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INDICATION OF MRI AND PATTERNS OF HYPOXIC-ISCHEMIC BRAIN INJURY IN PRETERM INFANTS

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Abstract:
Hypoxic-ischemic brain injury that occurs during the antenatal, perinatal, or early postnatal period is a significant diagnostic issue in both term and prematurely born neonates. Magnetic resonance imaging (MRI) has been more widely available in recent years. Using databases such as PubMed and Embase, we searched the literature for all published publications in our field through until the end of 2022. As a result, he can assist medics in identifying whether the newborn's brain damage is to blame for its clinical condition, as well as contribute to determining the infant's future development prognosis. The goal of this study is to give current understanding of various forms of hypoxic-ischemic brain lesions based on our personal experience and MR pictures from the Institute of Mother and Child's Department of Diagnostic Imaging archives.

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INTRODUCTION:

Perinatal asphyxia is a leading cause of morbidity and mortality in term infants all over the world. It refers to a disruption in blood flow or impeded gas exchange during labor and delivery, which can lead to multi-organ failure [1]. The most serious complication of neonatal hypoxia is brain involvement, which is known as hypoxic-ischemic encephalopathy (HIE). HIE is predicted to occur at 1-2 per 1000 live births in high-income countries and at 4-40 per 1000 live births in underdeveloped countries [2,3]. The only neuroprotective treatment demonstrated to minimize death and neurological sequelae in term newborns with moderate to severe HIE is therapeutic hypothermia initiated within 6 hours of birth [4]. Despite this, many newborns suffer from unfavorable consequences such as cerebral palsy (CP), cognitive, visual, or hearing damage, and even death [5]. Furthermore, a considerable number of newborns with moderate HIE who are not now eligible for therapeutic hypothermia have a negative prognosis at follow-up [6].

Neuroimaging is frequently utilized to aid in the diagnosis and prognosis of newborns with HIE. Although HIE is the cause of encephalopathy in the majority of neonates, other etiologies such as infectious diseases, metabolic abnormalities, trauma, and congenital disorders might mimic HIE and should be addressed in the diagnostic work-up [7].

Although cranial ultrasound can still be used in full-term infants with hypoxic-ischaemic brain damage, MRI is the preferred approach for obtaining more thorough and precise information [8]. In 2002, the American Academy of Neurology suggested that computed tomography (CT) be used to detect haemorrhagic lesions in encephalopathic term newborns, and MRI should be used only if CT findings are inconclusive [9].

DISCUSSION:

The expanded use of neuroimaging techniques, particularly MRI, has greatly aided in the timing of brain damage and the recognition of injury patterns [10, 11]. Cowan et al. [11] used MRI within the first two weeks after delivery to reveal that more than 90% of affected neonates exhibited evidence of perinatally acquired lesions on their MRI, with a very low rate of existing prenatal brain injury. At birth or during the first week, the presence of ventricular dilatation, widening of the subarachnoid space and interhemispheric fissure, and the presence of germinolytic cysts or cystic lesions in the white matter are all signs of an antenatal insult or an underlying problem, such as a metabolic disorder [12]. On admission, cranial ultrasonography is also useful because most of these abnormalities suggestive of a prenatal injury or an underlying condition will be detected with ultrasound. Because echogenicity in the white matter takes time to develop, the observation of enhanced echogenicity on a day 1 ultrasonography examination is also strongly predictive of a prenatal injury [12].

Brain Injury Patterns in HIE as shown in MRI:

CNS injury manifests itself as the first category of the above-mentioned injuries in preterm newborns (usually those born between 32 and 36 weeks of gestation [13] with a history of chronic hypoxia). It can be difficult to distinguish them from non-myelinated white matter in a normal newborn brain, and the examination requires an experienced radiologist. An MRI examination performed on an older child, preferably one over the age of two, reveals a hyperintense periventricular gliosis rim, dilatation of the lateral ventricles with uneven external outlines, and significant deepening of the convexity sulci that almost reach the side ends of the lateral ventricles. Lesions are typically bilateral and symmetrical (**Figure 1**) [13].

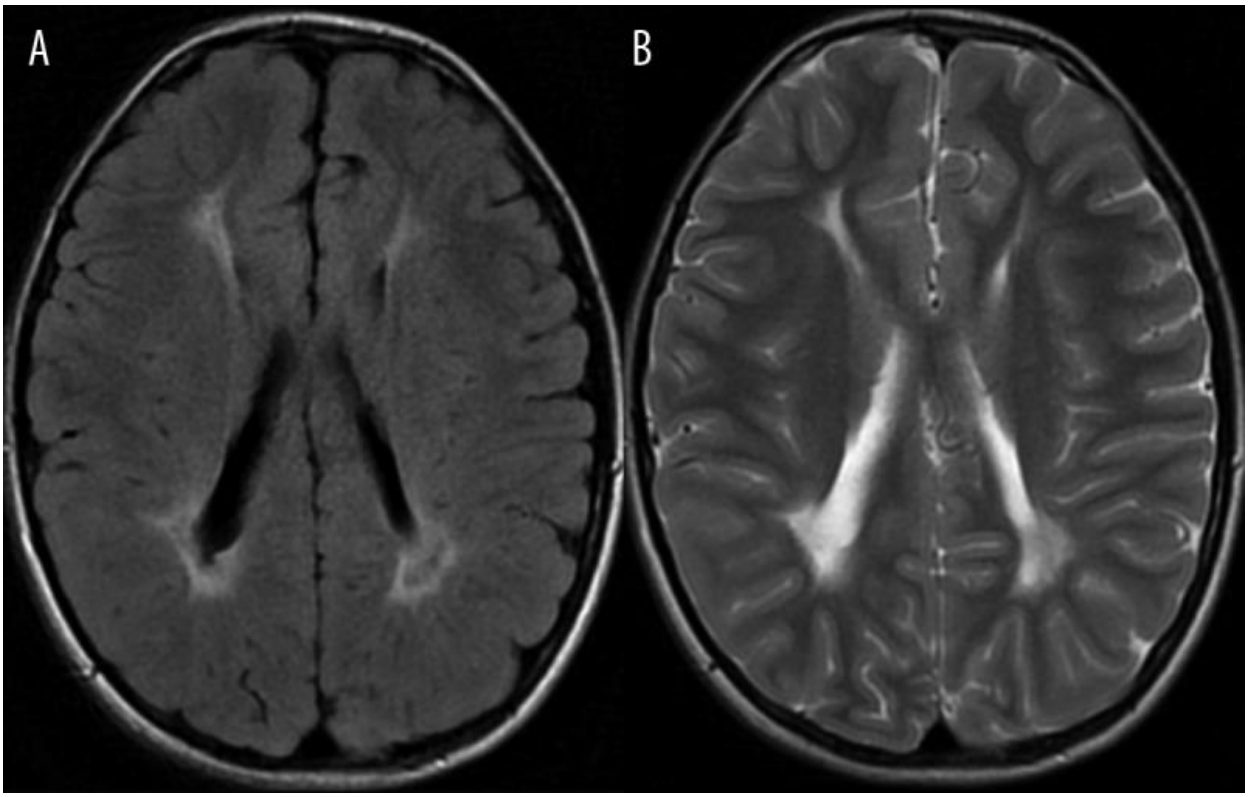

Figure 1: Bilateral, symmetrical paraventricular gliosis, uneven external outlines of the lateral ventricles, thinning of the white matter layer

Several disorders, including placental abruption, umbilical cord prolapse, shoulder dystocia, and uterine rupture, can result in an abrupt disruption of placental perfusion or oxygen supply in the umbilical cord, leading in severe profound hypoxia. Because there is insufficient time for autoregulation to shift blood flow, these sentinel events will disproportionately harm brain locations with the highest metabolic demand. The basal ganglia and thalami (BGT), particularly the ventrolateral thalami and posterior putamina, as well as the perirolandic cortex (**Figure 2**), are among these [14]. These abnormalities are linked to poor motor neurodevelopment, including cerebral palsy [15].

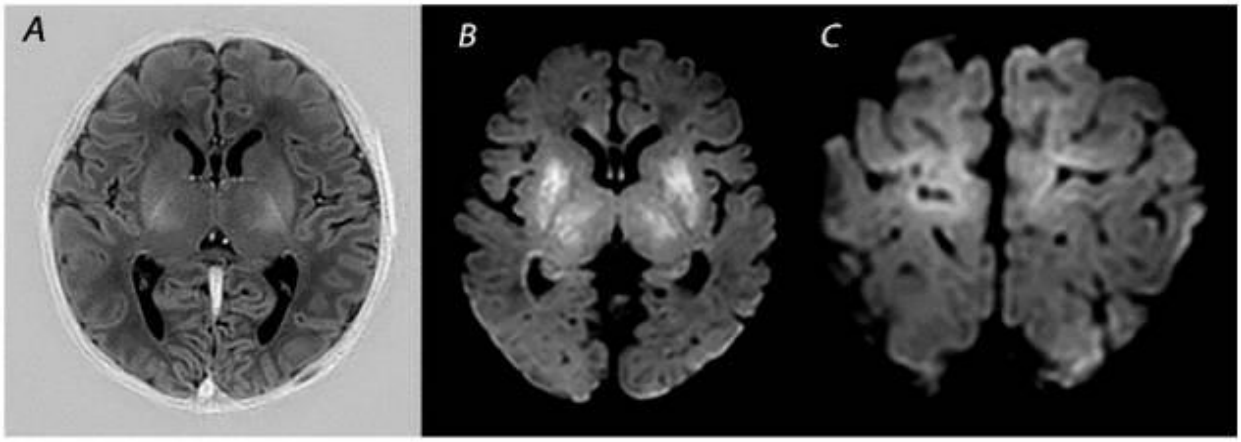

Figure 2: (A), DWI shows extensive diffusion restriction in the basal ganglia and thalami (B), and the perirolandic cortex (C). The infant died after redirection of care.

Mild to moderate asphyxia, also known as partial protracted asphyxia, enables time for cerebral autoregulation to reroute blood flow to high metabolic brain areas at the expense of the anterior, middle, and posterior cerebral arteries' vascular watershed zones [15]. As a result, there is a watershed prominent pattern of injuries. Because injury occurs in a parasagittal pattern along these vascular boundary zones, this pattern is often referred to as the "parasagittal pattern of injury" [16]. Injury to the parasagittal cortex and subcortical white matter can also be found in the more severely affected neonates. In contrast to infants with BGT pattern of injury, who have lower Apgar scores, require more intensive resuscitation at birth, and present with more severe encephalopathy [17], physiological and neurological manifestations in infants with this type of injury are often not severe and only present for a short time after birth. Despite this, clinical and neurological problems might progress over time, emphasizing the significance of closely monitoring newborns with neonatal asphyxia [18]. Severe motor impairment is uncommon in newborns with mild to moderate White Matter/Watershed (WM/WS) damage patterns [19].

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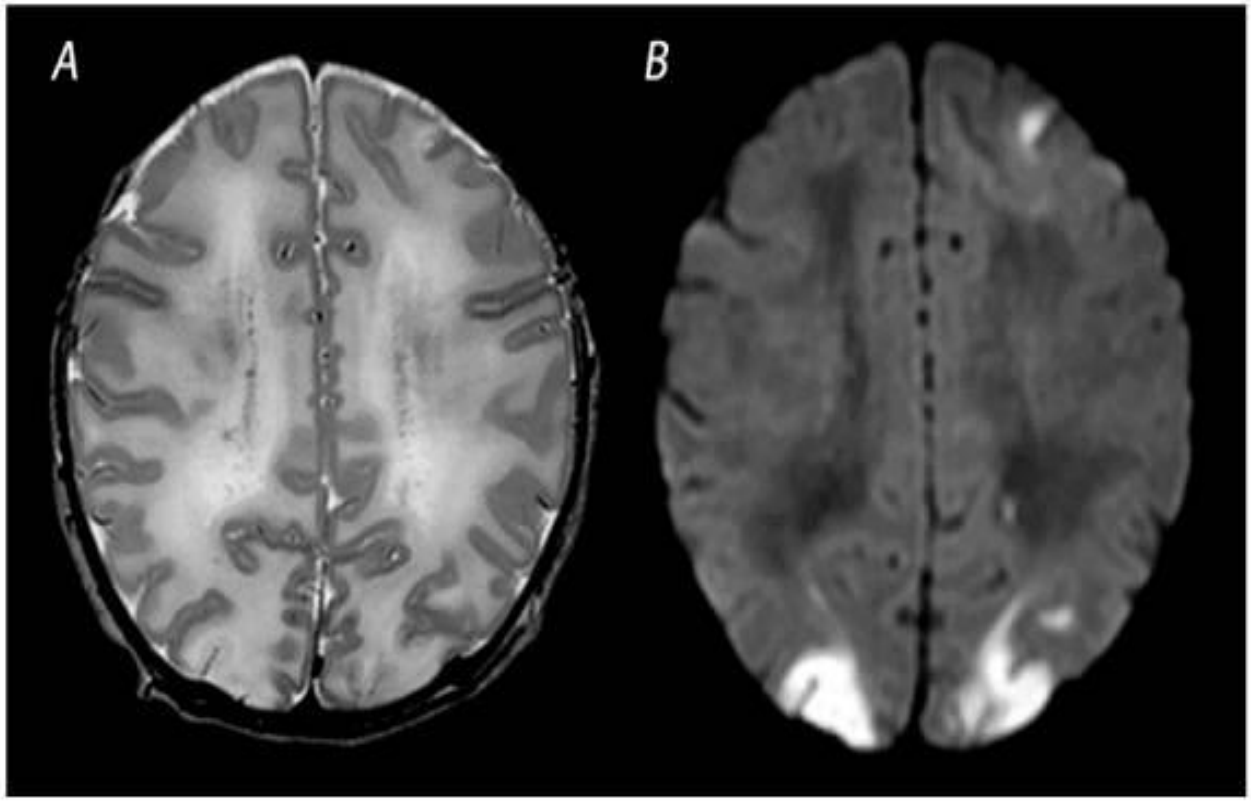

Figure 3: MRI was performed on day 4 in a term infant who was born with Apgar scores of 1/5. Amplitude integrated EEG initially showed a continuous normal voltage background pattern and cooling was not started.

Near complete injury, often known as "global injury," is distinguished by diffuse injury in both the BGT and white matter. This pattern is exhibited in children who have suffered from severe and persistent hypoxia and is thus less frequently documented than the BGT or WM/WS patterns of injury, as these infants may be too unwell to be scanned after birth and die before an MRI can be conducted [16]. The cerebellum may appear somewhat normal on DWI in comparison to a fully white cerebrum, which is why this pattern is often known as the "white cerebrum" [20]. Yet, it has been proven that cerebellar abnormalities on DWI may be overestimated, and that abnormalities are only identified in cases of severe cytotoxic edema [21]. There is mounting evidence that improved MRI modalities, such as quantitative DTI analysis, are superior in detecting cerebellar injury [22].

The other pattern of injury is the Watershed predominate pattern of injury (WS), which is also known as a pattern found after "prolonged partial hypoxia." The vascular watershed zones (anterior-middle cerebral artery and posterior-middle cerebral artery) are involved, affecting white matter as well as the overlaying cortex in more severely affected neonates. Lesions can be unilateral or bilateral, posterior or anterior. Although standard MRI can show loss of the cortical ribbon and thus grey-white matter separation, DWI accentuates the abnormalities and is especially useful in making an early diagnosis [23].

Neurological manifestations at birth may be moderate and do not always match the criteria for perinatal asphyxia, and the start of neurological indications may be delayed [24]. When evaluated at 12-18 months, severe motor impairment is unusual in this group of newborns, and they are often thought to have an early normal result. Nonetheless, until early childhood, inadequate head growth, behavioral difficulties, and language delay are prevalent [25]. Miller et al. [26] identified cognitive abnormalities related with the watershed pattern of injury for the first time at 30 months, although the issues were mostly disregarded at 12 months. They also demonstrated a link with verbal IQ at 4 years of age [26]. Later in childhood, symptomatic parieto-occipital epilepsy may develop, which is frequently associated with lower intelligence quotients and visuospatial cognitive skills [26].

Children with PVL's first form are likely to develop spastic CP involving the lower limbs (diplegia), quadriplegia (tetraplegia - with more prominent symptoms in the lower limbs), or hemiplegia (also involving predominantly the lower limb). Epilepsy is a rather frequent condition. When the subcortical white matter is also damaged, the prognosis is worse: spastic tetraplegia, significant mental impairment (typically moderate in the case of PVL), seizures, and commonly poor eyesight occur.

When the lesions are fairly intense in the second form (BGTL), we expect the kid to have extrapyramidal CP and normal intellectual development.

The prognosis is the worst in the third type (MCE), in which a child develops a severe quadriplegic form of cerebral palsy, with choreoathetoid signs, subsequent microcephaly, mental retardation, and bulbar symptoms. They are frequently associated with epilepsy [18,25].

Another type of brain injury is "lesions localized to the periventricular white matter," which are similar to so-called punctate white matter lesions in preterm infants. Li et al. [27] identified this pattern of injury in 23% of their infants and noted that infants with this type of injury are significantly less mature, have milder encephalopathy, and have fewer clinical seizures than other newborns in their cohort who were diagnosed with the two more common patterns of injury. This pattern of brain injury can also be seen in newborns with congenital cardiac abnormalities [27].

CONCLUSION:

Throughout the last few decades, there has been remarkable advancement in the MRI technology. Both MRI and proton MRS can identify various types of brain injury in (full-term) human neonates after hypoxic-ischemic brain injury and are particularly valuable in predicting neurodevelopmental prognosis. MRI is rapidly being employed for CNS diagnoses in preterm neonates, neonates with extremely low birth weight, and term neonates with substantial perinatal history, as it is the most effective tool for detecting hypoxic-ischemic lesions. Accurate radiographic diagnosis can truly aid in the effective delivery of care to the ill child. PVL, BGTL, and MCE are the three most common patterns of Brain lesions. Based on the MRI pattern of hypoxic-ischemic lesions, we can anticipate neonatal development.

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