Role of Paediatrician during Labour and Delivery period- Golden moments for Primary Prevention of Cerebral Palsy

Dr.R.Premalatha, Professor & Head, Pediatrics, BMCRI, Bangalore

There's an adage that an ounce of prevention is worth a pound of cure – this is especially true in cases of cerebral palsy because there is no cure. Hence new knowledge regarding causative factors and pathogenesis is required, so that preventive strategies can be planned and implemented. Often the cause of cerebral palsy is not known, and nothing can be done to prevent it. However, some important causes of cerebral palsy can be prevented in many cases, including premature birth, low birth weight, infections, and head injuries.

Prior epidemiologic studies suggested that 69% of cerebral palsy were the result of factors occurring prior to the onset of labor, whereas only 29% were associated with intrapartum events and only 5% could be exclusively linked to intrapartum factors. Major risk factors included preterm and post term birth, fetal growth restriction, pre eclampsia, peri-natal infections, chorioamnionitis, maternal thyroid disease, placental abnormalities, infertility treatment (with resultant multiple gestations and prematurity).

Recent MRI data in neonatal encephalopathy has improved our understanding of etiology and timing of insult to growing brain. By using MRI (24-96 hours of birth) to reassess intrapartum factors, scientists now suggest a far more common role for peripartum and intrapartum factors (1). MR spectroscopy measures the presence and relative abundance of specific molecular markers of neural injury (eg, lactate-to-N-acetylaspartate ratios) over specific locations in the brain (eg, the thalamus). While early MRI assists in timing insults, repeat MRI at 10 days (7–21 days) can characterize the extent of lesions and provide prognostic information.

Recent studies suggest that majority of term Neonatal encephalopathy (NNE) cases are in fact acute and due to potentially avoidable causes, amenable to quality improvement such as training of staff in fetal surveillance in labor (2). Two primary patterns of acute neural injury have been observed that are associated with CP: a) basal-ganglia-thalamus injury and b) watershed or borderline cortical white-matter injury (3).Animal studies suggest that the former pattern is associated with acute, near-total "asphyxia" and the latter with a less severe but more prolonged asphyxia process. The impact of hypoxic-ischemic insults on the fetal and neonatal brain is dependent on not only the severity and duration of oxygen deprivation but also gestational age. In

addition, prior chronic hypoxia and nutrient deprivation due to uteroplacental and vascular pathology have strong link to fetal growth restriction and CP.

Genetic susceptibility factors such as polymorphisms for cytokine genes may also exacerbate inflammation-associated neural damage following HIE, helping to explain why low Apgar scores and umbilical artery pH values are such poor predictors of eventual outcome. Also, most of the damage due to hypoxic-ischemic events occurs following reperfusion. Hypothermia therapy blunts reperfusion injury to growing brain. Absence of discrete findings on early MRI in an infant with apparent NNE should suggest genetic causes (eg. chromosomal micro deletions and inborn errors of metabolism) and can trigger timely and potentially lifesaving interventions.

Role of obstetrician in prevention of CP

1. Before Pregnancy:
Prepare women for healthy pregnancy by scheduling a pre-pregnancy visit, so they can be advised regarding the following.

- Avoid using cigarettes, alcohol and illicit drugs during pregnancy; these increase the risk of premature delivery.
- Unnecessary exposure to antenatal x-rays should be avoided.
- Prevent TORCH (toxoplasmosis, rubella, cytomegalovirus, herpes simplex) infections by immunization where possible and institute quick treatment when encountered.
- Folic acid supplementation to at risk women

2. During antenatal period:
Prevention and treatment of the following conditions

- Infections should be screened for and treated early like bacterial infections, urinary tract infections, chorioamnionitis (Escherichia coli, Group B streptococcus and methicillin-resistant Staphylococcus aureus), viral infections (chickenpox) and TORCH infections.
- Premature birth – Preterm infant faces an increased risk of developing cerebral palsy. The risk is even greater if it is associated with low birth weight. Prevention of preterm birth by specific approaches and when preterm delivery is imminent use of glucocorticoids to enhance lung maturity helps prevent CP. Magnesium sulphate infusions may reduce the incidence of CP in premature infants. The mechanism of this therapy is due to a neuroprotective effect of antenatal magnesium sulphate. Preterm neonates are also at risk of infection.
- Multiple births -Twins, triplets and other multiple births have a higher risk for CP, especially if a baby’s twin or triplet dies before birth or

shortly after birth. Some, but not all of this increased risk is due to the fact that children born from multiple pregnancies often are born early or with low birth weight, or both.

- Assisted reproductive technology (ART) infertility treatments— Increased risk of CP is explained by preterm delivery or multiple births, or both; both preterm delivery and multiple births are increased among children conceived with ART infertility treatments. Can be prevented by reducing the chance of a multiple pregnancy (twins, triplets, or more), by transferring only one embryo at a time.
- Medical conditions of the mother- Hypertension, diabetes, severe anemia, chronic illnesses, thyroid problems, iodine deficiency, intellectual disability, or seizures have a slightly higher risk of having a child with CP.
- Blood incompatibility – Testing for Rh factor and treating with Rh immunoglobulin.
- Screening and treatment of asymptomatic bacteriuria, prevention of chorioamnionitis with prophylactic antibiotics in premature rupture of membrane
- Antiplatelet drugs to prevent preeclampsia
- 17α-progesterone caproate, and cervical cerclage for women with previous preterm birth and short cervix.
- Fetal distress – Due to placental abruption, preeclampsia, eclampsia, uterine rupture or multiple pregnancies. An emergency LSCS may be required in these cases to prevent HIE.
- Genetic disorders – Recent research indicates that genetic factors play a major role in CP. Hence prenatal testing and counseling are important.
- Identification of high risk pregnancies and in utero transfer to tertiary care centers

3. During Birth: Role of Obstetrician

As recent MRI data suggests intra partum and peripartum events are more often causative factors for CP, increased attention must be placed on optimizing intrapartum monitoring(2,4). Intrapartum event as a cause of cerebral palsy is more likely if significant fetal acidosis (such as pH < 7.0) and neonatal encephalopathy are observed. While these events might be the result of intrapartum hypoxia, they might also be the result of fetal infection.

Intrapartum asphyxia can some times be prevented by avoiding some "sentinel" events and by responding appropriately to cardiotocograph (CTG) anomalies. The (CTG) is a screening tool that is used to assess fetal well-being during labour. Identification of the possibility of asphyxia and taking timely and appropriate action based on the findings may help prevent birth asphyxia.

Additional factors, which help in identifying asphyxia, include

- A well-documented partogram
- Complete analysis of fetal blood sampling for pH or lactate
- Placental pathology to detect occult thrombotic processes affecting the fetal circulation, patterns of decreased placental reserve and adaptative responses to chronic hypoxia, and evidence of chorioamnionitis

Complications during labor and delivery causing HIE include:

- Breech delivery and Shoulder dystocia –cerebral palsy is four times more likely to occur following breech delivery.
- Umbilical cord compression / prolapse – Compression of the umbilical cord can occur during uterine contractions leading to fetal hypoxia or anoxia.
- Placenta previa or Placental abruption

Delivering these fetuses by cesarean sections may prevent CP.

4. Role of Pediatrician during labor and delivery:

Pediatrician should monitor the growth of the fetus along with the obstetrician from conception and discuss intervention required if there are high risk factors. For example, a simple intervention like correcting anemia in a pregnant mother will improve fetal wellbeing greatly. High-risk mother should be identified early and transferred to delivery units, which are attached to Level 2 or Level 3 NICU. Pediatrician should anticipate neonates who may require extensive resuscitation and be prepared.

Most newborns require no/minimal assistance. Only10% requires assistance to begin breathing at birth and 1%requires extensive resuscitative measures.

First Golden Minute Project: emphasizes skill based training on following steps:

- Anticipation
- Presence of skilled personnel
- Adequate preparation of the resuscitation station and equipments
- Accurate evaluation
- Prompt initiation of support

1. Initial steps, which are required for all neonates

•Provide warmth
•Head position in " sniffing position"
•Clearing the airway
•Drying the baby
•Tactile stimulation for breathing

2. Ventilation-required in 10% of neonates

3. Chest compressions-required only in 1% of neonates

Rarely administration of epinephrine & /or volume expansion is required.

Golden minute

•In <30 seconds: complete initial steps
Warmth, Drying, Clear airway if necessary, Stimulate
•30-60 seconds: assess 2 vital characteristics
Respiration (apnea/gasping/labored/unlabored) and
Heart rate (<100/>100bpm)
By <60 seconds of birth
If gasping/apnea or if heart rate<100 beats per minute
Start PPV (positive pressure ventilation) and
Spo2 monitoring by pulse oximeter
(Simultaneous evaluation of 3 vitals-HR, respiration and oxygenation Status)
If HR remains <100 beats per minute
•Endotracheal intubation
•Chest compression
•Drugs

Neonates requiring extensive resuscitation may be at risk of HIE and they may benefit from hypothermia therapy. Using room air for resuscitation also reduces oxygen induced cerebral damage.

Summery

Everyone has a role in preventing cerebral palsy

After a child is diagnosed with cerebral palsy, a parent is likely ask, "How did this happen to my child?" and "How could this have been prevented?" Preventing it requires the involvement of the medical and research communities, as well as the government.

Government's role in cerebral palsy prevention centers on training of health workers, nurses and doctors in emergency obstetric care and essential newborn care & neonatal resuscitation. Setting various levels of neonatal care units with equipments and skilled personnel has provided much required newborn care services in all remote areas. Providing transport in the form of 108 ambulances has benefitted many high-risk mothers and neonates requiring tertiary care to be transferred to such facilities in time. Collecting data electronically from all neonatal care units across the country and analyzing the causal factors through its various agencies, including the National Health Mission and UNICEF is on going and will provide much needed information regarding preventive factors. Data compiled by the government on preventative factors will help in allocating funds and resources to vital areas in future.

Research communities will help in formulating preventive strategies and labor

room management guidelines. Adherence to these guidelines may protect the Obstetricians and Pediatricians from medico-legal threat and “cerebral palsy lawsuits”, apart from preventing cerebral palsy.

References:

1. Gunn AJ1, Bennet L. Timing of injury in the fetus and neonate. Curr Opin Obstet Gynecol. 2008 Apr; 20(2): 175-81

2. Dr Lynn C. Sadler, MPH, Prof Cynthia M. Farquhar, MD, Ms Vicki L. Masson, MPH, Dr Malcolm R. Battin, MPH On behalf of the Neonatal Encephalopathy Working Group. Contributory factors and potentially avoidable neonatal encephalopathy associated with perinatal asphyxia

Presentations: A summary of the findings of this study were presented as a poster presentation at the RCOG/ RANZCOG World Congress 2015 in Brisbane Australia 12-15 April and at the PMMRC Conference 2015 in Wellington, New Zealand 17 June. Published Online: December 23, 2015

3 Jaw TS1, Jong YJ, Sheu RS, Liu GC, Chou MS, Yang RC. Etiology, timing of insult, and neuropathology of cerebral palsy evaluated with magnetic resonance imaging. J Formos Med Assoc. 1998 Apr; 97(4): 239-46.

4. Cowan F1, Rutherford M, Groenendaal F, Eken P, Mercuri E, Bydder GM, Meiners LC, Dubowitz LM, de Vries LS. Origin and timing of brain lesions in term infants with neonatal encephalopathy. Lancet. 2003 Mar 1; 361(9359): 736-42.