Early premature rupture of membranes: A silent cause of Cerebral Palsy

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INTRODUCTION

- Along with acquired causes, many genetic causes of cerebral palsy (CP) have been recently described.
- When genetic work up is negative and there are no obvious acquired causes, prenatal counselling becomes difficult.
- Premature rupture of membranes (PROM) when prolonged (i.e., > 12-24 hours), by causing chorioamnionitis with or without premature delivery and predisposing to perinatal asphyxia, is associated with CP.
- However, some babies are born before 12 hours of rupture of membranes (Early PROM). These babies cry after birth and are not usually admitted in NICU. The parents here report a normal perinatal history.
- These babies mostly present with milder dyskinetic or mixed (dyskinetic/spastic) type of cerebral palsy that is associated with paucity of white matter, ventriculomegaly and rarely periventricular leukomalacia like picture on MRI brain.
- This pattern is not well described in literature and many pediatricians and neurologists are not aware of this.

OBJECTIVES

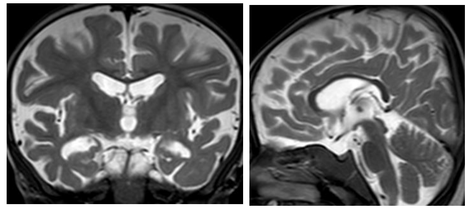
1. To characterize the cerebral palsy that is associated with premature/ early rupture of membranes which is less than 12 hours both clinically and neuroimaging wise
2. To know the response to treatment

MATERIAL AND METHODS

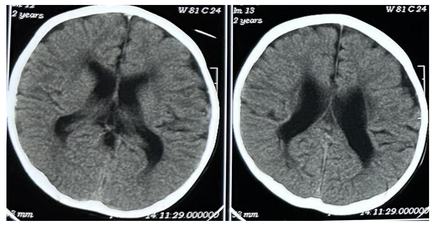
- Retrospective review of charts of children with CP who had a normal birth history and negative genetic evaluation. Parents were interviewed in detail for presence of PROM and those who had rupture of membranes of less than 12 hours before the onset of labour pains were labelled as early PROM.
- The clinical and neuroimaging features of these children was analysed.
- The clinical follow up of these children for at least 2 years after enrolling in the study was done

RESULTS

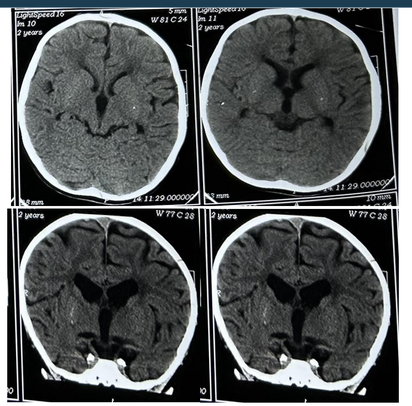
- We had four children fulfilling our study criteria in the last 4 years.
- Age ranged from 2 to 5 years. All had cried after birth.
- None were admitted in NICU.
- Mothers reported PROM from 3 to 12 hours.
- All presented mild mixed (spastic and dyskinetic) type of cerebral palsy.
- Neuroimaging showed paucity of white matter, thin corpus callosum, ventriculomegaly and rarely periventricular leukomalacia. CT scan of the brain in two children showed mineralising angiopathy. Cognition was well preserved.
- Extensive work up like screening for inborn errors of metabolism (IEMs) and whole exome sequencing was negative.
- Follow up for 2-3 years did not show any progression/ worsening confirming the clinical diagnosis as CP and not an undiagnosed genetic/metabolic disease.



MRI: Coronal and saggital sections of brain showing thin corpus callosum



CT: Axial sections of brain showing paucity of white matter and periventricular leucomalacia



CT: Axial and coronal cuts shows mineralizing angiopathy

CONCLUSIONS

1. Early premature rupture of membranes (PROM < 12 hours) can predispose to mild cerebral palsy of spastic or mixed type.
2. These babies do not have history of NICU admission.
3. MRI resembles genetic spastic paraplegia with thin corpus callosum, ventriculomegaly and periventricular leukomalacia.
4. Few of them show mineralising angiopathy also on computed tomography

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18th ICNC 2024 Footprints to the Future
INTERNATIONAL CHILD NEUROLOGY CONGRESS
Cape Town, South Africa | May 6-10, 2024