

Transient tachypnea of the newborn following vaginal delivery in a primigravida: A case report managed with low-flow oxygen via concentrator

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ABSTRACT

Transient tachypnea of the newborn (TTN) is a self-limiting respiratory condition typically associated with delayed clearance of fetal lung fluid. It is more commonly reported after cesarean delivery; however, it may occasionally occur following an uncomplicated vaginal birth. We report a term neonate born to a primigravida who developed tachypnea shortly after birth despite stable oxygen saturation on room air. The infant was managed with low-flow oxygen delivered through a concentrator and supportive care, resulting in gradual clinical improvement. Sepsis and congenital pneumonia were considered and excluded through clinical and laboratory evaluation. Arterial blood gas analysis revealed mild respiratory alkalosis. The neonate recovered completely and remained well on follow-up.

Key words: Neonatal respiratory distress, Oxygen therapy, Term neonate, Transient tachypnea of newborn, Vaginal delivery

Transient tachypnea of the newborn (TTN) is one of the most common causes of early neonatal respiratory distress, particularly in term and late preterm infants. It is characterized by tachypnea developing within the first few hours of life due to delayed clearance of fetal lung fluid, leading to reduced pulmonary compliance and mild impairment of gas exchange [1]. The transition from fetal to neonatal life requires rapid absorption of lung fluid through epithelial sodium channels and pulmonary lymphatics. Failure of this process results in persistence of interstitial fluid and clinical manifestations of respiratory distress [2]. Several risk factors have been associated with TTN, including cesarean delivery without labor, prematurity, maternal diabetes, and male sex. Among these, cesarean section is the most significant due to a lack of thoracic compression and reduced catecholamine-mediated fluid clearance [3]. Although TTN is commonly linked with cesarean delivery, it can also occur following vaginal birth. This suggests that additional mechanisms, such as delayed epithelial sodium channel activation or subtle perinatal influences, may contribute to its development [4]. Clinically, TTN presents with

tachypnea, mild retractions, nasal flaring, and occasional grunting, usually without severe hypoxemia. Chest radiography typically shows prominent pulmonary vascular markings, fluid in interlobar fissures, and mild hyperinflation [5]. Management of TTN is primarily supportive, with oxygen therapy and monitoring as needed. Most cases resolve within 48–72 h; however, diagnostic uncertainty may lead to unnecessary antibiotic use and prolonged hospitalization [6].

CASE REPORT

A male neonate was born at 38+1 weeks of gestation to a primigravida mother via spontaneous vaginal delivery. The pregnancy was uneventful. Birth weight was 2.5 kg. Based on gestational age and weight, the neonate was appropriate for gestational age. The infant cried immediately after birth with Apgar scores of 8 and 9 at 1 and 5 min, respectively. Within 2 h of life, the neonate developed tachypnea with a respiratory rate of 70 breaths/min, without cyanosis or grunting. Oxygen saturation remained between 92% and 94% on room air. Despite acceptable saturation, oxygen therapy was initiated via low-flow nasal prongs (1 L/min) due to persistent tachypnea and mild chest retractions, indicating increased work of breathing.

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Figure 1: Chest X-ray showed prominent pulmonary vascular markings with fluid in interlobar fissures, suggestive of TTN

On examination, heart rate was 148/min, respiratory rate was 70/min, and oxygen saturation was 93% on room air. Mild subcostal retractions were present.

The sepsis screen was negative. C-reactive protein was within normal limits. The procalcitonin test was not performed due to low clinical suspicion and a normal sepsis screen. Chest X-ray showed prominent pulmonary vascular markings with fluid in interlobar fissures, suggestive of TTN (Fig. 1). Arterial blood gas analysis showed pH: 7.47, $p\text{CO}_2$: 32 mmHg, $p\text{O}_2$: 78 mmHg, HCO_3^- : 23 mEq/L, consistent with mild respiratory alkalosis. Blood culture showed no growth after 48 h.

The neonate was managed with low-flow oxygen via concentrator, maintenance fluids, and close monitoring. Respiratory distress gradually improved over 48 h. Oxygen was weaned and discontinued by day 3 of life. The infant was followed up at 2 weeks of age and was asymptomatic with normal feeding and activity.

DISCUSSION

Transient tachypnea of the newborn remains the most frequent cause of respiratory distress in term neonates, but its presentation can vary. In this case, the occurrence after vaginal delivery without classical risk factors highlights the variability in its clinical profile [7].

The underlying pathophysiology involves delayed resorption of fetal lung fluid, resulting in decreased lung compliance and compensatory tachypnea. Hormonal and molecular mechanisms, including impaired epithelial sodium channel function, play a critical role in this process [1]. While cesarean delivery is a well-known risk factor, studies have shown that TTN can occur in vaginally delivered neonates, emphasizing that mode of delivery alone does not exclude the diagnosis [4]. An important clinical consideration in this case was the initiation of oxygen therapy despite oxygen saturation above 92% on room air. Current evidence supports that oxygen supplementation should be guided by overall respiratory distress rather than saturation alone [8]. Differentiation from neonatal sepsis and congenital

pneumonia is essential. In this case, a negative sepsis screen, normal inflammatory markers, and absence of radiological consolidation supported the diagnosis of TTN and avoided unnecessary escalation of therapy [9]. Recent advances, such as lung ultrasound, have improved diagnostic accuracy and may help distinguish TTN from other causes of neonatal respiratory distress without radiation exposure [10]. Although TTN is generally considered benign, emerging evidence suggests a possible association with later respiratory morbidity, including wheezing and bronchiolitis, indicating the need for follow-up.

CONCLUSION

This case highlights that transient tachypnea of the newborn can occur even after an uncomplicated vaginal delivery. Clinical judgment should guide oxygen therapy, not saturation alone. Prompt recognition, careful exclusion of sepsis, and supportive management ensure favorable outcomes while avoiding unnecessary interventions and antibiotic use in otherwise stable neonates.

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