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Late Presentation of Hypoxic Injury of Brain in an Infant

Pradeep Raj Regmi,[1] Aalok Kumar Yadav,[1] Bibek Koirala,[1] Shreelal Yadav,[2] Isha Amatya[3]

[1]Tribhuvan University Teaching Hospital, Kathmandu, Nepal, [2]Malangawa Hospital, Sarlahi, Nepal, [3]Nepal Health Research Council, Ramshahpath, Kathmandu, Nepal.

ABSTRACT

Perinatal asphyxia is one of the leading causes of hypoxic-ischemic encephalopathy. In a developing country like Nepal, home delivery is the leading cause of perinatal asphyxia. Neuroimaging remains the diagnostic modality of choice. We present a case report of a 10-month-old infant who presented to the pediatric Out-patient-department with complaints of being unable to hold his head and unable to sit without support. Detailed history, physical examination, and developmental assessment along with lab investigation flash visual evoked potentials and Magnetic Resonance Imaging of the brain was performed. Hypoxic ischemic injury has common five types of imaging patterns in neonates. There are a few imaging differentials to be considered while evaluating the case for hypoxic injury. Clinicians and radiologists must go hand in hand to narrow down the possibilities which can fasten the treatment thereby decreasing morbidity and mortality.

Keywords: Hypoxic ischemic encephalopathy, Infant, Magnetic Resonance Imaging

INTRODUCTION

Hypoxic Ischemic encephalopathy (HIE) is a syndrome of disturbed neurological function manifested by difficulty in initiating and maintaining respiration, abnormal muscle tone and reflexes, subnormal level of consciousness and often seizures. HIE is a serious birth complication affecting full-term infants; 40% to 60% of affected infants die by 2 years old or have severe disabilities[1]. Most underlying pathologic events of HIE are a result of impaired cerebral blood flow and oxygen delivery to the brain with resulting primary and secondary energy failures.[1] By the age of 2 years, up to 60% infants with HIE will die or have severe disabilities including mental retardation, epilepsy, and cerebral palsy (CP).[1,2] Neuroimaging with ultrasonography (US), computed tomography (CT) and magnetic resonance (MR) imaging has become increasingly valuable in the work-up of particularly MR imaging- has the potential to play a significant role in diagnosis and early intervention in case of HIE[3,4].

CASE PRESENTATION

A 10-month-old male infant presented to our hospital with chief complaints of unable to hold head and unable to sit without support since birth. He was born from 21 years primi-gravida female at home. Mother was healthy with no significant past and present medical history. Her antenatal visits were regular and took all multivitamins during pregnancy. There was no history of fever, rash, pregnancy induced hypertension, Diabetes and radiation exposure during pregnancy. Baby was delivered by spontaneous vaginal delivery at home. Birth weight was 2.75 kgs. He didn't cry immediately after birth for which he was admitted in Neonatal intensive care unit (NICU) for 5 days. History of intubation during admission was present and baby had seizure for which he was prescribed anti-epileptic medication for 3 months. No imaging investigations were done due to financial constraints and unavailability of imaging modalities in that remote health facility. He was immunized according to Nepal immunization protocol. The baby was unable to speak, give social smile and unable to hold neck since birth.

Physical examination and Imaging findings

Head circumference (HC): 38 cm (HC for Age : < -3 Z score), Height (HT): 63cm (HT for Age : < -3 Z score), Weight (WT): 7.5kg (WT for Age: 0 to -2 Z score), WT for HT: 0 to +1 Z score

Flash visual evoked potentials showed bilateral delayed

Correspondence: Dr Pradeep Raj Regmi, Department of Radiology, Tribhuvan University Teaching Hospital/Maharajgunj Medical Campus, Maharajgunj, Kathmandu, Nepal. Email: pradeep.iom@gmail.com/pradeepregmi@iom.edu.np

P2 latency with bilateral normal N2-P2 amplitude, suggestive of bilateral conduction defect (Image 1). With all these findings and clinical history, baby was suspected to be suffering from hypoxic ischemic encephalopathy- for which MRI was requested.

Plain MRI of the Brain shows gross atrophy of brain parenchyma with severe thinning of cortex, numerous gyri and deepened sulci in bilateral cerebral hemisphere with thinning of corpus callosum and cingulate gyrus. There was relative sparing of bilateral thalamus and cerebellar hemispheres. Encephalomalatic changes in bilateral fronto-parieto-occipital lobes - findings consistent with sequelae of chronic hypoxic ischemic encephalopathy (Images 2, 3, 4). Baby was discharged home with symptomatic care and advice for need of physiotherapy.

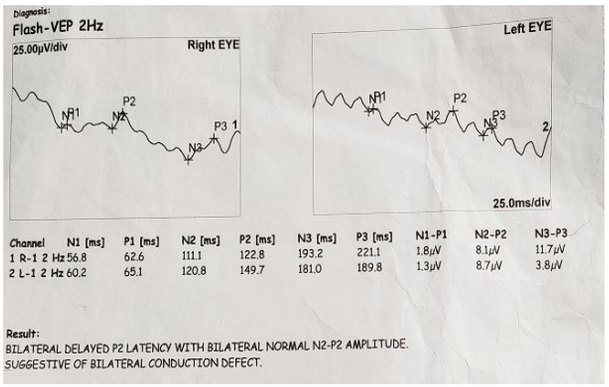

Image 1. Flash visual evoked potentials showed bilateral delayed P2 latency with bilateral normal N2-P2 amplitude.

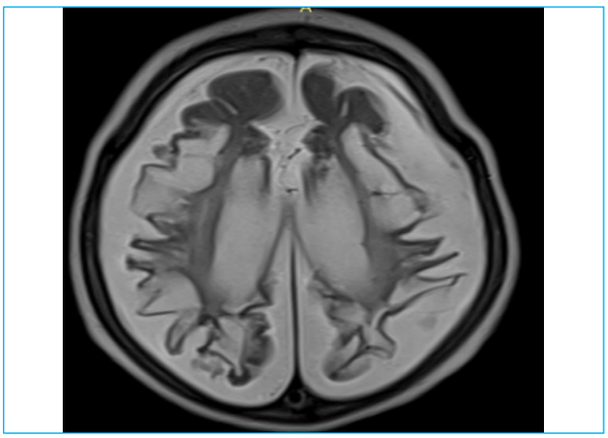

Image 2. Axial T2 MRI shows gross atrophy (shown by white arrows) of the bilateral cerebral hemispheres involving bilateral frontal, parietal and occipital lobes along with loss of white matter. Multiple variable sized cystic spaces seen in the bilateral periventricular white matter suggesting encephalomalatic/gliotic changes.

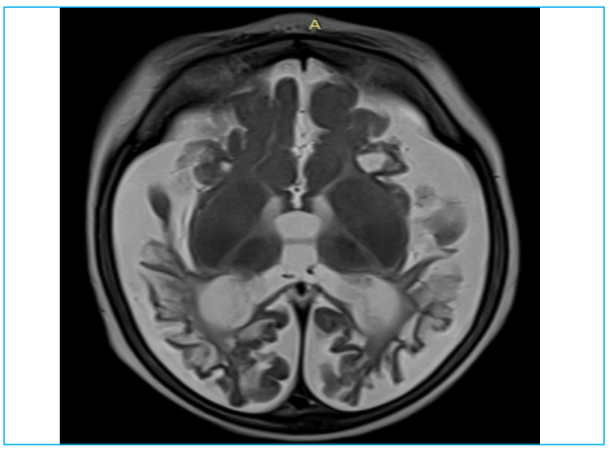

Image 3: Axial T2 MRI shows atrophy of bilateral occipital lobes along with loss of white matter. Bilateral basal ganglia are spared (shown by white arrow)

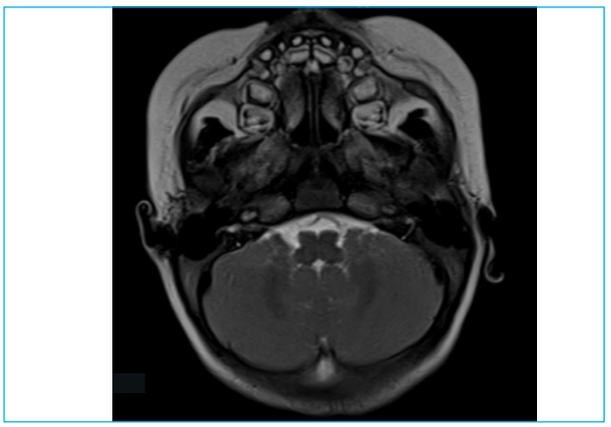

Image 4: Axial T2 MRI shows normal posterior fossa; medulla and cerebellum (shown by white arrow).

DISCUSSION

The hypoxic-ischemic event can be caused by multiple events, but ultimately, brain injury occurs because of impaired cerebral blood flow and oxygen delivery to the brain[1]. The ischemic insults depending on severity and duration of insult and the age of the neonate makes particular brain areas selectively vulnerable to damage. Areas of high metabolic demand containing a high concentration of excitatory amino acid receptors, such as the deep gray matter and perirolandic cortex are commonly damage in acute severe ischemia.[3] In comparison to this, less severe but more prolonged ischemic insults affect the cortical and sub-cortical watershed regions. Perilolandic involvement remains the most common area involved in the patients of cerebral palsy.[4]

MRI is more sensitive for the evaluation of Hypoxic-ischemic injury (HII). MR imaging protocols should

include, at a minimum, axial T1- and T2-weighted sequences, diffusion-weighted sequences. Fluid attenuated inversion recovery images can be useful in evaluating adults and older children but are of limited use in detecting edema in newborns. If MR imaging performed in the first 24 hours is negative, a repeat examination performed at 2-4 days may help rule out the possibilities of delayed injury, given that MR imaging may occasionally produce a false result in the acute period. Although some institutions routinely performed MR spectroscopy for suspected neonatal brain injury, this modality is reserved for situation in which diffusion weighted imaging findings are normal but the clinical suspicion for HII remains high.[3]

The major patterns of perinatal hypoxic injury can be 1. Basal ganglia-thalamus (BGT) pattern, 2. Watershed predominant pattern of injury, 3. Severe involvement of subcortical white matter and cortex sparing the deep periventricular white matter (white cerebrum), 4. Lesions restricted to periventricular white matter and 5. Perinatal arterial ischemic stroke (PAIS), perinatal hemorrhagic stroke (PHS) and sinovenous thrombosis.[5] BGT pattern is present in acute total asphyxia in the cases like ruptured uterus, placental abruption or prolapsed cord. Absence of normal high signal intensity of posterior limb of internal capsule can be present in adverse sequelae. Diffusion weighted imaging can show earliest changes in basal ganglia and thalami. Watershed pattern can be seen in the prolonged partial hypoxia. The vascular watershed zones are involved. Severe motor impairment is uncommon in this group of infants, and they are often considered to have an early normal outcome. In white cerebrum pattern, there is diffuse increased signal of white matter along with relative sparing of the central grey matter. Later, diffuse cystic encephalomalatic changes prevails. The prognosis is fatal in these type. The fourth type can show punctate periventricular white matter signal intensity showing diffusion restriction especially in the preterm infant. The fifth pattern presenting with PAIS, PHS and thrombosis usually has eventful obstetric history like instrumental delivery or emergency delivery. It is usually related to prenatal asphyxia in a neonate presenting as seizure or neonatal encephalopathy.[5]

Preterm infants usually show periventricular border zone of white matter injury due to ventriculopetal vasculature whereas term neonate show more peripheral border zone including subcortical white matter as well as parasagittal involvement due to ventriculofugal vasculature (Image 5).[6]

One of the most important differentials for HIE in imaging is neonatal hypoglycemia. Though, both can exist together. However, neonatal hypoglycemia has predilection for occipital and parietal lobes (Image 6).[7]

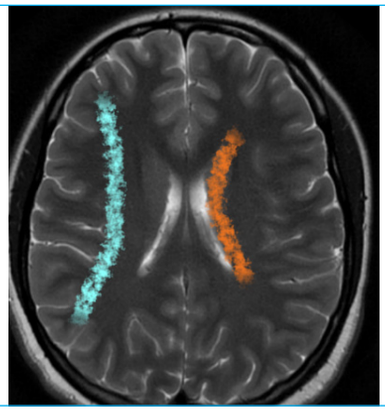

Image 5. T2 axial MRI shows different zonal predominance in HIE; subcortical border zone in term neonate (sea green color on right cerebral hemisphere) and periventricular border zone in preterm neonate (orange color on left cerebral hemisphere)

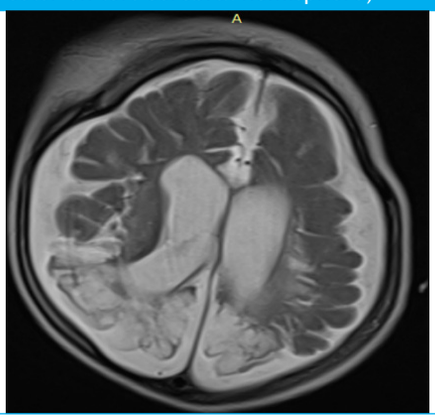

Image 6. T2 axial MRI image shows white matter high signal intensity along with atrophy involving bilateral parieto-occipital lobes (white arrows)

CONCLUSIONS

Perinatal asphyxia is common cause of hypoxic ischemic encephalopathy and home delivery being leading cause of perinatal asphyxia in underdeveloped and developing country. Most of the child in underdeveloped country don't present immediately after birth rather they present with sequel of HIE like cerebral palsy. Neuroimaging remains modality of choice for diagnosis of such condition. While reporting MRI, radiologist should focus on vulnerability areas for ischemic insults for diagnostic accuracy. In addition, hospital delivery should be promoted instead of home delivery for prevention of perinatal asphyxia and hypoxic brain injury.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

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