#### Case Report

# Isolated Pneumopericardium in a Neonate: A Case Report

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# **ABSTRACT**

Pneumopericardium (PPC) is a rare but life-threatening condition that mostly accompanies air leak syndromes in neonates receiving positive pressure ventilation. PPC results from the rupture of the alveoli and subsequent dissection of air through the tissue planes into the mediastinum, pleura, or pericardial sac. This report describes the case of a term low birth weight neonate born in a peripheral hospital who developed respiratory distress soon after birth. He was transported with manual bag and tube ventilation, and was found to have air leak syndrome in the form of isolated PPC. The neonate was managed conservatively with spontaneous resolution of the PPC.

# **CASE REPORT**

A term low birth weight neonate, vaginally delivered at 40 weeks of gestation with a birth weight of 2050 grams, presented at 24 hours of age in the neonatal emergency with respiratory distress since birth. The neonate was born to a primiparous mother with regular antepartum follow-up and normal antenatal ultrasonography scans. The neonate had cried immediately after birth, but developed fast and laboured breathing associated with cyanosis and was put on oxygen support and later intubated and referred from the delivery centre. He was transported in a private ambulance on intermittent manual bag and tube ventilation on the way.

On arrival, the neonatal Emergency Endotracheal Tube (ETT) was found to be dislodged, the temperature was 36°C, there were minimal respiratory efforts, heart rate was <60/minute, capillary refill time was delayed, the neonate was cyanosed and there was no spontaneous activity. He was resuscitated, ETT was changed, fluid bolus (normal saline 10 mL/kg) and inotropes (Dopamine 5  $\mu$ g/kg/min and dobutamine 10  $\mu$ g/kg/min was started). Arterial blood gas was suggestive of severe respiratory and metabolic acidosis (pH 6.80/pCO<sub>2</sub> 72/pO<sub>2</sub> 17/Lactate 11.6/HCO<sub>3</sub> uncalculated). The chest radiograph was suggestive of hyaline membrane disease (white out lung) and isolated PPC with no evidence of pneumothorax [Table/Fig-1].



[Table/Fig-1]: Chest X-ray showing air in the pericardial sac (Halo Sign), with feature of hyaline membrane disease.

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The neonate did not show any features of cardiac tamponade, an informed decision of close observation and conservative management was made. Surfactant (Survanta 4 mL/kg) was administered and after stabilisation of haemodynamic parameters he was shifted to the neonatal intensive care unit. He was put on mechanical ventilation blood gases were normalised after six hours of admission and a repeat chest X-ray after 12 hours of admission showed no evidence of PPC or other air leaks [Table/Fig-2]. The neonate died on day three of admission due to severe Persistent Pulmonary Hypertension (PPHN) and cardiogenic shock.



[Table/Fig-2]: Chest X-ray showing resolution of pneumopericardium with improvement in hyaline membrane disease at 6 hours of admission.

## DISCUSSION

The PPC is a collection of air in the pericardial sac. It is mostly associated with pneumothorax and pneumomediastinum, and isolated PPC is rare in neonates [1]. This condition has the potential to cause cardiac tamponade and may be fatal if not managed promptly. PPC is usually associated with air leak syndromes associated with positive pressure ventilation, and air leak is thought to occur owing to the increased intra-alveolar air resulting in migration of gas into the mediastinum, pleura, and pericardium [2]. Alveolar rupture releases air in the interstitial spaces which further dissects into connective tissue sheaths and results in pneumomediastinum, pneumothorax, and PPC. PPC is thought to occur through a potential weak area of pericardial reflection over the pulmonary veins [3]. Neonates with hyaline membrane disease are particularly susceptible to developing air leaks with increased chances if they require respiratory support [4]. Treatment options for PPC include observation, needle pericardiocentesis, and continuous pericardial drainage depending upon the presence or absence of symptoms of cardiac tamponade [5].

The PPC may present clinically as acute onset of haemodynamic compromise, bradycardia, cyanosis, poor perfusion, hypotension, and respiratory distress [6,7]. A case report by Unkar ZA et al., describing a preterm neonate with Respiratory Distress Syndrome who developed sudden hypotension and cyanosis after 18 hours of stable course was found to have PPC [7]. Case report by Haley A et al., similar clinical deterioration was found to be associated with the detection of PPC [8]. This cardiovascular compromise at times may be unresponsive to oxygen administration, ventilatory adjustments, catecholamines, and cardiopulmonary resuscitation [1]. In the index case, the neonate presented with cardiorespiratory compromise but responded well to the initial resuscitative measures, and the treating team was able to stabilise the neonate in the paediatric emergency.

Definitive diagnosis can most often be confirmed by chest X-ray where air can be seen surrounding the heart including the inferior surface which has been described as the halo sign by Burt and Lester [9]. Most of the case reports found the presence of PPC with the halo sign in the radiograph [7,8]. But the severity of the condition and rapid progression may often prohibit adequate diagnostic procedures [10]. Ecocardiography may be non conclusive as reported by Unkar ZA et al., due to the presence of poor acoustic window because of the presence of air [7]. In this case, PPC was diagnosed based on the halo sign.

The distinctive feature in this case was the neonate being brought to emergency with bag and tube ventilation which is a common occurrence in the Indian scenario which many times results in air leaks most commonly pneumothorax. But isolated PPC has been less reported in this clinical scenario.

Treatment depends on symptomatology. In asymptomatic neonates, close observation and ventilatory pressure adjustments if associated with other air leak syndromes are usually recommended. As in the case reports most of the times isolated PPC resolves spontaneously. Even in complex cases, a spontaneous resolution is possible as reported by Haley A et al., [8]. Spontaneous resolution without intervention [4] or with nitrogen washout using oxygen by hood has also been reported [8]. In symptomatic cases immediate drainage by pericardial tap followed by continuous pericardial drainage in persistent cases can be done [5,11]. Considering the index neonate responded well to the initial resuscitative efforts, management was essentially conservative and a repeat chest X-ray after 12 hours

showed spontaneous resolution of the PPC. Provision of adequate positive pressure ventilation may have aided in the resolution of PPC as previously reported by Tani M et al., [11]. In the case of tension PPC, the mortality range is 80-100 percent, often the underlying condition precipitating the PPC is the cause of death [12]. The prognosis depends on the underlying disease as the underlying disease may progress even after the resolution of PPC as reported in various case reports. The index patient died after two days of treatment due to PPHN and cardiogenic shock even after the resolution of PPC. In this case, an echocardiography could not be done for decision-making as it was not available at the point of care, and complete resolution of PPC was made based on radiographs only.

## **CONCLUSION(S)**

This case emphasises the fact that air leak syndromes should always be suspected in any neonate presenting with respiratory distress with prior history of receiving positive pressure ventilation. This condition assumes more significance when the neonate is transported on a manual bag and mask or bag and tube ventilation. Air leaks can also be present as isolated PPC and management can be done conservatively if the neonate is haemodynamically stable. Neonatal healthcare providers should be aware of the condition and trained and skilled in prompt pericardiocentesis if required.

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