Pediatric Neurology Part I: Chapter 68. Diffuse malformations of cortical development (Handbook of Clinical Neurology)

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Malformations of cortical development (MCD) represent a major cause of developmental disabilities and severe epilepsy. Advances in imaging and genetics have improved the diagnosis and classification of these conditions. Up to now, eight genes have been involved in different types of MCD. Lissencephaly-pachygyria and subcortical band heterotopia (SBH) represent a malformative spectrum resulting from mutations of either LIS1 or DCX genes. LIS1 mutations cause a more severe malformation in the posterior brain regions. DCX mutations usually cause anteriorly predominant lissencephaly in males and SBH in female patients. Additional forms are X-linked lissencephaly with corpus callosum agenesis and ambiguous genitalia associated with mutations of the ARX gene. Lissencephaly with cerebellar hypoplasia (LCH) encompass heterogeneous disorders named LCH types a to d. LCHa is related to mutation in LIS1 or DCX, LCHb with mutation of the RELN gene, and LCHd could be related to the TUBA1A gene. Polymicrogyria encompasses a wide range of clinical, etiological, and histological findings. Among several syndromes, recessive bilateral fronto-parietal polymicrogyria has been associated with mutations of the GPR56 gene. Bilateral perisylvian polymicrogyria has been associated with mutations in the SRPX2 gene in a few individuals and with linkage to chromosome Xq28 in a some other families. X-linked bilateral periventricular nodular heterotopia (PNH) consists of PNH with focal epilepsy in females and prenatal lethality in males. Filamin A (FLNA) mutations have been reported in some families and in sporadic patients. It is possible to infer the most likely causative gene by brain imaging studies and other clinical findings.

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