Autoimmune Encephalitis: Bridging Neuroimmunology and Clinical Neuroscience for Better Outcomes

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Abstract

Autoimmune encephalitis (AE) represents a diverse group of inflammatory brain disorders characterized by autoantibody-mediated neuronal dysfunction. This review provides a comprehensive overview of the neuroimmunological mechanisms underlying AE, focusing on the roles of autoantibodies, T cells, B cells, and the blood-brain barrier (BBB). We explore the clinical presentations, diagnostic approaches, and current therapeutic strategies for various subtypes of AE, highlighting the importance of early diagnosis and intervention for improved patient outcomes.

Keywords: Autoimmune encephalitis; Neuroinflammation; Autoantibodies; NMDA receptor; LGI1; CASPR2; T cells; B cells; Blood-brain barrier

Introduction

Autoimmune encephalitis (AE) encompasses a spectrum of conditions where the immune system mistakenly targets neuronal

immunoglobulin (IVIG), or plasma exchange. In cases refractory to these treatments, B cell depletion therapy with rituximab or other immunomodulatory agents may be considered. Early diagnosis and prompt initiation of immunotherapy are critical for achieving optimal patient outcomes and minimizing long-term neurological sequelae [8]. Further research is needed to fully elucidate the speci c triggers and pathways involved in AE pathogenesis and to develop more targeted and e ective therapies.

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Autoimmune encephalitis is a complex group of disorders characterized by autoantibody-mediated neuronal dysfunction and