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Case Report

Conservative Management in a Ventilated Preterm Neonate with Pneumopericardium, Pleural Effusion, and Pulmonary Collapse: A Case Report

Yazdan Ghandi[1*], Niloofar alsadat Motamedi[1]

1. Amirkabir Hospital, School of Medicine, Department of Pediatrics, Pediatric Cardiology Arak University of Medical Sciences, Arak, Iran

ABSTRACT

Background: Neonatal pneumopericardium (PPC) is a rare clinical condition usually associated with other air leak syndromes. It increases morbidity and mortality due to cardiac tamponade.

Case report: A preterm male neonate weighing 1260 g was born with the gestational age of 28 weeks. Cardiopulmonary resuscitation was routinely performed without any medical therapy. The newborn was transferred to the neonatal intensive care unit due to marked respiratory distress with tachypnea and cyanosis. Pulse oximetry showed oxygen saturation of 70%. A whiteout of the lung and air-bronchogram pattern was found on the chest X-ray. The arterial blood gases demonstrated PH=7.14, PCo_2=51 mmHg, Po2=36 mmHg, bicarbonate=15.8 mg. The neonate was intubated with a tracheal tube size 2.5 and mechanical ventilation was initiated with 90% fraction of inspired oxygen (FiO_2), PIP=14 cmH_2O, and PEEP=4 cmH_2O.

After intubation, the newborn received two doses of surfactant. On the third day, patent ductus arteriosus was established and appropriate treatment was performed. After five days, the chest X-ray was repeated due to increasing respiratory distress and an increasing FiO_2, which revealed a pneumopericardium with right-sided pleural effusion and left upper lobe collapse. Afterwards, pleurocentesis was performed.

Left ventricular ejection fraction (65%) was preserved and no evidence of cardiac tamponade was observed. Serial chest X-ray and echocardiography were performed daily. After the next seven days, the chest radiograph demonstrated a complete resolution of the pneumopericardium, pleural effusion, and lung collapse.

Conclusion: The present case study presented a preterm neonate suffering from pneumopericardium along with pleural effusion and lung collapse. Despite the complexity, the PPC was asymptomatic and eventually resolved without pericardiocentesis.

Keywords: Air leak syndrome, Pneumopericardium, Preterm, Respiratory distress syndrome

Introduction

Pneumopericardium (PPC) is a rare form of air leak syndrome, which is usually associated with other air leak syndromes, such as pneumomediastinum and pneumothorax. It may increase morbidity and mortality via the development of cardiac tamponade. The PPC is a rare clinical condition and in comparison to other air leak syndromes isolated PPC is rare in neonates (1, 2). In premature neonates, the PPC is often observed in the cases suffering from respiratory distress syndrome under mechanical ventilation. In term neonates, it is often associated with a difficult resuscitation, severe respiratory distress syndrome, meconium aspiration syndrome, aggressive mechanical ventilation, or continuous positive airway pressures (3, 4). In the present study, a ventilated preterm neonate suffering from isolated PPC without any concomitant air Leak syndromes was presented that responded to conservative treatment without surgery or pericardiocentesis and eventually it demonstrated a favorable outcome.

Case report

A preterm male neonate weighing 1260 gm was born with the gestational age of 28 weeks to

* *Corresponding author:* Yazdan Ghandi, Amirkabir Hospital, School of Medicine, Department of Pediatrics, Pediatric Cardiology, Arak University of Medical Sciences, Arak, Iran. Tel: 09123833712; Email: y.ghandi@arakmu.ac.ir

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a healthy mother (24-year-old and G1 P0). Membranes ruptured 8 h prior to delivery; however, the amniotic fluid was clear. She had not received any antenatal steroids during the 28 weeks. One and five min Apgar scores were 7 and 8, respectively. Routine resuscitation was immediately performed after birth. Marked respiratory distress with tachypnea and cyanosis was developed; therefore, the newborn was transferred to the neonatal intensive care unit (NICU).

On admission, the neonate's vital parameters demonstrated the heart rate of 110 beats per min and respiratory rate of 80 beats per min. The blood pressure of the studied case was 49/20 mmHg and oxygen saturation was 70%. A whiteout of lung and air-bronchgram pattern was observed on the chest X-ray. The initial arterial blood gases showed PH=7.14, PCo_2=51 mmHg, Po2=36 mmHg, bicarbonate =15.8 mg. Accordingly, the neonate was intubated with an oral/nasal tracheal tube size 2.5 and mechanical ventilation was initiated with 90% fraction of inspired oxygen (FiO_2), PIP= 14 cmH_2O, and PEEP=4 cmH_2O.

Following the intubation, the two doses of surfactant were administered. After initial therapy, oxygen saturation was maintained between 90% and 95%. Peripherally inserted central catheter 2 Fr/30 cm was placed successfully in the right brachiocephalic vein (Figure 1). On the third day, patent ductus arteriosus with a left to right shunt was established on the first echocardiogrphy preformed and appropriate treatment was carried out with ibuprofen/pedea and hydrochlorothiazide without the limitation of serum maintenance.

On the fifth day due to increased respiratory distress and FiO_2, chest X-ray was repeated revealing pneumopericardium, left upper lung collapse, right-sided pleural effusion, and perihilar infiltration with no evidence of other air leak syndromes (Figure 1). Right-sided pleural effusion was confirmed via ultrasound and pleurocentesis was performed using sterile technique. After the diagnosis of the PPC in echocardiography, left ventricular ejection function of 65% was preserved and no evidence was found regarding the distention of inferior vena cava or cardiac tamponade.

High risks of developing cardiac tamponade made continuous hemodynamic monitoring mandatory via pulse oximetry, bihourly automatic oscillatory blood pressure, and arterial blood gases. Serial chest radiographs and echocardiography were performed daily in order to evaluate biventricular cardiac function. Since cardiac tamponade did not occur, the aspiration of the PPC was not attempted. Serial chest radiographs demonstrated complete resolution of the PPC, pleural effusion, and lung collapse over the next seven days (Figure 2).

Oxygen requirement was gradually weaned to room air over 14 days, and supplemental oxygen was discontinued up to the 18th day of life. Cardiovascular examination revealed normal heart sounds, normal peripheral pulses, and no audible murmurs were heard. The use of antibiotics was stopped after 14 days due to negative blood culture results. Eventually respiratory distress improved after two weeks. Breastfeeding was initiated by 14th day and the neonate was discharged on 21st day of life. No

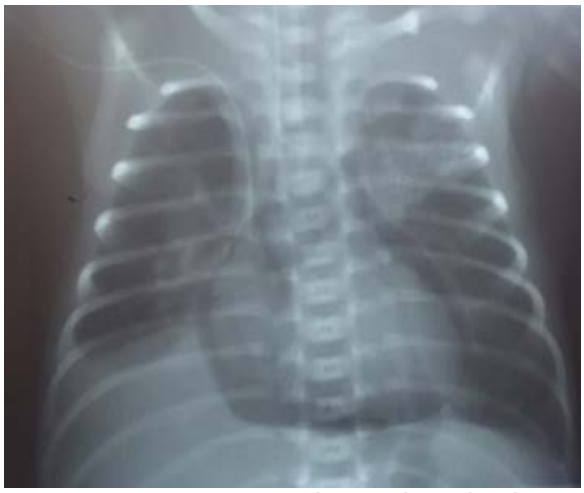

Figure 1. Anteroposterior chest radiograph showing pneumopericardium, plural effusion on the right side, collapse consolidation on the left side, and right perihilar infiltration

Figure 2. Chest radiograph on 7th day showing improvement of pneumopericardium, plural effusion, and pulmonary collapse

recurrence of air leak, cardiac dysfunction, or pulmonary hypertension occurred within the three-month follow-up.

Discussion

The overall incidence of neonatal PPC in very low birth weight (VLBW) ≤1.5kg is 2% and in the ventilated VLBW without surfactant administration is 3.5% (1, 3). According to the literature, the PPC is more common in male neonates (4). The majority of patients are low birth weight (LBW) neonates requiring respiratory support for respiratory distress syndrome. The PPC is usually associated with other air leak syndromes, including pneumothorax, pulmonary interstitial emphysema, pneumomediastinum, subcutaneous emphysema, pneumoperitoneum, and systemic vascular air embolus (4).

Hook et al. demonstrated that the mean age of PPC occurrence was 3.3 days in ventilated VLBW newborns (median: 1.25 days, range: 0-34 days) (4). The present case had multiple risk factors for developing pneumopericardium, including male gender, VLBW, receiving respiratory support at birth for respiratory distress syndrome, and mechanical ventilation with surfactant. The pathophysiology of neonatal pneumopericardium may be air dissected from ruptured alveoli along with the perivascular sheaths to the hilum and mediastinum, which causes air leak into the pericardium atan anatomical site of weakness in the parietal and visceral pericardium near the area of the pulmonary veins (1, 5).

In pneumopericardium, the standard diagnostic method is chest X-ray. The “Halo sign” is the classic finding of the PPC defined as air completely surrounding and outlining the heart but not extending beyond the pericardium the level of the great vessels (1). The clinical manifestations of the PPC depend on the volume of pericardial air leak ranging from asymptomatic to signs of cardiac tamponade. The signs of cardiac tamponade include hypotension, cyanosis, tachycardia, and bradycardia with muffled heart sounds on auscultation.

The PPC is divided into two forms, namely (a) spontaneous PPC occurring in the absence of mechanical ventilation, and (b) the PPC occurring with positive pressure ventilation. The PPC may occur in isolation or in association with other forms of air leak syndromes, such as pneumothorax, pneumomediastinum, or subcutaneous emphysema (6). Pericardiocentesis is mandatory if cardiac tamponade is confirmed. It must be noted that a drainage tube into the pericardial sac may be required in extensive or recurrent cases.

The PPC with concomitant air leak syndromes in ventilated preterm infants has unfortunately demonstrated a poor prognosis (2). Neonates under Mechanical ventilation are particularly at risk for tension pneumopericardium secondary to barotrauma, which is a life threatening condition. In addition, percardiocentesis plays an important role in the management of tension pneumopericardium and must be early performed (7).

In the absence of cardiac tamponade, it is recommended to perform close hemodynamic monitoring without aspiration or surgery. This conservative approach was carried out regarding the management of the mentioned case. Asymptomatic PPC with concomitant pulmonary pathology may resolve with a conservative approach of close hemodynamic monitoring, even after respiratory support for respiratory distress syndrome at birth and mechanical ventilation with surfactant (8).

In recent years, the incidence of air leak syndromes has dramatically decreased due to the antenatal steroid administration, exogenous surfactant therapy, and development of ventilation techniques. The present study reported a case of isolated PPC concomitant with otherpulmonary pathology, right-sided pleural effusion, and left-sidedlung collapse that despite the complexity, improved spontaneously without the need for pericardiocentesis.

This case report highlighted the possibility of close clinical examination and hemodynamic monitoring without pericardiocentesis in the management of ventilated preterm neonates suffering from isolated pneumopericardium. Consequently, conservative management with close monitoring is recommended as a reasonable option for pneumopericardium in asymptomatic ventilated neonates.

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Conflicts of interests

The authors declare that there is no conflict of interest.

References

1. Burt TB, Lester PD. Neonatal pneumopericardium. Radiology. 1982; 142(1):81-4.
2. Suresh P, Tagare A, Kadam S, Vaidya U, Pandit A.

Spontaneous pneumopericardium in a healthy full-term neonate. Indian J Pediatr. 2011; 78(11):1410-1.
3. Hook B, Hack M, Morrison S, Borawski-Clark E, Newman N, Fanroff A. Pneumopericardium in very low birth weight infants. J Perinatol. 1995; 15(1):27-31.
4. Roychoudhury S, Kaur S, Soraisham AS. Neonatal pneumopericardium in a non ventilated term infant: a case report and review of the literature. Case Rep Pediatr. 2017; 2017:3149370.
5. Mansfield P, Graham CB, Beckwith JB, Hall DG, Sauvage LR. Pneumopericardium and pneumomediastinum in infants and children. J Pediatr Surg. 1973; 8(5):691-98.
6. Heckmann M, Lindner W, Pohlandt F. Tensionpneumopericardium in a preterm infant without mechanicalventilation: a rare cause of cardiac arrest. Acta Paediatr. 1998; 87(3):346-8.
7. Karadžić R, Antović A, Ilić G, Kostić-Banović L. Pneumopericardium: a possible rare cause of neonatal death. Med Biol. 2007; 14(2):98-100.
8. Shaireen H, Rabi Y, Metcalfe A, Kamaluddeen M, Amin H, Akierman A, et al. Impact of oxygenconcentration on time to resolution of spontaneous pneumothorax in term infants: a population based cohort study. BMC Pediatr. 2014; 14(1):208.