

## Persistent Pulmonary Hypertension of the Newborn Secondary to Meconium Aspiration

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**A**bstract  
Persistent pulmonary hypertension of the newborn is the result of elevated pulmonary vascular resistance to the point that venous blood is diverted to some degree through fetal channels (i.e.) the ductus arteriosus & foramen ovale.

We presented here a 36 hrs old neonate who is delivered by complicated vaginal delivery with history of prolonged labor & meconium leaking liquor, cyanosis, tachypnea, grunting, poor feeding and irritability. Body weight was 5 Kg, head circumference 36.5cm, length 50 cm, respiratory rate 85 / min, heart rate 130 / min., temp 37.5 C and O<sub>2</sub> saturation was 77 %.

Physical examination showed macrocosmic meconium stained neonate with suffused face, chest examination revealed bilateral fine crepitations with poor air entry.

### Investigations revealed

pcv = 65%, B. urea = 25, CRP = positive, T.S.B = 6.5mg/dl, RBS = 75 mg/dl, Blood group O+, CXR revealed C/T ratio 70 % while echo showed cardiomegaly with tricuspid valve regurgitation (functional).

The baby treated with O<sub>2</sub>, i.v. fluid, antibiotics, frequent suction, anticonvulsant & mannitol.

On the 5th day of admission, the baby became better with no need for O<sub>2</sub>. New CXR revealed C/T ratio of 50 %. Echo done on 8th day of treatment which revealed completely normal heart.

### Introduction

Persistent pulmonary hypertension of newborn (PPHN), first described in 1969, remains a

challenging condition with high mortality & morbidity in the neonatal intensive care unit (NICU). The incidence of PPHN has been reported to be 1-5 / 1000 live birth in US. It accounts for about 3-4 % of NICU admissions & yet has been reported to be the commonest cause of death in normally formed infants above 1000 gm.

PPHN is a cardio-pulmonary disorder characterized by systemic arterial hypoxemia secondary to elevated pulmonary vascular resistance. This disorder is classified into

- 1- PPHN associated with pulmonary parenchymal disease like meconium aspiration, hyaline membrane disease, or transient tachypnea of newborn as the cause of alveolar hypoxia, this type known as secondary PPHN or appropriate PPHN.
- 2- PPHN with radiographically normal lung and no evidence of parenchymal disease, this frequently called persistent fetal circulation (PFC) or primary or inappropriate PPHN.
- 3- PPHN associated with hypoplasia of lungs most often in the form of diaphragmatic hernia.

The specific abnormality which produces persistent hypertension is not known, possible pathogenic mechanisms include:-

- 1- Repeated intrauterine closure of the ductus with redirection of blood flow into the high resistant fetal pulmonary vasculature. This may occur in mother who takes high dose of Aspirin<sup>(1)</sup>.
- 2- Abnormal response of the pulmonary vasculature to hypoxia with an inability to relax after the stimulus for vasoconstriction is removed i.e. birth asphyxia.

- 3– Repeated intrauterine hypoxia which stimulate the hypertrophy of medial smooth muscle which surrounds pulmonary arterioles , enabling vessels to constrict to an extreme degree for long period of time . There is a pathological evidence that babies with PPHN have greater thickness of medial muscle in pulmonary arteries than in normal full term infant ( 2 - 5 ) .
- 4 – Regional alveolar hypoxia due to poor ventilation which is not apparent radiographically.
- 5 – Undergrowth of pulmonary vascular tree, this frequently is the cause in infant with congenital diaphragmatic hernia .
- 6 – Alteration in vasoactive mediators level.
- 7- Micro thrombus formation in the pulmonary vascular bed. Thrombocytopenia and pathological evidence of platelet –fibrin micro thromboembolism has been reported

in infant succumbing to PPHN <sup>(6,7)</sup> . These micro thrombi accompanying released mediator may induce pulmonary hypertension.

PPHN is most often associated with prenatal asphyxia in 50-70% of reported cases ,other conditions associated with this syndrome include hypoglycemia,hypocalcemia,hyperviscosity syndrome and sepsis.

### Case report

We presented here a 36 hrs old neonate ,admitted to NICU in Karbala teaching hospital for children ,at the second of October 2007 ,with history of complicated vaginal delivery , meconium stained leaking liquor. cyanosis, poor feeding, tachypnea, grunting and irritability. (Figure 1)

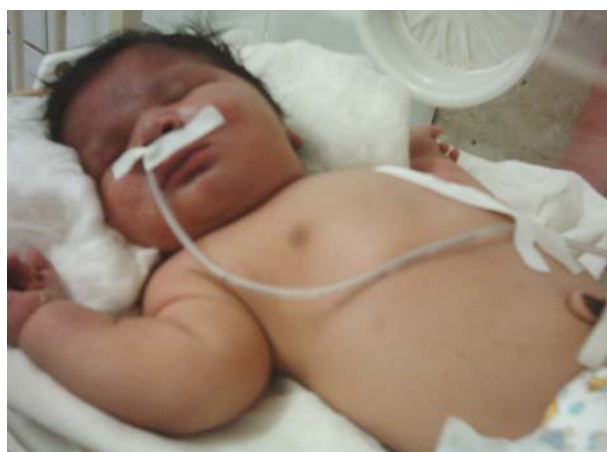


Figure 1 Thirty six hours neonate with PPHN

### On examination

Body weight was 5kg ,heart rate (H.R ) 130 beat/min ,respiratory rate ( R.R) 85 breath/min, Temp 37.5 C, head circumference (H.C.) 36.5 cm and O<sub>2</sub> saturation was 77%.

Chest examination showed poor air entry with bilateral fine crepitations, on auscultation of the heart , normal 1st heart sound , normal second heart sound with ejection systolic murmur grade II / VI at LSB.

Three hours after admission to NICU, the baby developed generalized tonic clonic seizure aborted by luminal . Investigations results were RBS =75 mg / dl , CRP = + ve , PCV =65%, B. urea = 25mg/dl, Blood culture -ve, T. S. B = 6 . 5 mg/dl ,Blood group O + ve CXR showed C /T ratio of 70 % with right atrial enlargement. (figure 2 )



Figure 2 Chest x ray of the neonate on 3rd day

On the second day, echo was done which showed cardiomegaly with tricuspid valve regurgitation (functional) due to PPHN secondary to meconium aspiration.

Treatment started with. Frequent suction of secretions, continuous O<sub>2</sub> supply, ampiclox vial 250 mg qid. claforan vial 200mg qid. Luminal bolus dose 20 mg / kg & After 12 hr given maintenance 5 mg / kg / day. Mannitol 1g / kg / single dose, vit K ampoule 5 mg and i.v. fluid. Follow up chart include O<sub>2</sub> saturation, H. R, temp, R. R and blood sugar. With oxygen, O<sub>2</sub> saturation became 92 % in the first 4 days of admission, on the 5th day, a trial of weaning from O<sub>2</sub> was done & O<sub>2</sub> saturation was 95 %.

On the 6th day, patient was without O<sub>2</sub> supply & O<sub>2</sub> saturation was 98 %, on the 5th day trophic feeding ( expressed breast milk ) was started ( 3cc every 3hr ) successfully without complications.

On 7th day, breast feeding was on demand. The vital signs on the 7th day were as follow RR 50 breath / min. H. R 125 breath / min Blood sugar 120 mg / dl O<sub>2</sub> saturation 98 % On the same day CXR repeated & showed C / T ratio of 50 % with clear lung zones as shown in figure 3.

Next day echo showed normal heart chambers with normal ejection fraction without any congenital anomaly.

The baby discharged well from NICU after 14 days.



Figure 3 Chest x ray of the neonate on 8th day

## Discussion

Primary pulmonary hypertension of newborn can cause death if it is severe. Prior to introduction of ECMO support, mortality rate were reported from 12 – 50 %<sup>(1, 2)</sup>, although ECMO support has increased survival to about 85 % in infant with severe PPHN, it may be associated with significant morbidity in 10 – 45 % of patients<sup>(3, 4)</sup>.

Our case diagnosed early as PPHN secondary to meconium aspiration syndrome because the baby was meconium stained with cardiomegaly on CXR with tricuspid valve regurgitation by echo while in primary PPHN, the CXR & echo findings are usually normal.

Our patient responded to hyperoxic test with 100 % O<sub>2</sub> when the O<sub>2</sub> saturation increases from 77 % to 92 % & more, which means that the PPHN is secondary to parenchymal lung disease (meconium aspiration syndrome) without right to left shunt.

## Conclusion

PPHN is not uncommon & should be suspected in any newborn with persistent cyanosis in the first few days of life if there is no clear cause for the cyanosis.

## References

1. Perkin RM , Levin DL , Clark R : 1984 serum salicylate level and right to left ductus shunts in newborn infants with persistent pulmonary hypertension .J Pediatr 96:721-726
2. Haworth SG, Reid L, 1976. Persistent fetal circulation : newly recognized structural feature , J Pediatr 88:614-620
3. Levin DL, Hyman AI, Heymann MA, Rudolph AM:1978aFetal hypertension and the development of increased pulmonary vascular smooth muscle :a possible mechanism for persistent pulmonary hypertension of the newborn infant .J Pediatr 92:265-269
4. Murphy JD,Rabinovitch M,Gold stein JD,Reid LM,1981 the structural basis of persistent pulmonary hypertension of the newborn infant .J pediatr 98:962-967
5. Geggel RL, Reid L : 1984 the structural basis of PPHN .clin perinatol 11:525-549
6. Steinhorn RH,Millard SL , Morin FC, 1995 .PPHN .Role of nitric oxide and endothelin in pathophysiology and treatment .Clin Perinatol 22:405-428
7. Levin DL,Weinberg AG, Perkin RM:1983 Pulmonary microthrombi syndrome in newborn infant with unresponsive persistent pulmonary hypertension .J Pediatr 92:265-269