Pediatric Neurology Part III: Chapter 162. Inborn errors of brain myelin formation (Handbook of Clinical Neurology)

Odile Boespflug-tanguy

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Inborn errors of brain myelin formation or hypomyelinating leukodystrophies (HLD) represent a heterogeneous group of white matter diseases related to a primitive impairment of oligodendrocytes to produce myelin in the central nervous system (CNS). Cerebral magnetic resonance imaging (MRI) allows an assessment of the myelination pattern. The clinical presentation is related to the degree of hypomyelination and its consequences on axonal functions. When the gene defect interferes with the active infantile phase of myelination, the consequences might be severe, with delayed and loss of psychomotor development, absence of myelin signal on cerebral MRI and of identifiable waves on cerebral evoked potentials, as described by Pelizaeus and Merzbacher (PMD). When the pathophysiological mechanism is less severe, myelin production is maintained, although signs of progressive axonopathy are observed, related to progressive spastic paraplegia (SPG) associated with cognitive or behavioral disturbances. HLDs have been classified according to gene defects or associated signs. The X-linked HDL1 (PMD and SPG2) is related to the gene that controls the production of the major CNS myelin proteins, the proteolipid proteins (PLP). The gap junction protein, gamma 2 gene (GJC2) encoding oligodendrocyte-specific connexin, has been shown to be involved in the autosomal recessive HLD2 (PMLD1 and SPG44).

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