Content available at: https://www.ipinnovative.com/open-access-journals

IP Journal of Paediatrics and Nursing Science

Journal homepage: https://www.jpns.in/

Case Report

Preterm baby with different challenges in resource limited setting

Kamaldeep[1]*

[1]Dept. of Pediatrics, District Hospital, Gurdaspur, Punjab, India

Abstract

A preterm baby with POG 35 weeks weight 2.6kg delivered at Civil Hospital, Gurdaspur by LSCS to a 30 years old mother (Gravida 3, Abortions 2) with H/O leaking PV for more than 24 hours. Indication of LSCS was foetal distress, bad obstetric history. Antenatal USG of mother at 20 weeks POG shown anomalous origin of right subclavian artery going behind the trachea. Baby cried immediately after birth with APGAR score 9/9 at 1/5 minutes of birth, but soon developed RDS. Baby was shifted to SNCU, put on CPAP, maintenance IV fluids and antibiotics started. After 6 hours baby developed signs of fluid overload. RBS at 6 hours of birth was 152mg/dl. Fluid restriction was done. RBS was 138mg/dl at 12 hours of birth. So D5% was started.Septic screen was positive. Baby was shifted from CPAP to nasal prongs for oxygen therapy at 24 hours of life, which was continued for 6 hours and stopped. RBS was 106mg/dl, 79mg/dl on further monitoring. On 3rd DOL baby had GI bleed about 2ml fresh. Injection Vit K 2mg was given iv stat. IV antibiotics were changed to Piperacillin/Tazobactum. When no episode of GI bleed was present for about 6 hours EBM was given to the baby. To our surprise breast milk was of chocolate color. There were no cracks on mother's nipples. Color of milk was same for 3 expressions but we continued mother's feed. On 4th DOL on CVS examination baby grade 3 systolic murmur was heard on 2nd/3rd ICS, left parasternal area which was not audible earlier. CXR was WNL.2D Echo shown dilated right atrium, patent foramen ovale and tricuspid regurgitation. Mother's milk got cleared after 3 days. Baby was discharged after 7 days on furosemide drops.

Keywords: Chocolate coloured breast milk, Rusty Pipe Syndrome, Anomalous origin of right Subclavian Artery, CPAP, hyperglycemia, Necrotising enterocolitis, respiratory distress.

Received: 12-04-2025; **Accepted:** 31-05-2025; **Available Online**: 27-06-2025

1. Introduction

Infants born before 37 weeks from the first day of LMP are termed premature by WHO. The American College of Obstetricians and Gynaecologists defines Late preterm infants as those born between 34 and 36 6/7 weeks. These infants have significantly increased morbidity and mortality. There is increased incidence of congenital anomalies, an increased risk of requiring resustitation, increased incidence of hypoglycaemia, respiratory distress, apnoea, feeding difficulties and jaundice.[1]

These complications sometimes are difficult to manage in resource limited settings.

Here I am reporting a case of late preterm baby who developed many complications during hospital stay.

2. Case Presentation

A preterm male baby born to a 30 years old mother with birth weight 2.6 kg at gestation 35weeks by LSCS. Indication for LSCS was preterm labour and leaking per vaginum for 24 hours. Maternal history: $G_3P_1A_2L_0$, euthyroid, no GDM, no PIH, no infections, Amniotic fluid adequate. Foetal Echo at 18 weeks of gestation showed anomalous origin of right subclavian artery going behind the trachea, normal cardiac chambers dimensions, valves and intracardiac velocities. H/O Leaking PV for >24hours present. Two doses of injection betamethasone were given prior to LSCS. At the time of birth APGAR was 9/9. O/E HR-150/Min, RR-68/Min, CRT-<3Sec, Chest –vesicular breathing, clear, no indrawing, AE equal on both sides. CVS-S1S2 N, no murmur heard. P/A – Soft, $Liver_1$cm, Spleen not palpable, genitalia normal, CNS: baby was active, PNRs present. Baby was shifted to SNCU immediately after birth. Oxygen inhalation started @ 1litres

*Corresponding author: Kamaldeep
Email: deepkamal4242@gmail.com

https://doi.org/10.18231/j.ijpns.2025.012

per minute. Few minutes later baby started grunting. His DOWNES Score was 5. So baby was shifted to CPAP. SPO2 was 92% with PEEP 6cm of H2O. CVS – S_1S_2 present, no murmur. HR was 150/Min.inj vit k, I/V Cefotaxime and inj Amikacin were given. I/V D10 was started @ 80ml/ kg/ day. After 2hours RBS was 131mg/dl and at 6hours 152mg/dl. At 6 hours of life, baby had periorbital puffiness, so rate of fluid administration was reduced to 2/3rd of maintenance and changed to D5%. INJ furosemide was given I/V stat. RBS values were WNL after that.

Investigations: Hb 13.4g/dl, TLC:20160/mm^3, P:62,L36, ANC:16520, platelets:3,08,000, Serum Calcium: 9.2, B.Urea; 34.1mg/dl, S.Cr.1.0,CRP : negative, Blood culture: no growth Chest X Ray WNL. After 24 hrs baby was shifted from CPAP to O_2 @1L/Min with nasal prongs and O_2omitted after 12hrs. Baby had one episode of GI Bleed on DOL-2, Inj vit k repeated, inj piperacillin and tazobactum started. On DOL3 spoon feed was planned with EBM. When mothers milk was brought it was chocolaty in colour. We saw it for first time. There were no cracks or lesions on mother`s breast. After going through literature and ruling out hepatitis B, hepatitis C and HIV in mother we continued breast milk to the baby initially with spoon for one day. On DOL 4 on CVS examination grade 3 systolic murmur was heard in 2nd and 3rd intercostals space left parasternal area. We omitted fluids, kept baby on breast feed, completed i/v antibiotics and got an echo done. 2D Echo shown dilated right atrium, patent foramen ovale and tricuspid regurgitation. As vitals of baby were normal, baby was clinically stable and accepting breast feed, baby was discharged after 7days and referred to Cardiology Department, GMC Amritsar for further management. He was started on furosemide drops. On follow up after one week baby was gaining weight and was active.

Cardiologist there advised same treatment and follow up after 3 months.

Final Diagnosis: Preterm 35weeks, 2.6 kg AGA baby with congenital heart disease with treated respiratory distresss, hyperglycemia, suspect stage NEC and probable sepsis

3. Discussion

Most commonly late preterm newborns develop hypoglycemia, but this baby developed hyperglycemia during first 2 days of life which could be due to foetal distress. Neonatal hyperglycemia is usually defined as serum glucose greater than 150 mg/dl (8.3 mmol/L) or whole blood glucose greater than 125 mg/dl (6.9 mmol/L), irrespective of gestational or postmenstrual age. Usually, the safe target for a neonate's blood glucose level is 70 to 150 mg/dl.[2] The cut-off for a safe target is based on the renal glucose threshold of preterm neonates. Hyperglycemia can be due to increased insulin resistance, inadequate insulin secretion and inability to suppress glucose production in the liver.[3] Sepsis and necrotizing enterocolitis can be considered if hyperglycemia develops without a change in glucose infusion rate. Neonatal respiratory distress syndrome, or RDS, is a common cause of respiratory distress in a newborn, presenting within hours after birth, most often immediately after delivery. RDS primarily affects preterm neonates, with more severe disease in the smaller and more premature neonatess. There are numerous causes of neonatal respiratory distress, including transient tachypnea of the newborn, pulmonary air leak disorders (pneumothorax, pneumomediastinum), neonatal pneumonia, meconium aspiration, persistent pulmonary hypertension of the newborn, and the broad categories of cyanotic congenital heart disease and interstitial lung disease.[4]

Antenatal administration of glucocorticoids to women at risk for preterm delivery reduces the incidence and severity of RDS as well as the rate of mortality. Soon after birth, preterm infants with RDS develop rapid breathing, grunting, poor color, and crackling or diminished breath sounds breathing requires increased work. RDS is an acute illness treated with respiratory support (oxygen, positive airway pressure, ventilator, or surfactant) as needed and improves in 2 to 4 days and resolves in 7 to 14 days. The current preferred strategy is the early initiation of continuous positive airway pressure (CPAP) with selective surfactant administration.[5] This baby also had respiratory distress which settled within two days with CPAP and oxygen through nasal prongs. Feeding intolerance is also a common complication of preterm birth. The immature GI tract has difficulty in digesting food necessary for ongoing growth and development. Necrotizing enterocolitis (NEC) is an acute injury of the small or large intestines that causes inflammation and injury to the bowel lining and that primarily affects preterm infants. Incidence worldwide varies between 0.3 to 2.4 infants per 1000 live births. Nearly 70% of these cases occur in premature infants born before 36 weeks gestation. Necrotizing enterocolitis affects 2% to 5% of all premature infants and is responsible for nearly 8% of all NICU admissions.[6] It typically occurs within 2 weeks of birth and presents as feeding difficulties, abdominal swelling, hypotension, and other signs of sepsis. When NEC is suspected, infants are treated with antibiotics and bowel rest (i.e., no feedings).

Other uncommon problem was chocolate coloured breast milk. Coffee/chocolate brown coloured breastmilk may mean there is some blood. The most common cause of blood in breastmilk is a cracked nipple. A less common condition is an intraductal papilloma — a small benign wart-like growth on the lining of a milk duct, which bleeds. It is common to have blood-stained colostrum or milk in the first days after giving birth. This is referred to as 'rusty pipe syndrome'. Swallowed blood does not harm the baby but tends to irritate the stomach and baby may vomit it. The rusty color is usually due to mixing of colostrum, or first breast milk, with a small amount of blood.[7] Rusty Pipe Syndrome is characterised by painless bloody nipple discharge with no

visible evidence of mechanical injuries within the breast. It could be due to structure of blood vessels and changes that occur during stage I and II of lactogenesis. It resolves spontaneously within 5-7 days.[8]

4. Conclusion

Above case was a different case in our resource limited setting, which gave us new challenges daily and managed well.

5. Conflict of Interest

None.

6. Source of Funding

None.

References

1. Jennifer M. Brady and Brenda B. Poindexter. Moderate and Late Preterm Infant. Nelson Textbook of paediatrics,21 edition©2020.Page 902,906part XI
2. Şimşek DG, Ecevit A, Hatipoğlu N, Çoban A, Arısoy AE, Baş F, et al. Neonatal Hyperglycemia, which threshold value, diagnostic approach and treatment?: Turkish Neonatal and Pediatric Endocrinology and Diabetes Societies consensus report. *Turk Pediatri Ars*. 2018;53(Suppl 1):S234–8.
3. Hay WW, Rozance PJ. Increased insulin resistance. Neonatal Hyperglycemia-Causes, Treatments, and Cautions. *J Pediatr*. 2018;200:6-8.
4. Reuter S, Moser C, Baack M. Respiratory distress in the newborn. *Pediatr Rev.* 2014;35(10):417–28.
5. American Academy of Pediatrics Steering Committee on Quality Improvement and Management. Classifying recommendations for clinical practice guidelines. *Pediatrics*. 2004;114(3):874–7.
6. Jacob G. Ginglen; Nikolai Butki. Necrotizing Enterocolitis. Treasure Island (FL): StatPearls Publishing; 2025
7. Çintesun E, Gul A, Akar S, Ezveci H, Celik C. Rusty pipe syndrome. *Perinatoloji Dergisi.* 2017;25:85–6.
8. Wszolek KM, Nowek A, Odor A, Piet M, Wilczak M. Rusty pipe syndrome. Safety of breastfeeding. *Ginekol Pol*. 2021;92(12):902–4.

Cite this article: Kamaldeep Preterm baby with different challenges in resource limited setting. *J Paediatr Nurs Sci*. 2025;8(2):56-58.