

Magnifying Glass on Why Newborns Die : Low Birth Weight and Maternal Factors

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ABSTRACT

Background: Low birth weight babies weighing less than 2000gms weight are immunologically deficient. In addition, they are at risk of birth asphyxia, hypothermia, respiratory distress, hyperbilirubinemia, hypoglycaemia, coagulopathy, sepsis and neonatal death. The apt way to reduce mortality is to identify the pre-pregnant and antenatal factors which ascertain the uterine milieu and nutrient bioavailability to the fetus in utero.

Aim: This study was designed to reduce the mortality in low birth weight babies. We studied the various maternal and placental factors which affect the fetus in utero. Intrapartum events like presentation, premature rupture of membranes and cord accidents were also recorded.

Materials and methods: This prospective study was carried out on 100 low birth weight babies out of 1800 deliveries in the Department of Paediatrics at Saveetha Medical College and hospital, Chennai, India between 1 October 2012 and 30 September 2013. Maternal factors like age, parity, prepregnancy body mass index, haemoglobin levels, preeclampsia,

placental abnormalities, presentation, premature rupture of membranes and mode of delivery were studied. Cases of still birth, chronic maternal illness and gestational diabetes mellitus were excluded.

Result: Seven newborns out of hundred died despite the level 2 neonatal intensive care. We found that maternal age (<20 and >30), weight <45kg, preeclampsia, anaemia, placental abnormalities and breech presentation were the maternal factors significantly associated with increased mortality in low birth weight babies. However in 28.6% of low birth neonate mortality no maternal cause could be identified. A literature search revealed that these could be inherent foetal genetic and/or chromosomal anomalies.

Conclusion: Maternal age, Prepregnancy Body mass index, preeclampsia, maternal anaemia, breech presentation and placental abnormalities are significant maternal factors resulting in mortality in low birth weight babies. Only in one third cases no maternal cause can be identified. Timely correction of maternal body mass index and anaemia can reduce the death rates of our low weight newborns.

Keywords: Low birth weight mortality, Preeclampsia, Prepregnancy body mass index

INTRODUCTION

Low birth weight babies are forty times more likely to die within their first four weeks of life than normal birth weight infants. Low birth weight babies are also more likely than normal weight babies to have neuro developmental complications and congenital anomalies [1]. While the under than five childhood mortality rates have declined, the neonatal mortality rates have remained same [2]. Globally, there are 4 million neonatal deaths out of the approximately 130 million annual live births [3]. Low birth weight accounts for nearly 80% of neonatal deaths. The proportion of Low birth weight has increased in the past 20 years. This increase is attributed to changes in the frequency of multiple births, increase in obstetrical intervention, improved ascertainment of early preterm births, and increased use of ultrasound for estimating the gestational age. Unfortunately the documentation of low birth weight is not proper [4].

A Low birth weight baby can be preterm baby or an intrauterine growth restricted baby. With respect to gestational age, a low birth weight baby can be a preterm appropriate for gestational age or a term intrauterine growth retarded baby. At times, we face a triple problem of a low birth weight intrauterine growth retarded baby who has been delivered before 37 completed weeks of gestation. Millennium development goals, are broad global commitment to address general poverty and health, the goal four of which aims to reduce under five mortality rate by two third between 1990-2015 [5]. With better care of low birth weight babies the forth Millennium development goal can be achieved. The care of a low birth weight newborn necessitates judicious resource utilization as the countries with highest mortality have least of modern infrastructure [6]. Defining the risk factors and timely intervention can reduce the burden of low birth weight mortality, thereby accelerating the trend towards Millennium Development.

Outcome	Preterm AGA	Preterm SGA	Term IUGR	Total
Death	5	2	0	7
Survival	46	27	20	93
Total	51	29	20	100

[Table/Fig-1]: Gestational age and birth weight as a marker of neonatal mortality

Age	Neonates	Death	Mortality Percentage
Mother <20 Years and >35 Years	22	2	9.09
Mothers 20-35 Years	78	5	6.41
Total	100	7	7.00

[Table/Fig-2]: Maternal age and low birth weight mortality

Nutritional Status	total	Survival	Mortality
Normal	77	74	3(3.9%)
Undernourished	23	19	4 (17.39%)

[Table/Fig-3]: Maternal weight less than 45 Kg and low birth weight mortality

Outcome	Not Present	Present	Total
Death	3(43.85%)	4(18.18%)	7
Survival	5(96.15%)	18(81.82%)	93
Total	78	22	100

[Table/Fig-4]: Pregnancy induced hypertension and low birth weight mortality

Outcome	Not Present	Present	Total
Death	4(4.55%)	3(25%)	7
Survival	84(95.45%)	9(75%)	93
Total	88	12	100

[Table/Fig-5]: Placental problems and low birth weight mortality

Medical risk factors for Low birth weight before pregnancy are maternal age (<20 or >35), chronic conditions like hypertension, renal disease, cardio respiratory, autoimmune, endocrine or infectious diseases. The risk factors for low birth weight during pregnancy are hypertensive disorders, Gestational diabetes, malnutrition, antepartum haemorrhage, anaemia, infections, placental or foetal anomalies and multiple pregnancies. Common foetal factors are genetic and/or chromosomal aberrations. The IUGR problems are due to uteroplacental insufficiency and inadequate substrate transfer leading to birth asphyxia, hypothermia, meconium aspiration, polycythaemia, hypoglycaemia, hypocalcaemia and thrombocytopenia. The preterm complications are caused by anatomic and physiological immaturity e.g. respiratory distress due to delayed alveolar clearance of water and surfactant deficiency, delayed post natal circulatory adaptation

with pulmonary hypertension, systemic hypotension and delayed closure of foetal shunts. Liver immaturity and reduced substrata explain high prevalence of jaundice and coagulation factor deficiency. Immature vascular development in central nervous system and retina predisposes to intraventricular haemorrhage and retinopathy. Immature skin and mucosal barrier and undeveloped cellular and humoral immunity lead to neonatal sepsis and nosocomial infections. Gastrointestinal mucosal and immunological immaturity results in necrotising enterocolitis. In a growth restricted preterm baby the clinical picture is superimposed and confounded. It poses a diagnostic and therapeutic challenge in Neonatal intensive care unit.

Perinatal asphyxia is recognised as a major cause of neonatal mortality and morbidity the effects of which are more pronounced in Low birth weight [7, 8]. In our study prenatal maternal BMI, age, haemoglobin has been assessed as a risk factor of Low birth weight mortality. Pregnancy induced hypertension, placental problems like infarcts, small placenta and retroplacental haemorrhage have been explored as a causative associate of mortality in low birth weight. We have also ascertained the impact of podalic presentation in increasing mortality in a low birth weight newborn.

MATERIALS AND METHODS

A prospective study was carried out on 100 low birth weight babies out of 1800 deliveries in the Department of Paediatrics at Saveetha Medical College and hospital, Chennai, India between 1 October 2012 and 30 September 2013. Pregnancies with previous still births, gestational diabetes and chronic prepregnancy illness like renal, cardiac, hypertension were excluded. Detailed maternal factors like age, gestational age, parity, prepregnancy body mass index, previous low birth weight, haemoglobin levels, Urinary tract infection and preeclampsia were recorded. Foetal Presentation, intrapartum foetal hypoxia, premature rupture of membranes, instrumental delivery and placental problems like infarcts, retroplacental calcifications, small placenta, and premature separation were noted.

In newborn, gestational age as calculated by Naegle's formula was confirmed according to the Ballard's modification of Dubowitz et al.,. The detailed account of postpartum events like apgar score and resuscitation were recorded. The post natal course in the hospital including the presence of apnoea, seizures, patent ductus arteriosus, intraventricular haemorrhage, hyaline membrane disease, hyperbilirubinemia, necrotizing enterocolitis, duration of oxygen therapy and ventilator support were noted. Statistical analysis was done using Fisher Exact test.

RESULTS

During the study period, 100 (5.55%) pregnancies resulted in Low birth weight neonates out of total 1800 pregnancies. Out of 100 low birth weight infants, 93 survived beyond four weeks of life. In 2 neonates (28.6%) no maternal cause of mortality could be identified. [Table/Fig-1] tells us that the preterm AGA has a significant

incidence of mortality as compared to preterm SGA and IUGR. Gestational age rather than weight is a predictor of neonatal mortality. [Table/Fig-2] brings up the fact that maternal age less than 20 and above 35 years as compared to 20 to 35 years confers a significant risk (9.09% v/s 6.41%) for low birth weight baby mortality ($p < 0.05$). [Table/Fig-3] tells us that maternal weight less than 45 kg is a significant risk factor for LOW BIRTH WEIGHT mortality (17.39% v/s 3.85%). An abnormal placental examination was associated with significant risk (25% v/s 4.55%) for low birth weight mortality ($p < 0.5$) as shown in [Table/Fig-4]. Breech presentation was associated with significant worsening (33.33% v/s 5.32%) of low birth weight mortality rate. Several other factors were also found to have bearing on low birth weight infant mortality. It was found that maternal anaemia (Hb < 8 gm/dl) in seventeen cases had a mortality of 11.76% . Premature rupture of membranes was noticed to have a mortality rate of 8.33%. Urinary tract infection was found in five pregnancies which was associated with two low birth weight neonatal deaths.

DISCUSSION

The frequency of low birth weight found in this study is 5.55%, the reported incidence varies from 6-18% (1,9,10,11). Maternal age less than 20 years is associated with compromised placental nutrient transfer as the metabolic needs of a growing teenager competes with the requirements of an intrauterine foetus [12]. Maternal age above 35 years is believed to be associated with foetal genetic and /or chromosomal aberrations [13]. The mechanisms of association between prepregnancy BMI and IUGR and preterm delivery are not clear, but probably related to protein energy bioavailability and changes in maternal hemodynamic status [14-16].

In this study anaemia was a significant factor but contribution of anaemia leading to Low birth weight is controversial [17,18]. The mechanism by which anaemia could produce this effect is unknown, but other concurrent nutrient deficiencies are an important contributing factor [19]. One study even reported harmful effects of Iron supplementation [20].

In this study women with preeclampsia are more likely to have Low birth weight mortality. Preeclampsia by reducing plasma volume reduces the supply of nutrients to the fetus thus affecting foetal growth [21, 22]. Maternal age, prepregnancy BMI, haemoglobin, placental aberrations, preeclampsia and Breech presentation are reliable significant factors contributing to mortality in Low birth weight babies [23].

CONCLUSION

The results of this study suggest that for reducing the problem of Low birth weight, the public health strategy needs to focus on avoidance of teenage pregnancy . Pregnancy beyond 35 years should be monitored more closely. Prepregnancy maternal nutritional status and anaemia have to be corrected. Pregnancy induced hypertension and other placental pathologies need to be screened and managed timely to reduce the death rates of our low weight newborns.

CONSENT

We have obtained the patient's consent for participation in the study of low neonatal birth weight.

COMPETING INTERESTS

We do not have any commercial association that might pose a conflict of interest in connection with the manuscript. We certify that neither this manuscript nor one with substantially similar content under our authorship has been published or is being considered for publication elsewhere.

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