Cerebral Palsy: Cause, Risk Factors and Outcome in Birol and Sador Upazila of Dinajpur District

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To identify cause, risk factors and outcome of cerebral palsy a case-control study was done in Dinajpur sador and Birol Upazila of Bangladesh. Baby with cerebral palsy who attended at health camp in selected area was study case. Selected 200 cerebral palsy patients for the study. Factors associated with an increased risk of cerebral palsy identified antenatal and intrapartum and post natal risk factors.There were 106 (53%) boys and 94 (47%) girls. Hospital delivary were 20%, 51% had antenatal checkup regularly at antenatal clinic or obstetrician, 18% were Cissarian section and 82% were NVD. All were control bowel and blader. 50% were mentaly retarded. 33% hearing impairments, 37% speech defect. 2% blind. 30% squint and 43% had behavior change. 70% multiple born and 30% first baby of the parents.75% were preterm low birth weight and 25% were term baby. 90% experience convulsion one or more then one episode. All patients had delayed mile stone.10% mother reported they have infection(fever, rash). No history of blood transfusion, 2% mother smoker. 43% were prolonged lobour. all the baby have abnormal movement and posture. 15% had perinatal asphyxia. Birth trauma were 7%. post natal infections(meningitis) were 2%. Prenatal infection is responsible 9% in our study. premature low birth weight is responsible 52%in our study and compare to term and preterm babies 70% and 30% respectively . Multiple gestation is responsible 10% of all causes and compare to multiple gestation 75% than single gestation 25%. physical injury contribute a major role. A reduction in the rate of cerebral palsy in babies requires an integrated approach to management throughout the antenatal, intrapartum, and neonatal periods.

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Key words: Cerebral palsy, risk, cause, Dinajpur, Birol

Introduction

Two of every 1000 live-born children develop cerebral palsy (CP) in all age. Cerebral palsy is caused by damage to the motor control centres of the developing brain and can occur during pregnancy, during childbirth or after birth up to about age three. Resulting limits in movement and posture cause activity limitation and are often accompanied by disturbances of sensation, depth perception, and other sight-based perceptual problems, communication ability; impairments can also be found in cognition, and epilepsy is found in about one-third of cases. CP, no matter what the type, is often accompanied by secondary musculoskeletal problems that arise as a result of the underlying disorder.

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Improvements in neonatology (specialized medical treatment of newborn babies) have helped in reduction of the number of babies who develop cerebral palsy and increased the survival of babies with very low birth weights (babies which are more likely to have cerebral palsy). Cerebral palsy (CP) represents a group of disorders in the development of movements and posture, secondary to non-progressive changes in the developing brain of fetuses and children, which limits activities. This motor disorder is frequently accompanied by changes in sensation, cognition, communication, perception, behavior, and seizures.[1] The incidence of children who survive organic aggressions endured in the pre-, peri-, or post-natal period has not changed in the last decades in developed countries.[2] Evidence suggests that 70–80% of CP cases are due to prenatal factors and that perinatal asphyxia plays a relatively minor role (<10%). Some antenatal risk factors are repeatedly observed to be related to CP. Recently, intrauterine infection/inflammation with a maternal response (consisting of chorioamnionitis) and a fetal inflammatory response (consisting of funicitis or elevated interleukin-6 in fetal plasma) has been found to be related to white matter injury and CP Preterm birth is associated with a clear increase in risk of cerebral palsy.[1-5] Infants that are born with low birth-weight are higher more likely to develop cerebral palsy than infants who are born with a normal birth weight. The average prevalence of CP was approximately 3.3 per 1,000 or 1 in every 303 8-year-old children living in areas of Alabama, Georgia, Missouri, and Wisconsin.

CP prevalence varied by site, ranging from 2.9 per 1,000 8-year-olds in Wisconsin to 3.8 per 1,000 8-year-olds in Georgia;CP, on average, occurred 1-2 times more frequently among boys than among girls;Spastic CP was the most common type of CP, found among approximately 80% of children with CP and while 56% of children with CP were able to walk independently, 33% had limited or no walking ability. CP is often accompanied by disturbances of sensation, perception, cognition, communication, behaviour, epilepsy, and secondary musculoskeletal problems. An Excel was used to present and analyze the data.

Greater risk of CP with preterm deliveries (but since most deliveries happen close to term, most infants with CP (75%) are born after 36 weeks). There is a U-shaped association between CP and gestational age, where incidence of CP is increased in both preterm and postterm babies. The mechanism may be related to the physiological changes that trigger labour. Parturition is hypothesized to be partially related to fetal brain maturity, as fetuses with cerebral abnormalities tend to be delivered either preterm or postterm. JAMA. 2010 Sep 1;304(9):976-82.Intraventricular hemorrhage (IVH) is predominantly associated with prematurity and is due to fragility of developing blood vessels in the infant's brain. IVH may cause PVL or ischemia in other parts of the brain.Infections.Fetoplacental and uterine infection or inflammation can cause initiation of preterm labour, which can lead to CNS injury and CP. Underdeveloped fetal brains are more susceptible to inflammation and inflammatory cytokines. These cytokines are hypothesized to be responsible for the development of PVL. Chorioamnionitis is an infection of the chorion and amnion, the two membranes surrounding the developing fetus. It is the most frequently associated maternal infection in CP.

TORCHS is an acronym for perinatal infections: toxoplasmosis, other infections (varicella zoster, adenovirus, enterovirus), rubella, cytomegalovirus, herpes simplex virus, syphilis. TORCHS infections are

associated with approximately 5% of all CP cases.

Increases the risk of antenatal complications, such as preterm labour, growth restriction, low birth weight, and death of a co-twin. Death of a co-twin in utero has been shown to induce neuropathologic changes that can lead to CP in the surviving twin. Prevalence of CP in the surviving twin was found to be 15times higher than average. Twinning is the single strongest risk factor for the development of CP.

Thrombophilias can lead to placental vascular injury and clotting of the fetal vessels.Hemorrhage and preeclampsia (placental abruption, placenta previa, and other causes of third trimester bleeding) seem to lead to premature delivery, conferring the same risks for CP as a premature infant according to some evidence.

Perinatal asphyxia (10%) is commonly associated with CP. CP is associated with complicated labour and delivery, but there is not a clear association between CP and the quality of perinatal care. Despite the advancement of prenatal and obstetrical care in the past 30 years, the incidence of CP has remained constant. This may be due to increased survival rates of premature and low birth weight babies. Non-accidental injury, Head trauma, meningitis and encephalitis are also important cause of cerebral palsy. This study was designed to identify cause, risk factors and outcome of cerebral palsy

Methods

Study Design - a population based retrospective study

Study population - population included all CP children who attended in our medical camp in Sador and Birol upzila

Data collection and study factors - study participants were identified by the attending physician.

Once all children diagnosed with cerebral palsy at the different centres by a paediatrician according to history and clinical feature.

Statistical methods - in the study sample as a whole, the associations of cerebral palsy with cause and risk factors the data analyses by Excel

Results

Boys were 106 (53%) and Girls are 94(47%). All the babies experience abnormal posture and movement. Behavioral change 43%(84). squint 30% compare to 43%.[22] 50% were mentally retarded.33% were hearing imparment.75% were multiple birth which are very similar to many studies [24,26,27.] 15% were perinatal asphyxia. only 2% were post natal infections(meningitis)

Certain infections in the mother, including rubella (German measles), cytomegalovirus (a usually mild viral infection) and toxoplasmosis (a usually mild parasitic infection) can cause brain damage and result in cerebral palsy. Recent studies suggest that maternal infections involving the placental membranes (chorioamnionitis) may contribute to cerebral palsy in full-term as well as preterm babies . A 2003 study at the University of California at San Francisco found that full-term babies were four times more likely to develop cerebral palsy if they

were exposed to chorioamnionitis in the womb. Our study preterm responsible for 57%. Reproductive/urinary tract infections also may increase the risk of preterm delivery, another risk factor for cerebral palsy.

Premature babies who weigh less than 3 1/3 pounds are up to 30 times more likely to develop cerebral palsy than full-term babies. Many of these tiny babies suffer from bleeding in the brain, which can damage delicate brain tissue, or develop periventricular leukomalacia, destruction of nerves around the fluid-filled cavities (ventricles) in the brain. This study show 75% is responsible compare to 25% term babies.

It was widely believed that asphyxia (lack of oxygen) during a difficult delivery was the cause of most cases of cerebral palsy. In this study 15% were perinatal asphyxia. The ACOG/AAP report shows that fewer than 10 percent of the types of brain injuries that can result in cerebral palsy are caused by asphyxia. In our study this figure is 15%.

A pigment called bilirubin in the blood, occasionally becomes severe. Without treatment, severe jaundice can pose a risk of permanent brain damage resulting in athetoid cerebral palsy. Our study kernicterus contributed 7%.

Acquired cerebral palsy- About 10 percent of children with cerebral palsy acquires it after birth due to brain injuries that occur during the first two years of life. The most common causes of such injuries are brain infections (such as meningitis). Our study contributed 2% and head injuries. This study findings 7% birth trauma. Another most important infection is post natal infection like meninigitis, our study shows 2% CP due to meningitis

Multiple babies -The risk of cerebral palsy increases with the number of babies sharing the uterus. if one or more of the babies die the chance that survivors may have CP increase. Present study contributed 10% multiple birth. Compare to multiple 70% than 30% in single born in our study.

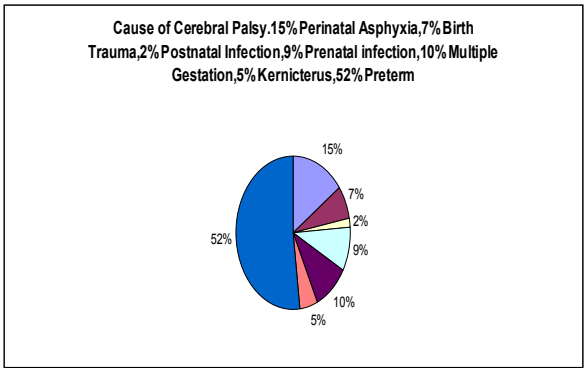

Figure 1. History of labour

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Figure 2. History of Antenatal checkup

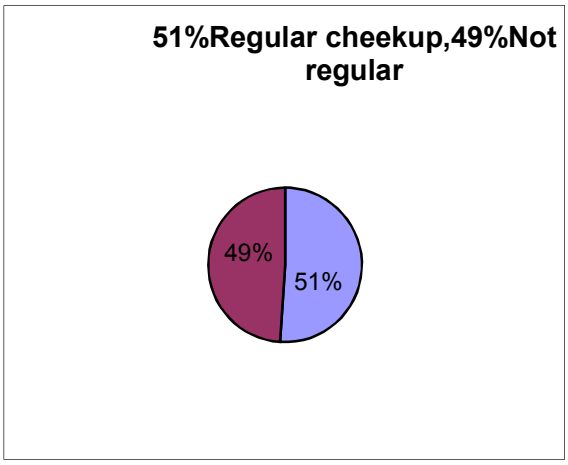

Figure 3. Percentage of convulsion

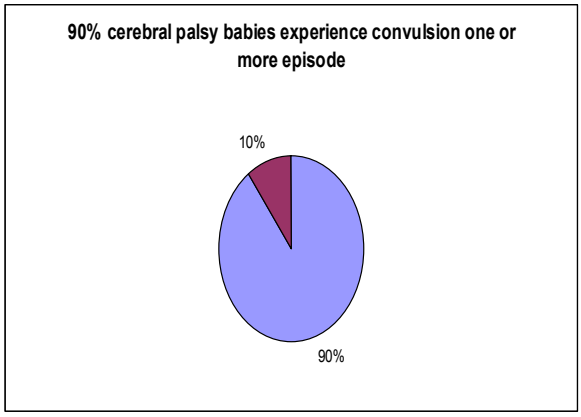

Figure 4. Percentage of convulsion

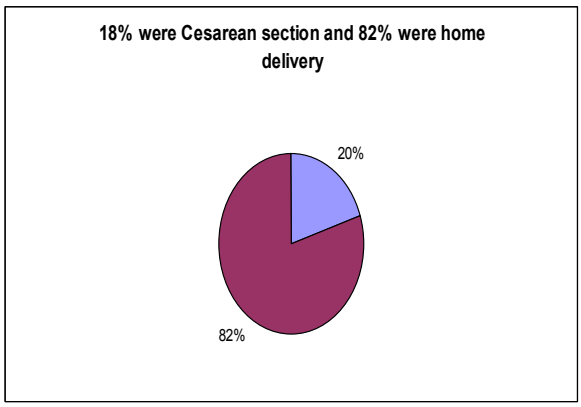

Figure 5. Modes ofdelivery

Table I: Complications of cerebral palsy

Mentaly retarded	50%
Hearing impairment	33%
Speech defect	37%
Blind	2%
Squint	30%
Behavior	43%
Convulsion	90%

Discussion

Although the incidence of CP has remained constant in developed and developing countries, speech defect are 50% which are similar to in gram 1966 study.[24] In our study, we tried to demonstrate to considered risk factors for the development cerebral palsy.[15] When Hagberg et al.[9] presented their epidemiologic data on the incidence and etiology of CP, The predominance of perinatal etiology in pre-term births (54%) in comparison with full term births (28%). Prenatal etiology was more common in full term births(33%) than in pre-term births (8%). A hypothesis concerns infection, and several studies have shown associations between neonatal sepsis and [17-19] cerebral palsy.[32] Our results support this hypothesis as neonatal sepsis and cerebral palsy were strongly associated even after other potentially confoundingneonatal complications. These findings suggest a role for several neonatal complications in the aetiology of cerebral palsy in preterm babies.[31,32] The difficulty in interpreting these findings, however, lies in determining which neonatal factors are causes of cerebral palsy and which are consequences of earlier disturbances in the antenatal and intrapartum periods and already part of the outcome. Some neonatal factors, such as transfusion, may be markers of severity of neonatal illness or may be the consequence of a disabling cerebral haemorrhage. Antenatal and intrapartum risk factors for cerebral palsy in very preterm babies found a strong association between maternal infection and, in particular, chorioamnionitis and an increased risk of cerebral palsy. It is possible, therefore,

that the origins of cerebral palsy lie in the neonatal period for a large proportion of very preterm babies. In addition, because of the design of a case-control study it is not possible to predict the timing of cerebral damage in relation to the insult and it is possible that the ischaemia associated with chorioamnionitis is not manifest until the neonatal period and may occur only if the baby suffers an additional further insult in the neonatal period. Our finding that the sequence of maternal infection followed by neonatal sepsis was strongly associated with cerebral palsy lends some strength to the theory of a continuum of insults in the pathogenesis of preterm cerebral palsy. However, this sequence of events affected only a small proportion of the study population. Perinatal asphyxia was previously considered the major cause of cerebral palsy, whereas current knowledge suggests that prenatal causes are most important.[1, 2]

Figure 6. Patient of cerebral palse

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Recommendations
In many cases, the cause of cerebral palsy is not known, so there is nothing that can be done to prevent it. In spite of improvements in the care of pregnant women and sick babies, the number of babies with cerebral palsy seems to be increasing. This is due in part to the survival of an increasing number of very premature babies, who are at high risk of cerebral palsy.

However, some causes of cerebral palsy have been identified and cases of cerebral palsy that result from them often can be prevented. Women can be tested for immunity to rubella before pregnancy and be vaccinated if they are not immune. Babies with severe jaundice can be treated with special lights (phototherapy). Head injuries in babies, a significant cause of cerebral palsy in the early months of life, often can be prevented. Routine vaccination of babies (with the Hib vaccine) prevents many cases of meningitis, another cause of brain damage in the early months. A woman can help reduce her risk of preterm delivery when she seeks early ideally starting with a pre-pregnancy visit and regular prenatal care and avoids cigarettes and illicit drugs

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