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Opinion Article

Auditory Neural Maturation in Newborns Affected by Latent Iron Deficiency

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DESCRIPTION

The Central Nervous System (CNS) needs proper nutrition to grow normally, and this is especially true during pregnancy and in young children. These are crucial times for the development of the brain and for ensuring proper cognitive, motor, and psychosocial development in both childhood and adulthood. Therefore, nutritional deficits throughout these times have the potential to affect cognition, behaviour, and productivity in later life, including in the classroom and in the workplace. Iron insufficiency is one of the most common nutritional deficits.

By taking part in the myelination and synaptogenesis processes, iron plays a crucial function in brain development. Regarding embryonic life, the first three months are when the nervous system is mostly developing, and during the last trimester of pregnancy, the foetus needs to develop its own iron stores. According to studies, the myelination peak and the peak of iron uptake in the CNS occur at the same time, particularly in the late foetal and early postnatal periods [1].

A quarter of the world's population suffers from Iron Deficiency (ID), the most widespread nutritional deficit, and during pregnancy, this percentage rises to 59%. The perinatal period is particularly vulnerable to nutritional insufficiency of this element because certain maternal and foetal pathologies, such as hypertension, diabetes mellitus, intrauterine foetal growth restriction, smoking during pregnancy, and premature birth, may influence the newborn's iron storage [2].

ID anaemia has well-known effects on the CNS, including decreased nerve conduction velocity and alterations in cognition and behaviour. The CNS can also be harmed by iron shortage without anaemia, which is 3-5 times more common than iron deficiency anaemia. It has been demonstrated that NBs exposed to intrauterine iron deficiency presented long-term CNS damage, reaching lower language scores and motor development at 5 years of age compared to those born with normal iron levels. Oligodendrocytes, the cells responsible for producing myelin, are particularly sensitive to iron deficiency. Due to the accelerated processes of neuronal growth and development, myelination, and the establishment of the synaptic network, the developing auditory system is particularly vulnerable to nutritional changes during early foetal and postnatal life because it is a part of the Central Nervous System [3]. The auditory system's neural development moves in a caudal-rostral direction, and the myelination of the auditory nerve pathway is viewed as a measure of cerebral myelination.

The goal of Brainstem Evoked Response Audiometry (BERA) is to measure the electrical response of the brainstem to an auditory stimulation. Using the cochlear nerve, cochlear nuclei, superior olivary complex, and the bridge to the inferior-mesencephalic colliculus as a starting point, this test assesses the electrical activity of the auditory system at the level of the brainstem. Three larger waves (I, III, and V) that can be accurately quantified in neonates with a gestational age of more than 34 weeks make up the waves considered by BERA [4]. The degree of myelination, neuronal development, synaptic function, and axonal growth of the auditory nervous system all have an impact on the absolute latencies for each of the waves and their intervals, which BERA measures as the nerve conduction velocity at various stages of the auditory route. Less latency equals more myelination and vice versa since the association between these latencies and myelination is negative.

Even while research indicates a direct connection between the development of the auditory system and iron homeostasis, it is still unclear exactly what effects ID without anaemia, also known as Latent Iron Deficiency (LID), causes in the body [5]. This study sought to examine the association between LID through serum ferritin of the umbilical cord and myelination of the auditory nerve through BERA in NBs of Gestational Age (GA) equal to or greater than 37 weeks in light of the potential adverse effects of LID on the CNS myelination process.

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