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Original Article

Perinatal factors that contribute to the prevalence of cerebral palsy in Townsville, North Queensland

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1. Introduction

Cerebral palsy (CP) is an umbrella term which describes a group of disorders involving movement and posture causing activity limitations that can occur during fetal or infant brain development (Ferrari and Cioni, 2010). CP is the most common childhood physical disability and in 94% of children who acquire this disability, the brain injury occurs within the pre/perinatal period (Australian Cerebral Palsy Register Group, 2016).

It is difficult to ascertain why the extremely preterm infant is at an increased risk of CP. Recent research has identified an increased survival of the extreme premature infant (Blencowe et al., 2012) and this could be related to advances in perinatal care, including the administration of antenatal steroids or magnesium sulphate, intrapartum antibiotics, delayed cord clamping, postnatal use of caffeine and therapeutic cooling (Badawi and Keogh, 2013). Advances in perinatal care specifically therapeutic cooling may have increased the survival of term infants and consequently, resulted in an increased number of term babies developing CP (Jacobs et al., 2013; Himmelmann and Uvebrant, 2014).

In the past ten years, there have been several studies investigating the prevalence of CP worldwide (Himpens et al., 2008; Wu et al., 2010; Andersen et al., 2011; Reid et al., 2011; Stoknes et al., 2012; Oskoui et al., 2013; Himmelmann and Uvebrant, 2014; Vincer et al., 2014). These studies have identified specific groups either by gestation or birthweight as being at an increased risk of developing CP (Reid et al., 2011; Stoknes et al., 2012; Oskoui et al., 2013). However, these studies did not consider the advances within perinatal practice that could have contributed to reducing the prevalence, but suggested these changes have been attributed to the survival of the extremely preterm infant (Badawi and Keogh, 2013; Van Steenwinckel et al., 2014). Data collection was retrospective in many of these studies and the case group was spread across 26 years (1980–2006) therefore, the results may only reflect some changes in perinatal care. Nevertheless, two important areas for further research have been highlighted from these studies; that regardless of gestation, cause of preterm birth should always be documented and secondly, any information collected should be aligned to a set data collection process so that systematic review and meta-analysis can be conducted giving a better overview of the actual prevalence of CP.

When assessing the prevalence of CP other factors in addition to gestational age at birth must be considered. Risk factors highlighted within the last QCPR report (Queensland Cerebral Palsy Register, 2012) included male gender (57%) and low birthweight (less than 2500 g). The prevalence of CP was 32 times higher in those children born with a birthweight less than 1500 g (Queensland Cerebral Palsy Register, 2012). Multiple births are also a known risk factor for CP with children born one of twins were eight times more likely to develop CP and children born as triplets or of higher plurality were 30 times more likely to develop CP than a singleton pregnancy (Queensland Cerebral Palsy Register, 2012). Indigenous status was also identified as a risk factor for CP particularly post-neonatally acquired CP, thought to be related to infections such as meningitis, encephalitis or epileptic fits and cerebrovascular accidents (Blair and Stanley, 1982; Australian Cerebral Palsy Register Group, 2016). Another factor that has more recently been discussed in literature is maternal mental health, alcohol and drug abuse within this ethnicity as a direct cause for pre/perinatally acquired CP but also an indirect cause for post-neonatally acquired CP (O'Leary et al., 2012) It is important to draw attention to the fact that Indigenous children are 30% more likely to have a physical disability than non-

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Indigenous children of the same age (DiGiacomo et al., 2013). Furthermore, it is widely thought that the prevalence of CP is underestimated within this population because Indigenous people are more accepting of developmental differences (Blair et al., 2016).

This present study focuses on a regional area in North Queensland. The Townsville region was chosen specifically because it is the only hospital providing level six specialised tertiary neonatal care in North Queensland (State of Queensland (Department of Health), 2013) and has a high Aboriginal and Torres Strait Islander population. A level six neonatal service can provide neonatal surgery and care for complex congenital conditions. The objectives of the study were to determine the number of neonates diagnosed with CP over a five-year period (2008–2012) and review the perinatal data to ascertain the associated risk factors.

2. Materials and Methods

This was a retrospective case control study. Medical records for the case group were sourced through a data custodian and the control group was identified through the Townsville hospital neonatal and maternity databases. Ethics approval was obtained from the Townsville Hospital and Health Service (THHS) Human Ethics Committee and the James Cook University Human Ethics Committee. Neonates who were diagnosed with CP (cases) were eligible for this study and we randomly selected 100 infants without CP (control). The control group was matched proportionally for admissions to the neonatal intensive care unit (NICU; 3903 neonates) and those who received routine post-natal care/not admitted to NICU (7824 neonates). Infants diagnosed with post-neonatally acquired CP or infants who died at birth were excluded.

Data was retrieved from medical records based on the causal pathways that have been identified as contributing factors to CP (Australian Cerebral Palsy Register Group, 2013; Badawi and Keogh, 2013). The main data categories included; antenatal history including antibiotics or steroids, intrapartum; cause of birth, method of delivery or delayed cord clamping and neonatal factors such as gestation, birthweight and intervention received within the first 28 days. The outcome or severity of CP was also documented specifically, Gross Motor Function Classification Scale (GMFCS) level and classification of CP. Data collected was affiliated with a data collection form and entered into a Microsoft Excel (Version 16, Microsoft Corp., Redmond, WA) spreadsheet, before being uploaded into IBM Statistical Package for the Social Sciences ™ (SPSS) a data analysis program (Version 23.0, SPSS Inc., Chicago, IL). Given the relatively small number of cases with CP logistic regression could only be conducted on gestation and birthweight data. All other variables were analysed using descriptive statistics and Chi Square (SPSS Version 23). Alpha was set to 0.05.

3. Results

Twenty-three children were diagnosed with CP in the Townsville region within the five-year period reviewed. Eighteen of those 23 were admitted to a NICU (78%) and five received routine post-natal care (22%). Thus, 78% of the control cases were randomly selected from the neonatal database and 22% from the maternity database. Two of the 23 children developed CP post-neonatally, one a near sudden infant death syndrome and the other a non-accidental injury. Both were excluded from the study as perinatal factors had no influence on their disability. This left the case group with 21 participants of which 45% were born preterm.

Table 1 shows that only seven (33%) of the case group received either antibiotics or a complete course of antenatal steroids and in two cases both antibiotics and steroids were given (12%). However, no-one received magnesium sulphate or delayed cord clamping for neuroprotection in this cohort.

Logistic regression showed that neonates born less than 32 weeks were 5.4 times more likely to develop CP (Table 2). Gestational age was then further subdivided to ensure the extreme preterm group (less than 28 weeks) did not skew this data. Table 2 shows that the extreme preterm infant does not affect the statistical significant association of gestational age and CP. Neonates born between 28 and 32 weeks were nearly ten times more likely to develop CP ($p = 0.02$; Table 2). Interestingly, gestational age less than 28 weeks was not a significant risk factor for CP in this small cohort ($p = 0.84$; Table 2).

The mean birthweight of the case group was 2427 g (SD: 625–3830 g) whereas, the control group was slightly higher at 3104 g (SD: 400–5150 g). This was further analysed through logistic regression and identified that if an infant was born with a birthweight less than 2500 g they were 8.5 times more likely to develop CP ($p = 0.02$; 99% C I = 0.787–93.363) (see Fig. 1).

Fig. 2 shows that all the participants born less than 28 weeks ($n = 3$) went on to develop spastic diplegic CP. This result was also mirrored in the infants born between 28 and 32 weeks' gestation ($n = 3$). In contrast, infants born between 32 and 37 weeks' gestation had a more varied presentation. One infant developed spastic diplegia, one spastic quadriplegia and two hemiplegia. Five of the term infants were diagnosed with hemiplegia, four with spastic quadriplegia, one triplegia and one diplegia.

Comparison of the different ethnicities showed they were evenly matched for Caucasian (76.2% vs 78%) and Aboriginal and Torres Strait Islander (19% vs 15%). However, when the results were compared to the last Queensland report two areas where considerably higher, male gender (71% vs 57%) and those identified as Aboriginal and Torres Strait Islander ethnicity (19% vs 6%). This is illustrated in Fig. 3.

4. Discussion

The prevalence of CP within the Townsville region from 2008 to 2012 was 1.8 per 1000 live births, which is lower than two per 1000 live births reported by the ACPR (Australian Cerebral Palsy Register Group, 2016). This lower prevalence of CP might be a result of specific neuroprotection measures introduced into clinical practice within the last decade. However, there was insufficient data to perform logistic regression on the antenatal treatment, complications of pregnancy, or type of delivery therefore, it is not possible to evaluate whether changes in perinatal practice contributed to the reduction in the rate of CP.

The main factors found to be associated with CP in this study were a gestational age of 28–32 weeks and birthweight less than 2500 g. Interestingly, 45% of the case group in this study were born preterm, with a mean gestational age of 35 weeks. The latest ACPR report has acknowledged a slight rise in gestational age within the preterm cohort with low birthweight remaining as a risk factor (Australian Cerebral Palsy Register Group, 2016). Logistic regression analysis in the current study showed that infants born between 28 and 32 weeks' gestation were ten times more likely to develop CP, whereas, extreme prematurity (less than 28 weeks) was not statistically significant. Oskoui et al. (2013) identified neonates born at 28–31 weeks as having a prevalence of 144.72 per 1000 live births compared to 111.8 per 1000 live birth for neonates born less than 28 weeks. However, potential reasons for this were not further discussed. More research is required to identify if there is an underlying pathophysiological reason why neonates born at 28–32 weeks' gestation are more likely to develop CP and to clarify whether it is simply a reflection of birth rate and survival for this group.

The mean birthweight for the case group was 2427 g classified

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