Pediatric Neurology Part III: Chapter 166. Aicardi-Goutières syndrome (Handbook of Clinical Neurology)

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Aicardi-Goutières syndrome (AGS) is a genetically determined encephalopathy demonstrating phenotypic overlap both with the sequelae of congenital infection and with systemic lupus erythematosus (SLE). Recent molecular advances have revealed that AGS can be caused by mutations in any one of five genes, most commonly on a recessive basis but occasionally as a dominant trait. Like AGS, SLE is associated with a perturbation of type I interferon metabolism. Interestingly then, heterozygous mutations in the AGS1 gene TREX1, and the AGS5 gene SAMHD1, underlie a cutaneous subtype of SLE called familial chilblain lupus, and mutations in TREX1 represent the single most common cause of monogenic SLE identified to date. Evidence is emerging to show that the nucleases defective in AGS are involved in removing endogenously produced nucleic acid species, and that a failure of this removal results in activation of the immune system. This hypothesis explains the phenotypic overlap of AGS with congenital infection and some aspects of SLE, where an equivalent type I interferon-mediated innate immune response is triggered by viral and self nucleic acids respectively. These studies beg urgent questions about the development and use of immunosuppressive therapies in AGS and related phenotypes.

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