Commentary: Saying hi to HIE: Ocular signs, visual, and general developmental outcome in Indian children with radiologically proven periventricular leukomalacia

Neonatal hypoxic ischemic encephalopathy (HIE) is an acute, nonstatic encephalopathy caused by intrapartum or late antepartum brain hypoxia and ischemia. The patterns of brain injury from HIE depends on the child's age, severity of hypoxia, and duration of hypoxia.[1] The pattern of injury in a preterm born is different from a term baby since it is determined by the configuration of vascular supply to the brain. In term infants, the watershed areas of brain lie at the interfaces between the major cerebral arteries, which are the primary vascular supply of the brain. Hypoxic-ischemic injury in this period produces watershed infarctions in the parietooccipital and parasagittal cortex, resulting in cortical visual loss.[1] While in the developing brain, between 27th and 34th week of gestation, cortex and the underlying white matter receive their blood supply from the branches of vessels seen on the cerebral hemispherical surfaces. The watershed zone of vascular supply lies within the periventricular white matter. Hence, in preterm children, hypoxic ischemic injury to the developing brain results in injury to the subcortical white matter.[1] Thus, the magnetic resonance imaging (MRI) picture in preterm HIE is predominated by periventricular leukomalacia (PVL).[2]

As described by the authors HIE/PVL is commonly associated with ocular morbidities.[3] Optic disc changes, nystagmus, strabismus and ocular motor deficits, visual field defects, and functional deficits (acuity, assimilation, attention, apraxia) are commonly associated in children with asphyxial insult in the perinatal period.[4] PVL is characterized by optic nerve hypoplasia or pseudoglaucomatous optic discs.[5] Esotropias are more common in periventricular leukomalacia, and they may be associated with latent nystagmus, dissociated vertical deviation.[6] Dyskinetic strabismus, an entity in which the exotropia may alternate to esotropia with variable deviations, is reported to be seen in cases with PVL.[4] Even though the authors have reported nystagmus in one-third of their patients, it is generally rare in these children since an intact geniculostriate pathway is a prerequisite for the development of congenital nystagmus.[7] Visual field assessment is extremely challenging in these children, yet it is reported that generalized restriction of visual fields with a reduced mean visual field has been found in cases with perinatal asphyxia.[4] Central scotomas may be seen in cases with bilateral occipital lobe hypoxic-ischemic injuries, and the child may prefer to look at objects eccentrically in order to have better vision by avoiding the area of scotoma.[6] Cortical visual impairment is the most important sequel of perinatal asphyxia and an important cause of visual dysfunction in these patients.[4]

The grading system, used by the authors[3] to grade MRI features is a gross method and does not account for the subtle changes that occur in white matter, corpus callosum and cortical grey matter. Cioni *et al*.[8] have provided an extensive method to categorize the MRI features in these patients. They found the degree of visual impairment well correlated with the MRI characteristics. They also found that vision plays a major role in early motor and cognitive learning, and so the neurodevelopmental outcome is retarded. Similarly Tinelli *et al*.[9] observed a significant correlation between visual acuity and several features of brain damage such as the thinning of corpus callosum, the subscore of global lesion classification assessing the size of cystic area, and the atrophy index.

Table 1: Comparison of clinical grades and MRI scores

Clinical grade	MRI score
HIE Grade 1	8.6±1.51
HIE Grade 2	9±1.89
HIE Grade 3	14.5±2.12

Table 2: Comparison of clinical grades and MRI grades

MRI grade	Visual acuity
MRI Grade 1	0.91±0.31
MRI Grade 2	1.22±0.04
MRI Grade 3	1.13

As a subset of a larger study conducted at our center, the MRI scores of these children were compared with clinical severity[10] as well as visual acuity. Twenty-one children had neuroimaging records which was analyzed and the lesions were scored as per grading provided by Cioni *et al*.[8] Thirteen children had MRI grade 1 – mild impairment. Six children had MRI grade 2 – moderate impairment. Two children had MRI grade 3 – severe impairment. We found that there was a statistically significant relationship between the severity of lesions in MRI and clinical severity of HIE [Table 1]. The visual acuity was assessed on Cardiff visual acuity cards. Out of these 21 children, one each in grade 2 and grade 3 had poor visual fixation and visual acuity assessment could not be done. The mean visual acuity was 0.99 ± 0.26 [Table 2]. We found a statistically significant relationship between the MRI scores and the visual acuity ($r = 0.708$, $P = 0.01$).

To conclude, children with HIE have various ophthalmic features and CVI is a common cause of visual dysfunction. MRI scoring may help in prediction of visual acuity in these cases. Long-term and large-scale follow-up studies need to be conducted on the correlation between imaging and future visual outcome.

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