Original Article

Pediatric Education and Research
Volume 5 Number 3, September - December 2017
DOI: https://dx.doi.org/10.21088/per.2321.1644.5317.5

A Study on Maternal Risk Factors and Preterm Neonates

Divyarani D.C.*, Goudappa R. Patil**

*Assistant Professor, Department of Pediatrics, Kodagu Institute of Medical Sciences, Madikeri, Karnataka 571201, India.
**Senior Resident, Department of Pediatrics, Gulburga Institute of Medical Sciences, Kalaburagi, Karnataka 585101, India.

Abstract

Introduction: Factors that affect the state of lung development at birth include prematurity, maternal diabetes and genetic factors (white race, history of RDS in siblings, male sex). Thoracic malformations that cause lung hypoplasia, such as diaphragmatic hernia, may also increase the risk for surfactant deficiency. *Methodology:* The maternal and gestational variables studied were: Age (years), number of pregnancies, prior history of miscarriages, still births and premature deliveries; type of delivery (normal or caesarean); previous caesarean section, intercurrent clinical conditions observed during gestation – diabetes, hypertension, anemia, urinary infections at any point during pregnancy, syphilis, human immunodeficiency virus (HIV), toxoplasmosis, heart disease, hepatitis B, premature rupture of membranes (PROM) for longer than 18 hours, placental abruption *Results:* Maternal risk factors were present in 67% mothers which constitute about 67%. Among which Anemia (15%) and PROM (10%) has high incidence. PIH and previous history of LSCS constitute about 9% and 5% followed by Maternal Fever and younger age 9% & 4% . APH were found in 3 which constitute about 3%. BOH found in 2 mothers which accounts for about 2%. *Conclusion:* In the present study also PROM and anemia constitute the major maternal risk factor.

Keywords: Maternal Risk Factors; Preterm Neonates; PROM.

Introduction

After birth, infants with fetal lung structure and immature functional capacity are at greatest risk of respiratory distress need for oxygen and positive pressure ventilation and admission for intensive care. From 340/7 through 366/7weeks' gestation, terminal respiratory units of the lunge volve from alveolar saccules lined with both cuboidal type II and flat type Iepithelial cells (terminal sac period) to mature alveoli lined primarily with extremely thin type I epithelial cells(alveolar period) [1]. During the alveolar period, pulmonary capillaries also begin to bulge into the space of each terminal sac and adult pool sizes of surfacetant are attained. Functionally, this immature lung structure may be associated with delayed intrapulmonary fluid absorption, surfactant in sufficiency and in efficient gas exchange [1]. They pose resuscitation difficulties at birth, often followed by hyaline membrane disease, if associated with deficiency of pulmonary surfactant. Pulmonary aspiration and atelectasis are common [2].

Little is known about cardio vascular physiology and path biology in latepreterm infants; it is generally believed that structural and functional immaturity restricts the amount of cardiovascular reserve that is available during times of stress. Immature cardiovascular functional so may complicate recovery of the late-preterm infant with respiratory distress because of delayed ducts arteriosus closure and persistent pulmonary hypertension [3].

The primary cause of respiratory distress syndrome (RDS), also known as hyaline membrane disease, is in adequate pulmonary surfactant due to

Corresponding Author: Goudappa R. Patil, Senior Resident, Department of Pediatrics, Gulburga Institute of Medical Sciences, Kalaburagi, Karnataka 585101, India.
E-mail: goudappar1984@gmail.com

Received on 24.10.2017, **Accepted on** 04.11.2017

pretermbirth. The manifestations of the disease are caused by the resultant diffuse alveoli ate lactases, edema and cell injury. Subsequently, serum proteins that inhabit surfactant function leak into the alveoli. The increased water content, immature mechanisms for clearance of lung liquid, lack of alveolar-capillary apposition and low surface area for gas exchange typical of the immature lung also contribute to the disease. Significant advances made in the management of RDS include the development of prenatal diagnosis to identify infants at risk, prevention of the disease by antenatal administration of glucocorticoids, improvements in prenatal and neonatal care, advances in respiratory support and surfactant replacement therapy. As a result, the mortality from RDS has decreased. However, the survival of increasing numbers of extremely immature infants has provided new challenges and RDS remains an important contributing cause of neonatal mortality and morbidity [4].

Prenatal Risk Factors

1. Factors that affect the state of lung development at birth include prematurity, maternal diabetes and genetic factors (white race, history of RDS in siblings, male sex). Thoracic malformations that cause lung hypoplasia,such as diaphragmatic hernia, may also increase the risk for surfactant deficiency.
2. Factors that may acutely impair surfactant production,release or function include perinatal asphyxia in premature infants and cesarean section without labor. Infants delivered before labor starts do not benefit from the adrenergic and steroid hormones released during labor, which increase surfactant production and release. As a result, RDS may be seen in late preterm or early term infants delivered by elective cesarean section.

Antenatal corticosteroid therapy should be given to pregnant women 24 to 34 weeks gestation with intact membranes or with pretermrupture of the membranes (PROM) without chorioamnionitis, who are at high risk for preterm delivery within the next 7 days. Treatment at gestational ages <24 weeks is of questionable efficacy.

This strategy induces surfactant production and accelerates maturation of the lungs and their fetal tissues, resulting in a substantial reduction of RDS, intra ventricular hemorrhage (IVH), necrotizing enterocolitis and perinatal mortality. A fullcourse consists of two doses of betamethasone (12mgIM) separated by a 24-hourinterval, or four doses of dexamethasone (6 mg IM) at 12-hour intervals, although incomplete courses may improve out come. Contra indications to treatment include chorioamnionitis or other indications for immediate delivery [5].

A premature infant with RDS has clinical signs shortly after birth.These include tachypnea, retractions, flaring of the nasal alae, grunting and cyanosis.The classic radiographic appearance is of low volume lungs with a diffuse reticulo granular pattern and air broncho grams [6].

Methodology

The maternal and gestational variables studied were: Age (years), number of pregnancies, prior history of miscarriages, still births and premature deliveries; type of delivery (normal or caesarean); previous caesarean section, intercurrent clinical conditions observed during gestation–diabetes, hypertension, anemia, urinary infections at any point during pregnancy, syphilis, human immuno-deficiency virus (HIV), toxoplasmosis, heart disease, hepatitis B, premature rupture of membranes (PROM) for longer than 18 hours, placental abruption.

The neonatal variables studied were: Age at admission, days in hospital,sex, birth weight; gestational age (Calculated from modified Ballard's scoring); hypothermia/ hyperthermia (hypothermia: body temperature below 36°C, hyperthermia: temperature above 37.5°C); hypoglycemia (glucose below 40 mg/dL); hyperbilirubinemia requiring phototherapy/exchange transfusion; feed intolerance; respiratory pathologies – transient tachypnea of the newborn (TTN), hyaline membrane disease (HMD), pneumonia, sepsis,interventions done, deaths, rehospitalizations.

Inclusion Criteria

All late preterm babies ($34^{0/7}$ weeks to $36^{6/7}$ weeks) admitted to SNCU and postnatal wards for a period of Five months (September 2015 –February 2016).

Exclusion Criteria

1. Late preterm babies of parents who have not given consent.
2. Late preterm babies who had surgical conditions, congenital malformations, genetic disorders, metabolic disorders other than hypoglycaemia (suspected IEM), babies of multiple gestation.

Results

Maternal risk factors were present in 67% mothers which constitute about 67%. Among which Anemia (15%) and PROM (10%)has high incidence. PIH and previous history of LSCS constitute about 9% and 5% followed by Maternal Fever and younger age 9% & 4% . APH were found in 3 which constitute about 3%. BOH found in 2 mothers which accounts for about 2%. Diabetes and found in 1%. Maternal risk factor could not be elicited in 33 cases which constitute about33%. And 6 (6%)mothers had 2 or more identifiable risk factors.

Regarding mode of delivery, vaginal delivery was conducted in 81 neonates which accounts for 81%. 19 neonates were born through LSCS which accounts for 19%.

Regarding birth weight, 17 neonates were born with birth weight between 2 and 2.5kg which constitute about 17%. 1 neonate were born with birth weight above 2.5 kg which constitute about 1%. 68 neonates were born with birth weight between 1.5 and 2 kg which constitute about 68%. 17 neonates were born with birth weight of <1.5 kg which constitute17%.

Table 1: Maternal risk factor

Maternal risk factor	No. of patients (n=203)	%
No	33	33
Yes	67	67
1.PROM	10	10
2. Previous LSCS	5	5
3.Anaemia	15	15
4.PIH	9	9
5. Fever	9	9
6. APH	3	3
7.Younger age	4	4
8 BOH	2	2
9.Diabetes	1	1
10.Elderlyprimi	3	3
11.2 or more risk factors	6	6

Table 2: Mode of delivery

Mode of delivery	No. of patients	%
NVD(Normal Vaginal delivery)	81	81
LSCS	19	19
Total	**100**	**100**

Table 3: Birth weight (kg)

Birth weight	No. of patients	%
<1.5	14	14
1.5-2	68	68
2-2.5	17	17
>2.5	1	1
Total	100	100

Discussion

Maternal risk for preterm was elicited in 67 cases which accounts for about 67%. There were no recorded indication In 33 mothers which constitute about 33%. Among risk factors studied Anemia & PROM constitutethemajor one's of 15% and 10% respectively followed by PIH(9%) & more than two risk factors respectively.

Reddy et al. had studied the "Delivery indications of late-preterm gestations" in 2009 and he categorized delivery indications as follows: (1) maternal medical conditions; (2) obstetric complications; (3) major congenital anomalies; (4) isolated spontaneous labor: vaginal delivery without induction and without associated medical/obstetric factors; and (5) no recorded indication. Of the 292 627 late-preterm births, the first 4 categories (those with indications and isolated spontaneous labor) accounted for 76.8%. The remaining 23.2% (67,909) were classified as deliveries with no recorded indication. He concluded

that a total of 23% of late preterm births had no recorded indication for delivery noted on birth certificates and patient factors may be playing a role in these deliveries. It is concerning that these infants had higher mortality rates compared with those born after spontaneous labor at similar gestational ages [7].

In this study there were no recorded indications in 33% of the neonates which is inpar with Reddyetal. Prolonging pregnancy to the maximum safest gestation will result in decrease in morbidities.

Tucker J Metaldid a study, "Etiologies of preterm birth in an indigent population: is prevention logical expectation? " The study results were compared with births < 34 weeks, late preterm births are more likely to be the result of spontaneous idiopathic preterm labor or PPROM than medical or pregnancy indications [8]. It has been estimated that the relative distribution of etiologies of preterm birth < 34 weeks' gestation is 30% indicated, 30% PPROM, and 40% spontaneous preterm labor. For late preterm births, the relative distribution of etiologies changes to 20% indicated, 25% PPROM, and 55% preterm labor. As such, larger proportion of late preterm births are due to spontaneous preterm labor (two- thirds) compared with PPROM (one-third) [4].

In the present study also PROM and anaemia constitute the major maternal risk factor (15%) which is in par with the above study and spontaneous preterm labor seen in 5%.

Laughon et al. in 2010 reported similar findings, showing that a consider able number of preterms are born by caesarean with no record of any indication for caesarean delivery, which suggests that they are potentially avoidable. In that, 15,136 late preterm infants were studied and categories the precursors as "spontaneous labor," "premature rupture of the membranes (preterm PROM),"indicated" delivery and" unknown. "The study concluded PROM as the major precursor of latepreterm delivery constituting about 32.3% followed by "indicated" (obstetric, maternal, or fetal condition) and spontaneous labor which constitute 31.8% and 29.8% respectively. In 6.1% precursor were unknown. They concluded that one in 15neonates delivered late preterm for "soft" or elective precursors [9]. In this study also 33% of late preterm were with unknown precursor which is comparable with the above study. About 81 neonates had vaginal deliveries which constitute about 81%. LSCS was done in 19 neonates which constitute19%.

Jean-Bernard Gouyon et alin 2010 did a study, Neonatal outcome associated with singleton birth at 34–41 weeks of gestation. In this study they found vaginal delivery was more compared to Caesarean section which is inpar with the present study. They also found gestational age was positively correlated with vaginal delivery and negatively correlated with emergency caesarean section [10].

Conclusion

Prolonging pregnancy to the maximum safest gestation will result in decrease in such morbidities. Further studies in the physiology, developmental maturity that are specific to latepreterm infants are required.

References

1. William A. Engle, Kay M. Tomashek and Carol Wallman ''Late-Preterm'' Infants: A Population at Risk, American Academic of Pediatrics 2007;120:1390.
2. Cloherty John P, Eichenwald, Eric C, Stark, Ann R. Manual of Neonatal Care, 6th Edition. Lippincott, 2012.p.323-330.
3. Moster D, LieR T, Irgens LM, Bjerkedal T, Markestad T. The association of Apgar score with subsequent death and cerebral palsy: A population basedstudyin term infants. J Pediatr 2001;,138:798:803.
4. Beserth CL. Developmental anatomy and physiology of the gastrointestinal tract. In: Taeusch HW, Ballard RA, Gleason CA, eds. Avery's Diseases of the Newborn. 8th ed. Philadelphia, PA: Elsevier Saunders, 2005;1071:1085.
5. Sundaram V, Kumar P, Narang A. Bacterial profile of early versus late onset neonatal sepsis in a North Indian tertiary care centre: Heading towards a change. J Pediatr Infect Dis 2009;4:241-245.
6. Reddy R, Dutta S, and Narang A. Multi-variate analysis of risk factors of early onset neonatal sepsis in preterm infants.2004. Postgraduate Institute of Medical Education and Research, Chandigarh. 2004. Ref Type:Thesis/Dissertation.
7. Arpino C, Compagnone E, Montanaro ML, Cacciatore D, De Luca A, Cerulli A, et al. Preterm birth and neurodevelopmental outcome: a review. Childs Nerv Syst 2010;26:1139-1149.
8. Adams-Chapman I. Neurodevelopmental outcome of the late preterm infant. Clin Perinatol 2006; 33: 947-964.
9. Laughon SK, Reddy UM, SunL, Zhang J "Precursors for late preterm birth in singleton gestations" Obstet Gynecol. 2010 Nov;116(5):1047-55.
10. Ryan WL, Mounira H, Candice CS, Clint MC, David FL, Emily AD. Late preterm birth. Rev Obstet Gynecol 2010;3:10-19.