

Original Research Article

Association of Low Birth weight and Cerebral palsy

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Abstract

Introduction: Damage to the motor control centres of the developing brain causes cerebral palsy (CP), which can occur during pregnancy, childbirth, or after delivery. **Objective:** To study the associated risk factor low birth weight for spastic CP in a retrospective study involving children with CP. **Materials and methods:** The study population included 187 children with CP. Data was collected from the medical records and from the parents of affected 187 Cerebral palsy children attended / admitted during the period from 2014 to 2020 in Rani Chandramani Devi Government hospital and Rehabilitation centre, PedaWaltair, Visakhapatnam, Andhra Pradesh and analysed retrospectively. The analysis of data from the case records included the following: new born gestational age, mother's age, gender, birth weight, maternal health factors, CP child order in the family. **Results:** Cerebral palsy was seen more in males 122 (65.24%) than females. Taking 2500 grams is the average weight of a normal baby in our Indian set up, 187 Cerebral palsy children are below 2500 g. Out of 187 babies, 115 are of Low Birth Weight (LBW) with weight ranging from 2500 g to 1501 g. 55 very low birth weight (VLBW) weight ranging from 1500 g to 1001 g, and 17 were extremely low birth weight (ELBW) weight ranging below 1000 g. Among the total 187 low birth weight babies, 62 are Pre term and 125 are full term born. The maternal co-morbid factors anaemia 68 (36.36%) plays a major role in the present study. Gestational age is one of the major factor responsible for CP in children of low birth weight. More CP cases were seen in pre-term gestational age 28 weeks 23 (12.29%). **Conclusion:** Using recent advances in diagnostic modalities for detecting any deviations in the growing foetus and mother's health, appropriate and early interventions for problems that are treatable should prevent low birth weight and its serious consequences in the immediate or future neurodevelopmental status of the born child.

Keywords: Low birth weight, Cerebral palsy, anaemia, gestational age, genetic factor

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Introduction

Cerebral palsy (CP), the most prevalent motor impairment in children caused by early insults to the developing brain, is a syndrome that has a significant impact on the affected children's and their families' quality of life. In advanced areas, the prevalence of CP is estimated to be between 1.8 and 3.5 per 1000 live births [1-5]. Despite improvements in prenatal and neonatal intensive care, the frequency of CP has remained relatively consistent in wealthy countries over the previous several decades. Perinatal abnormalities may not be the primary cause of cerebral palsy. Strong evidence has shown that CP is a heterogeneous disease caused by genetic factors, intrauterine triggers, perinatal and neonatal diseases, or their interactions, such as maternal infection, gestational age, small for gestational age (SGA), birth defects, placental conditions, genetic mutations, birth asphyxia, and neonatal diseases, as well as their interaction effects, such as maternal infection, gestational age, SGA, birth defects, placental conditions, genetic mutation Gender and SGA, which refers to newborns whose birth weights are less than the 10th percentile for their gestational age and is used as a proxy for prenatal growth restriction, have been linked as key risk factors for CP [6-8]. Low birth weight (LBW) is defined by the World Health Organization

(WHO) as a weight at birth of less than 2,500g. This practical cut-off for worldwide comparison is based on epidemiological findings indicating neonates weighing fewer than 2,500g are 20 times more likely to die than babies weighing more than 2,500g. Low birth weight is linked to a variety of factors, including socioeconomic status, mother's age, diet, health status, and education. Male infants were more vulnerable to the effects of growth deviations than female newborns, with the effect being so severe that "the well-known excess rate of cerebral palsy among male babies is restricted to select portions of the weight for gestation distribution." This tight link could be explained by the fact that small size is frequently the result of intrauterine growth restriction (IUGR), which affects many anthropometric parameters at the same time (if not always equally). It's possible that some anthropometric measurements will be influenced differently in circumstances where causes other than IUGR are implicated [9-11]. Short maternal stature, early age, high parity, and narrow birth spacing have all been linked in studies. Many women in underdeveloped nations are short and underweight, and the number of low birth weight (LBW) newborns is very high (more than 30% in South Asia, 10-20% elsewhere). LBW babies have a lower probability of survival, and if they do survive, they are more susceptible to sickness, growth retardation, and mental development problems such as cerebral palsy [12-14]. Keeping all these in views, an attempt has been made in our institute to carry out a retrospective analysis in order to find out the association between low birth weight and cerebral palsy in newborn infants.

Materials and Methods

The study population included 187 children with CP. Data was collected from the medical records and from the parents of 187 below 2500 g Cerebral palsy children attended / admitted during the period from 2014 to 2020 in Rani Chandramani Devi Government hospital and Rehabilitation Centre, PedaWaltair, Visakhapatnam, Andhra Pradesh and analysed retrospectively. The analysis of data from the case records included the following: new born gestational age, mother's age, gender, birth weight, maternal health factors, CP child order in the family. A paediatrician who specialized in child neurology had diagnosed all the cases in the present study. Cerebral palsy was diagnosed as a primary or secondary diagnosis using the International Classification of Diseases, 9th revision (codes 343.0-3 and 8-9) or the International Classification of Diseases, 10th revision (codes 343.0-3 and 8-9) systems (codes G.80.0-9). The data was statistically represented as a percentage.

Results

Cerebral palsy was seen more in males 122 (65.24%) than females (Table 1). Taking 2500 grams is the average weight of a normal baby

in our Indian set up, out of 187 Cerebral palsy children, 115 are of Low Birth Weight (LBW) with weight ranging from 2500 g to 1501 g, 55 very low birth weight (VLBW) weight ranging from 1500 g to 1001 g, and 17 were extremely low birth weight (ELBW) weight ranging below 1000 g. Among the total 187 low birth weight babies, 62 are Pre term and 125 are full term born (Table 1). By studying the maternal factors involved in CP it was noticed that, maternal age below 20 years showed more CP cases 94 (50.24%) comparatively than other age groups (Table 2). Among the maternal co-morbid factors anaemia 68 (36.36%) plays a major role in the present study (Table 2) (Figure 1). While taking the family history details from the affected CP children, more cases were reported from the mother with first child (Table-3). Gestational age is one of the major factor responsible for CP in children of low birth weight. More CP cases were seen in pre-term gestational age 28 weeks 23 (12.29%) (Table-4). Among the other maternal factors involved, more CP cases were seen in mothers prone to premature rupture of membrane during pregnancy (Table 5).

Table 1: Distribution of CP cases based on gender, birth weight and maturity

| Sex | Number | Percentage |
|------------------|------------|------------|
| Male | 122 | 65.24 |
| Female | 65 | 53.27 |
| Total (N) | 187 | |
| Birth weight | Number | Percentage |
| <1000g | 17 | 9.09 |
| 1001g – 1500g | 55 | 29.41 |
| 1501g – 2500g | 115 | 61.49 |
| Total (N) | 187 | |
| Maturity | Number | Percentage |
| Preterm | 62 | 33.15 |
| Full term | 125 | 66.84 |
| Total (N) | 187 | |

Table 2: Maternal factors influencing Cerebral Palsy

| Maternal age | Number | Percentage |
|-----------------|------------|------------|
| Below 20 years | 94 | 50.24 |
| 21-30 years | 86 | 45.98 |
| Above 30 years | 7 | 3.74 |
| Total | 187 | |
| Co-morbidities | Number | Percentage |
| Anaemia | 68 | 36.36 |
| Hypertension | 17 | 9.09 |
| Diabetes | 01 | 0.53 |
| Thyroid defects | 06 | 3.20 |

Table 3: Cerebral palsy child order in the family

| CP order in family | Number | Percentage |
|-----------------------|------------|------------|
| 1 st child | 111 | 59.35 |
| 2 nd child | 52 | 27.80 |
| 3 rd child | 15 | 8.02 |
| 4 th child | 9 | 4.85 |
| Total | 187 | |

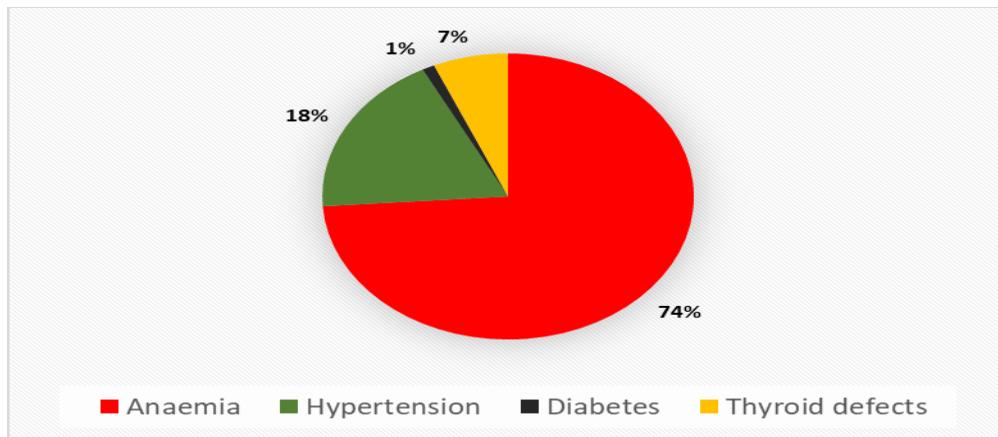
Table 4: Gestational age

| Gestational age (Weeks) | N=62 (33.15%) | Number | Percentage |
|-------------------------|---------------|--------|------------|
| 22 Weeks | | 1 | 0.53 |
| 24 | | 8 | 4.27 |
| 25 | | 3 | 1.60 |
| 28 | | 23 | 12.29 |
| 29 | | 2 | 1.06 |
| 30 | | 2 | 1.06 |
| 31 | | 4 | 2.13 |
| 32 | | 10 | 5.34 |

| | | | |
|----------------------|--|-----|-------|
| 33 | | 6 | 3.20 |
| 34 | | 2 | 1.06 |
| 35 | | 1 | 0.53 |
| >35 (full term) | | | |
| Total (N=187) | | 125 | 66.84 |

Table 5: Other maternal factors involved in CP

| Other conditions | Number | Percentage |
|---|--------|------------|
| Intra uterine growth retardation (IUGR) | 1 | 0.53 |
| Cervical Incompetence | 1 | 0.53 |
| Premature rupture of membranes (PROM) | 42 | 22.45 |
| Ante partum Haemorrhage (APH) | 10 | 5.34 |
| Total (N=187) | | |

**Fig 1: Maternal Co-morbidities in relation to CP children**

Discussion

Cerebral palsy is a musculoskeletal illness that causes disabilities owing to injury to the developing brain. The brain damage is permanent and non-progressive. Because the musculoskeletal system grows with age, the effects of brain damage progress. Even brain traumas are not all the same [15]. Muscle tone abnormalities, disturbances in balance and motor control, and musculoskeletal deformities are all symptoms of brain damage. The effects of brain injury last a lifetime. An infant with a low birth weight (LBW) is one whose birth weight is less than 2500 g, regardless of gestational age. Infants with very low birth weight (VLBW) weigh 1500 g or less, and infants with extremely low birth weight (ELBW) weigh 1000 g or less (WHO). In India, almost a third of neonates are under the weight of 2500 g. Preterm birth, socioeconomic status, dietary variables, and intrauterine variables are all variables that influence a baby's low birth weight. Pre-term and full-term newborns can both have low birth weight. The likelihood of survival is proportional to the birth weight. When it comes to survival, the baby's vigour at delivery is more crucial than birth weight. A newborn weighing more than 1500 g has a 95% chance of surviving. With intensive care, the survival percentage of a newborn weighing 751–1000 g is as high as 80%. Babies at a gestational age of less than 23 weeks have a mortality rate of more than 97 percent [15]. The pathophysiology of brain injury in preterm and full-term low-birth-weight kids differs and must be examined independently. The pathophysiology of LBW brain injury Anatomic and functional immaturity of blood arteries in the developing brain causes preterm birth. Both arterial and venous structural immaturity have a role when it comes to anatomic immaturity [16]. In a prospective cohort study of newborns admitted to neonatal units weighing less than 2000 g, MacLeod et al. [17] found that more than a third of neonates born weighing less than 2000 g had an IVH, with the majority of these occurring by day 7.

Vaginal birth, earlier gestation, and the requirement for resuscitation following admission to the Neonatal Unit all raised the incidence of IVH, according to the researchers. The conclusion that males have a higher risk of CP than females is consistent with previous results [18-19]. More CP cases were found in low birth weight newborns in this investigation, which is consistent with prior research showing that infants with low birth weight have a higher risk of developing CP than those with higher birth weight [20-21]. Roth et al. [22] investigated into the occurrence of LBW in adolescent mothers and proposed a theory for the young maternal age – the low birth weight gradient, which states that the younger the mother, the higher the risk of producing an LBW baby. The percentage of mothers with CP was highest in his study among women under the age of 20. Several factors such as gestational age, maternal health status such as anaemia, genetic factor, and others would influence CP in neonates in the current study, which is similar to research done by Mark Anez Conteras et al. and Rahman [23-24].

Conclusion

Because prematurity and term pregnancies can both result in low birth weight newborns, numerous steps must be done to prevent prematurity from preterm deliveries and in term births, thereby increasing the mother's and baby's well-being. Preterm labour is one of the leading causes of premature delivery, and it should be avoided to the greatest extent feasible by interventions such as primary care in the form of reducing high-risk factors such as infection, etc. Secondary care, such as early detection screening and preventative treatment, such as tocolytics, and, finally, Tertiary care, which aims to reduce perinatal morbidity and mortality after the diagnosis. Regular antenatal checkups from the start of the pregnancy, with a special focus on moms with high risk factors, as well as optimising the mother's diet, will be extremely beneficial in preventing low birth weight kids. Using recent advances in diagnostic modalities for

detecting any deviation in the growing fetus and mother's health, appropriate and early interventions for problems that are treatable should prevent low birth weight and its serious consequences in the immediate or future neurodevelopmental status of the born child.

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References

1. Tronnes H, Wilcox AL, Lie RT, Markestad T, Moster D. Risk of cerebral palsy in relation to pregnancy disorders and preterm birth: a national cohort study. *Developmental Medicine and Child Neurology*. 2014; 56:779–785.
2. Wang LW, Lin YC, Wang ST, Huang CC. Hypoxic/ischemic and infectious events have cumulative effects on the risk of cerebral palsy in very-low-birth-weight preterm infants. *Neonatology*. 2014; 106:209–215.
3. Neufeld MD, Frigon C, Graham AS, Mueller BA. Maternal infection and risk of cerebral palsy in term and preterm infants. *Journal of Perinatology: Official Journal of the California Perinatal Association*. 2005; 25:108–113.
4. Spinillo A. Antenatal and delivery risk factors simultaneously associated with neonatal death and cerebral palsy in preterm infants. *Early Human Development*. 1997; 48:81–91.
5. Beaino G. Predictors of cerebral palsy in very preterm infants: the EPIPAGE prospective population-based cohort study. *Developmental Medicine and Child Neurology*. 2010; 52:e119–125.
6. Sukhov A, Wu Y, Xing G, Smith LH, Gilbert WM. Risk factors associated with cerebral palsy in preterm infants. *The Journal of Maternal-Fetal & Neonatal Medicine: The Official Journal of the European Association of Perinatal Medicine, the Federation of Asia and Oceania Perinatal Societies, the International Society of Perinatal Obstetricians*. 2012; 25:53–57.
7. Jacobsson B. Cerebral palsy and restricted growth status at birth: population-based case-control study. *BJOG: an international journal of obstetrics and gynaecology*. 2008; 115: 1250 – 1255.
8. Gray PH, Jones P, O'Callaghan MJ. Maternal antecedents for cerebral palsy in extremely preterm babies: a case-control study. *Developmental Medicine and Child Neurology*. 2001; 43:580–585.
9. McIntyre S, Keogh J, Goldsmith S, Badawi N, Blair E. A systematic review of risk factors for cerebral palsy in children born at term in developed countries. *Dev Med Child Neurol*. 2013; 55:499–508.
10. Jarvis S, Glinianaia SV, Arnaud C. Case gender and severity in cerebral palsy varies with intrauterine growth. *Arch Dis Child*. 2005; 90:474–9.
11. Williams MC, O'Brien WF, Spellacy WN. Cerebral palsy, perinatal depression and low ponderal index. *Dev Med Child Neurol*. 1996; 38:661–7.
12. Rajaeefard A, Mohammadi M, Choobineh A. Preterm delivery risk factor: a prevention strategy in Shiraz, Islamic Republic of Iran. *East Mediterr Health J*. 2007; 13:17.
13. Zeleke BM, Mohammed N. Incidence and correlates of low birth weight at a referral hospital in Northwest Ethiopia. *Pan Afr Med J*. 2012; 12:4.
14. Dunin-Wasowicz D, Rowecka-Trzebicka K, Milewska-Bobula B, Kassur-Siemieńska B, Bauer A, Idzik M. Risk factors for cerebral palsy in very low-birthweight infants in the 1980s and 1990s. *J Child Neurol*. 2000; 15(6):417–20.
15. Dutta DC. Text book of Obstetrics. Nilratan Sircar Medical College and Hospital, Kolkata, India, 8th Edition, chapter 32, 2015; 527–540.
16. Kulak W, Okurowska-Zawada B, Sienkiewicz D, Paszko-Patej G, Krajewska-Kulak E. Risk factors for cerebral palsy in term birth infants. *Adv Med Sci*. 2010; 55(2):216–21.
17. MacLeod R, Paulson JN, Okalany N, Acom L, Ikiro J. Intraventricular haemorrhage in a Ugandan cohort of low birth weight neonates: the IVHU study. *BMC Pediatrics*. 2021; 21:12.
18. Johnston MV, Hagberg H. Sex and the pathogenesis of cerebral palsy. *Dev Med Child Neurol*. 2007;49(1):74–8.
19. Skjold B, Alexandrou G, Padilla N, Blennow M, Vollmer B, Aden U. Sex differences in outcome and associations with neonatal brain morphology in extremely preterm children. *J Pediatr*. 2014; 164(5):1012–8.
20. Lodha A, Sauve R, Chen S, Tang S, Christianson H. Clinical Risk Index for Babies score for the prediction of neurodevelopmental outcomes at 3 years of age in infants of very low birthweight. *Dev Med Child Neurol*. 2009; 51(11):895–900.
21. Surman G, Hemming K, Platt MJ, Parkes J, Green A, Hutton J, Kurinczuk JJ. Children with cerebral palsy: severity and trends over time. *Paediatr Perinat Epidemiol*. 2009; 23(6):513–21.
22. Roth J, Hendrickson J, Schilling M, Stowell DW. The Risk of Teen Mothers Having Low Birth Weight Babies: Implications of Recent Medical Research for School Health Personnel. *J Sch Health*. 1998; 68:271–5.
23. Martínez Contreras AM, Prince Vélez R, Medina Ramírez MC. [Preeclampsia: main maternal risk factor for low weight in preterm newborn]. *Ginecol Obstet Mex*. 2008; 76(7):398–403.
24. Rahman LA. Association between pregnancy induced hypertension and low birthweight; a population based case-control study. *Asia Pacific Journal of Public Health*. 2008; 20 (2):152–8..

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