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The innate immunity-A new player

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Statement of the Problem: Pediatric acute-onset neuropsychiatric syndrome (PANS) is a broad diagnostic criterion characterized by a severe, sudden onset of neuropsychiatric changes. Recently, we acquired evidence that other infectious pathogens (eg, *Mycoplasma pneumoniae*, Epstein-Barr virus; and *Borrelia burgdorferi*) may play a role in a similar neuro-inflammatory syndrome. Innate immunity is the first line of defense against pathogens and is comprised of effectors that provide rapid, robust, non-specific responses. Two aspects of innate immunity are toll-like receptors (TLRs) and the complement system. C1 esterase inhibitor (C1-INH) is the major inhibitor of the classical pathway. Recently, we explored the role of C1INH in a subset of patients with common variable immunodeficiencies (CVID) as well as the role of mast cell (MC) activation, which may lead to microglial activation and may function as a partner to TLR and C1INH signaling in developing neuro-inflammation. In this study, we explored whether TLR-3 signaling, C1INH function, and MC activation play a role in the pathogenesis of post-infectious neurological diseases.

Methodology & Theoretical Orientation: We reviewed clinical cases of patients who presented with neurocognitive changes and had evidence of neuro-inflammation based on autoimmune, neurological biomarkers such as anti-68kDa, anti-GAD antibody autoimmune panels. Patients with neurocognitive clinical presentations and positive biomarkers had an immune workup that included TLR signaling, C1INH levels, and atopic markers including MC signals.

Findings: The response to poly (I:C), the synthetic analogue dsRNA (TLR 3), was decreased by 86±8% compared to normal control. Average levels of C1INH were 16+2 mg/dL, while control levels were 22+5. All patients had evidence of atopy based on IgE, RAST tests, and MC activation.

Conclusion & Significance: We suggest that a complex of immune dysfunction, including TLR-3 signaling, C1INH levels, and atopic partners, specifically mast cell activation, are playing a crucial role in a neuro-inflammatory clinical presentation similar to PANS syndrome.

Biography

Isaac Melamed has focused on clinical practice and research for over 25 years. He has published more than 100 papers and presented and lectured in national and international settings. His major concentration is in primary and secondary immune deficiency, immune-related disease, crosstalk between the immune system and the central nervous system. His mission is early intervention to ensure his patients optimal health.

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