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THE PATHOPHYSIOLOGY OF NEONATAL HYPERBILIRUBINEMIA: DEAFNESS, RESTLESSNESS AND INCOGNITIVENESS

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Neonatal jaundice (NJ) is a condition that almost always resolves independently however, can result in permanent neurological deficits in the absence or delay of timely diagnosis. The neuropathological sequelae of neonatal hyperbilirubinemia are reviewed, with special emphasis on motor, auditory and cognitive dysfunctions.

Kernicterus is the most common cause of acquired deafness and increases risk of hearing loss. Bilirubin encephalopathy progresses with damage to the brainstem cochlear nuclei initially and then ascending pathways and eventually to higher cortical centers. No direct damage of cochlea occurs however may appear secondary to auditory nerve or cochlear nuclei involvement. Additionally, auditory neuropathy (dyssynchrony) has been noted to be associated with neonatal hyperbilirubinemia.

There is evidence involving oxidative stress, a disruption of the blood–brain barrier and the consequent neurotoxicity as the key mechanisms of neuronal damage in the basal ganglia, subthalamic nuclei, hippocampus and the diencephalon that leads to cognitive impairment. Furthermore, the impairment of synaptic plasticity in the dentate gyrus area in vivo has been noted to be associated with cognitive impairment as well. Recent studies also suggest that the severe hyperbilirubinemia has a significant increase in the risk of attention deficit hyperactivity disorder (ADHD).

The loss of neurons, demyelination and gliosis in centers that serve as a convergence of inputs of somatosensory and motor pathways (i.e. globus pallidus and subthalamic nucleus) result in a considerable loss of motor function coordination. Results of MRI scans have shown damage to these specific areas that clinically presents as mild to moderate dystonia, speech and ambulant impairments.

This research identifies potential targets of therapy: reduction of neuronal damage, minimizing of damage to the blood brain barrier, and decreasing unconjugated bilirubin accumulation. Understanding the etiology and scope of sequelae is essential for the development of neuroprotective treatment protocols and early detection of these pathologies related to hyperbilirubinemia.