctioning only if the infant is not vigorous, has decreased muscle tone, or has a heart rate less than 100 beats/minute).[15] Starting October 1st, 2016, the practice was changed again to not routinely perform endotracheal suctioning but provide Positive Pressure Ventilation (PPV) if the newborn who was meconium stained and nonvigorous remained apneic or bradycardic after the initial steps of resuscitation were completed. This included oronasopharyngeal suctioning. Endotracheal suctioning was reserved only for those depressed newborns with evidence of meconium obstructing the airway.[16]

The reduction in the incidence of MAS over the last two decades is attributed to the reduction in post-term delivery by elective induction of pregnancies more than 41 week of gestation, aggressive management of fetal distress, and decreased incidence of birth asphyxia. A skilled resuscitation team must be present in all deliveries complicated by MSAF. Elective induction of labor at 41 week of gestation is the most logical step towards prevention of maturation induced intrauterine meconium passage and hence, MAS. Intrapartum amnioinfusion with saline has been used to dilute the thick meconium and this procedure may prevent umbilical cord compression especially in pregnancies complicated by oligohydramnios with MSAF. It may also lead to reduced fetal hypoxia-induced gasping and subsequent meconium aspiration. [17]

Pneumothorax became a complication of MAS in this baby. The mechanism of pneumothorax caused by MAS is because this thick, viscid meconium can result in partial or complete airway obstruction. Partial obstruction causes air trapping and hyper-distension of the distal airway, commonly termed the ballvalve effect. The gas that is trapped may rupture into the pleura resulting in air leaks. [18] The air-leak syndromes include pulmonary interstitial emphysema (air in the tissues of the lungs between the air sacs), pneumopericardium (air in the sac around the heart), and, rarely, pneumoperitoneum (air in the abdominal cavity) and subcutaneous emphysema (air under the skin).[19]

Pneumothorax is an abnormal accumulation of air or gas between the visceral and parietal pleura that subsequently leads to partial or full collapse of the lung. It can be spontaneous or traumatic. A spontaneous pneumothorax is classified as primary or secondary. A primary categorization includes no obvious underlying lung disease, whereas a secondary categorization does include recognizable lung disease.[1]

Physical examination and chest radiograph of the baby was not support the sign of pneumothorax, since we found the breathing sound was symmetric. But chest xray examination of this patient showed lucent area in right lateral hemithorax as a sign of pneumothorax. The clinical presentation may vary from mild or severe signs of respiratory distress to a gradual decline in respiratory function. Patients with a pneumothorax typically present with chest pain, dyspnea, and cough. Vital signs may include tachycardia, tachypnea, hypoxia, and hypotension, particularly in the event of a tension pneumothorax, physical examination can reveal decreased breath sounds on the affected side, hyperresonant percussion, decreased vocal fremitus, decreased chest excursion and the chest xray will show midline pericardial shift to contralateral side. [1] But if the pneumothorax is small enough and not undertension, patients maybe asymptomatic with normal vital signs.

The treatment of neonatal Pneumothoraces depends on the clinical

picture. Small, asymptomatic Pneumothoraces (occupying <15% of hemi-thorax volume) may be left untreated, waiting for spontaneous reabsorption of intrapleural air[1]; Based on the etiology, pneumothorax can be divided into primary pneumothorax which occur in healthy people without underlying disease, and secondary pneumothorax with underlying disease.[1,20] Because this patient suffered from secondary pneumothorax caused by bag valve effect of MAS, drainage of intrapleural air should be done.

In symptomatic cases with cardiorespiratory involvement, drainage is mandatory. If the patient is symptomatic, the pneumothorax is large, or there is underlying lung disease, more invasive treatment may be necessary. In tension pneumothorax, a butterfly needle and syringe can be used to temporarily evacuate free air from the pleural space. This procedure is usually performed at the bedside. Definitive treatment is insertion of an 8 or 10 French chest tube attached for continuous suction. Follow-up auscultation, transillumination and X-ray will confirm that the tube is functioning properly.[5]

Surgical intervention consists of video assisted thoracoscopic surgery (VATS) or open thoracotomy. VATS is increasingly popular, but controversy exists regarding whether it should be performed after the first episode of primary spontaneous pneumothorax or only after recurrence. In the pediatric population, reports indicate recurrence as high as 50% to 60%, but at this point, many recommend VATS only after a second episode. If the pneumothorax is secondary innature, this may be an indication to intervene after the first event because pulmonary function is already jeopardized and a pneumothorax can be even more debilitating. During VATS, both resection of blebs or bullae and pleurodesis should be performed.[1]

SUMMARY

Pneumothorax as complication of Meconium Aspiration Syndrom in newborn have been reported. The early recognition of clinical deterioration during the observation, the baby became more cyanotic and respiratory distress. Although there was no specific clinical finding of pneumothorax, chest X-Ray examination revealed avascular lucent area at right lateral hemithorax. Pleural air drainage is mandatory in case of secondary pneumothorax with cardiorespiratory involvement.

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