*CASE REPORT*

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# Pneumopericardium in a preterm infant with severe Respiratory Distress Syndrome

**Joanna Wanat[1,C-D], Wojciech Stanisław Homa[1,C-D] ✉, Piotr Karol Homa[1,C-D], Elżbieta Homa[2,A-C,E-F], Anna Paulina Terpin[2,B,E-F], Andrzej Zawora[2,E-F]**

[1] *Students' Scientific Association at the Department of Paediatric Haematology, Oncology and Transplantation, Medical University, Lublin, Poland*
[2] *Department of Neonatology and Neonatal Intensive Care Unit, Pro-Familia Specialist Hospital,Poland*
*A – Research concept and design, B – Collection and/or assembly of data, C – Data analysis and interpretation, D – Writing the article, E – Critical revision of the article, F – Final approval of the article*

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## Abstract

Neonatal pneumopericardium (NPPC) is a rare clinical condition that typically occurs alongside other air leak syndromes. The key risk factors of its occurrence are advanced lung pathologies treated with high-pressure mechanical ventilation. The most severe form of NPCC is its tension form, which induces cardiac tamponade and may result in sudden cardiac arrest. Surfactant replacement therapy is currently the only pharmacological prophylaxis to prevent NPPC. In patients with a stable clinical condition, high-concentration oxygen therapy is the primary treatment option. If PPC complicates with cardiac tamponade, immediate pericardiocentesis remains the only effective intervention. A case is presented of a premature infant with severe respiratory distress syndrome (RDS) who developed an isolated tension form of PPC, complicated with cardiac tamponade.

## Key words

cardiac tamponade, pneumopericardium, pericardiocentesis, neonate, air leak syndrome

## INTRODUCTION

Neonatal Pneumopericardium (NPPC) is a rare air-leak syndrome characterized by the abnormal presence of air in the pericardial sac. While it may affect children of any age, it is most likely to occur in premature infants, especially those with a low birth weight [1–3]. In 2024, Beaton et al. presented data that indicate that NPPC had the lowest prevalence, at the level of 0.03 per 1,000 neonatal discharges, among all the air leak syndrome subtypes [2]. The same study described an inverse relationship between gestational age and the incidence rate of PPC. The prevalence was the lowest among term neonates, at 0.02 per 1,000 neonatal discharges. Among preterm infants, the frequency increased with the degree of pre-maturity: 0.07 in late preterm, 0.11 in moderate preterm, 0.39 in very preterm, and reaching its peak at 1.37 per 1,000 neonatal discharges in extremely preterm infants [2]. The group at the highest risk of NPPC are premature neonates with pulmonary pathologies, such as respiratory distress syndrome (RDS), meconium aspiration syndrome (MAS), congenital diaphragmatic hernia (CDH) or persistent pulmonary hypertension (PPHN), especially those receiving high positive pressure mechanical ventilation [2, 4, 5]. NPPC is also commonly associated with other air leaks, such as pneumothorax, pulmonary interstitial emphysema, pneumomediastinum, subcutaneous emphysema, pneumoperitoneum, and systemic vascular air embolus [1, 6].

In full-term neonates and older children, PPC is most often a consequence of previous trauma or iatrogenic chest injury, including active resuscitation [1, 3, 7, 8]. However, several reports describe cases of PPC in children without any history of trauma or prior mechanical ventilation. [1, 9, 10]. In 2019, Bonardi et al. collected case reports involving non-traumatic and non-ventilated patients. From among the 50 patients presented in the study, PPC occurred spontaneously in 19 patients [1].

The clinical presentation of the PPC varies from an asymptomatic course to a critical clinical condition with signs of cardiac tamponade, such as muffled heart sounds, hypotension, hypoxemia with cyanosis, tachycardia or bradycardia, and increased central venous pressure [1,8,10,11]. As indicated by Beaton et al. in the aforementioned study, NPPC exhibits the highest mortality rate among all air leak syndromes – 23.6% [2]. During the diagnostic process, PPC should be differentiated from aortic dissection, pericarditis, pneumonitis, medial pneumothorax, and pulmonary embolism, which may give a similar clinical presentation [9].

The therapeutic approach depends on the presence of cardiac tamponade. Asymptomatic and isolated NPPC should resolve spontaneously, requiring only conservative management with close patient monitoring [1, 9, 12, 13]. In stable, full-term infants, high oxygen concentrations may be used in the nitrogen washout process [9]. On the other hand, patients with tension PPC and those who develop symptoms of cardiac tamponade require prompt and aggressive oxygen therapy, pericardiocentesis, or pericardial drainage [3, 9, 14, 15].

The case is presented of a female premature newborn with RDS who developed isolated pneumopericardium, complicated with cardiac tamponade.

✉ Address for correspondence: Wojciech Stanisław Homa, Students' Scientific Association at the Department of Pediatric Hematology, Oncology and Transplantation, Medical University of Lublin, ul. prof. Antoniego Gębali 6, 20-093 Lublin, Poland
E-mail: wojciech.homa2@gmail.com

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## CASE REPORT

A female infant was delivered via caesarean section at 27 weeks of gestational age from the first bigeminal pregnancy complicated with premature rupture of membranes (PROM) and anhydramnios. The child was born as the first twin, weighed 600 grams, and demonstrated features of Intrauterine Growth Retardation (IUGR) and intrauterine constraint. In the first minute of life, the neonate achieved an APGAR score of 4. Immediately after delivery, the infant was intubated and placed on ventilation in SIMV (Synchronized Intermittent Mandatory Ventilation) mode with high parameters and a FiO2 of 1.0. Attempts to reduce the ventilation parameters led to rapid desaturation of the patient. Thoracic ultrasound examination revealed numerous symmetrical atelectasis, which is characteristic of RDS. Due to severe pulmonary symptoms, poractant alfa was administered. However, as the patient's clinical condition showed no improvement, the ventilation protocol was transitioned to high-frequency oscillatory ventilation (HFO). Over the subsequent two days, the patient received two additional doses of poractant alfa. Upon birth, the neonate exhibited signs of haemodynamic instability. Consequently, intensive fluid therapy, norepinephrine infusion, and hydrocortisone administration were initiated to stabilize cardiovascular parameters. Echocardiography revealed a patent ductus arteriosus measuring 1.5 mm with a right-to-left shunt and features of pulmonary hypertension; thus, nitric oxide was introduced to the therapy. Due to a high risk of intrauterine infection and an elevated level of procalcitonin, the patient received empiric broad-spectrum antibiotic therapy. Nutrition was administered parenterally through a central line inserted into the right femoral vein alongside enteral trophic feeding. Ultrasonographic examination revealed no evidence of intracranial haemorrhage or congenital abnormalities.

On the third day of life, the patient's condition began to deteriorate. Despite the use of high ventilation parameters, the patient exhibited desaturation, hypotension, and diminished heart sounds. Thoracic ultrasound excluded the presence of pneumothorax; however, the cardiac silhouette was covered by air signs in the pericardium. A thoracic radiograph confirmed the presence of a 7-mm thick radiolucency in the pericardial sac, which might correspond to pneumopericardium.

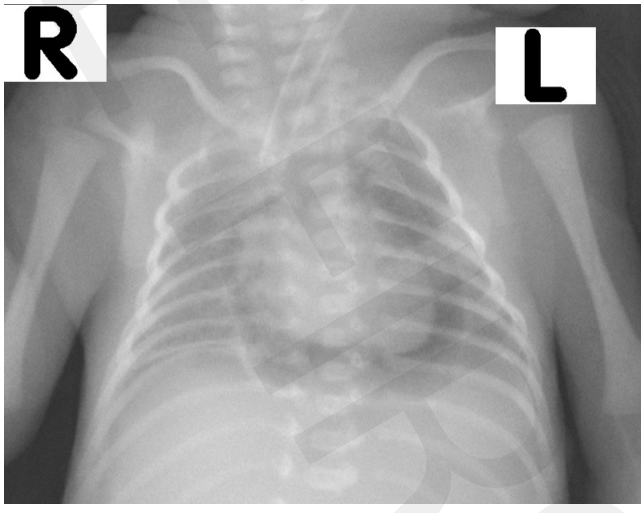


**Figure 1.** The heart surrounded by a rim of gas, a 'halo sign in the pericardium

A pericardiocentesis was performed immediately, resulting in the aspiration of 5 ml of air. Echocardiography confirmed the successful completion of the procedure; however, it did not result in an improvement in the infant's condition. Additional fluid resuscitation, an increased dosage of norepinephrine, and an infusion of adrenaline were administered. Due to severe acidosis, sodium bicarbonate was added to the therapy. Despite this, the patient exhibited bradycardia (<60 beats/min), accompanied by further declines in oxygen saturation and arterial pressure. Cardiopulmonary resuscitation (CPR) was initiated; however, the return of spontaneous circulation was not achieved, resulting in the patient's death.

## DISCUSSION

Clinically, PPC is categorized into non-tension and tension forms. Data presented in 2019 by Bonardi et al. indicate that tension PPC in paediatric patients is associated with a significantly worse prognosis, with a higher mortality rate compared to the non-tension one (28.6% vs 8.3%) [1]. The prognosis of the patient in the presented case was unfavourable. She presented a severely compromised clinical state, characterized by prematurity, extremely low birth weight, and RDS, and subsequently developed a tension form of PPC.

Despite adherence to the RDS management recommendations, which included immediate intubation with mechanical ventilation, administration of caffeine, hydrocortisone, and thrice-delivered surfactant, satisfactory oxygen saturation levels and parameters of arterial blood gas analysis were not achieved [16]. The patient's condition was further complicated by pulmonary hypertension and an unstable haemodynamic profile, requiring the persistent use of nitric oxide and vasopressors [16]. Moreover, different mechanical ventilation modes were applied, including SIMV and HFO. HFO is associated with effective ventilation achieved through high respiratory rates and low tidal volumes, along with reduced airway pressures and decreased oxygen leakage compared to conventional mechanical ventilation [16–18]. However, the meta-analyses did not indicate a significant difference in mortality rates between neonates with severe ARDS treated with HFO and those who received conventional mechanical ventilation [19, 20]. All of the above-mentioned efforts did not enable a reduction of high ventilation settings, which may have contributed to the development of PPC [2, 21].

The presence of air in the pericardial sac in non-traumatic patients is associated with pulmonary rupture. Theories distinguish two crucial factors: alveolar over-distention and increased airway pressure, which are observed in patients who require high peak inspiratory pressures or positive end-expiratory pressures [9, 20, 22]. Following the rupture, air accumulates in the interstitial tissue, then propagates to the hilum, from where it can enter the pericardial space at the junction where the parietal pericardium reflects onto the visceral pericardium, typically near the area of the pulmonary veins [10, 22].

Prompt identification of NPPC is pivotal for selecting appropriate therapeutic interventions and improving patient outcomes [9]. Echocardiography may reveal the presence of air in the pericardial sac when the heart is not visualized in a subxiphoid echocardiographic window [9]. A certain

diagnosis can be achieved through findings in thoracic radiography, including the 'halo' sign, which outlines the heart and may even extend above the great vessels [9, 14]. The same image, corresponding with descriptions from literature, was captured in the chest radiograph of the presented patient. This finding enabled prompt echocardiography-guided pericardiocentesis, the recommended course of treatment [15, 23]. An alternative therapeutic approach would have involved continuous drainage through a pericardial catheter [14, 23, 24]. Pericardiocentesis facilitates the rapid evacuation of air from the pericardial sac, simultaneously confirming the diagnosis. It should be performed in cases of sudden clinical deterioration. In contrast, the placement of a drainage catheter allows for continuous air evacuation and is typically employed in extensive or recurrent cases. The literature supports the efficacy of both methods, without implying a superiority of one over the other [1, 5, 8, 10, 24].

## CONCLUSIONS

This report emphasizes the importance of maintaining vigilance and considering rare air leak syndromes in neonates with severe pulmonary pathologies undergoing mechanical ventilation. It also highlights that, despite prompt initiation of therapeutic interventions, reversal of the PPC complications may not always be achievable.

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