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Immunopathological Basis of Autoimmune Diseases

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Abstract

Autoimmune diseases represent a diverse group of chronic illnesses where the immune system, typically a defender of the body, mistakenly targets its own tissues and organs. This article provides a comprehensive exploration of the immunopathological mechanisms underlying autoimmune diseases. By unraveling the complex interactions between immune cells, self-antigens, and genetic factors, we gain insights into the development, progression, and potential treatments of these enigmatic conditions.

: Autoimmune; Immunopathological; Genetic factors; Enigmatic

Autoimmune diseases are a class of disorders where the immune system, which normally guards against foreign invaders, turns its attack inward, targeting the body's own cells, tissues, and organs. is misguided immune response leads to a wide range of debilitating conditions a ecting millions worldwide. Understanding the immunopathological basis of autoimmune diseases is critical for developing targeted therapies and improving the quality of life for those a ected. Autoimmune diseases arise from a complex interplay of genetic, environmental, and immunological factors. Key immunopathological mechanisms include [1].

In healthy individuals, the immune system has mechanisms to distinguish self from non-self. Autoimmune diseases o en result from a breakdown in these tolerance mechanisms, leading to the activation of self-reactive immune cells. B cells can produce autoantibodies that target self-antigens, leading to tissue damage. ese autoantibodies are a hallmark of many autoimmune diseases, such as systemic lupus erythematosus (SLE) and rheumatoid arthritis (RA). Autoimmune diseases frequently involve abnormal activation or dysfunction of T cells. In conditions like multiple sclerosis (MS), T cells target the central nervous system, causing demyelination and neurological symptoms. Dysregulated production of cytokines, signaling molecules of the immune system, can promote in ammation and tissue damage. Interleukin-17 (IL-17), for instance, is implicated in psoriasis and ankylosing spondylitis [2].

Certain genetic factors increase susceptibility to autoimmune diseases. e presence of speci c human leukocyte antigen (HLA) genes is associated with a higher risk of developing autoimmune conditions. RA is characterized by chronic in ammation of the joints. Autoantibodies, particularly rheumatoid factor and anti-citrullinated protein antibodies, play a pivotal role in joint damage. Systemic Lupus Erythematosus is a multisystem autoimmune disorder. Immune complexes formed by autoantibodies and self-antigens can deposit in various tissues, leading to in ammation and organ damage [3].

Type 1 Diabetes is a T1D results from the destruction of insulin-producing beta cells in the pancreas. Autoreactive T cells are central to this process. MS involves demyelination of nerve bers in the central nervous system. Auto reactive T cells target myelin, leading to neurological symptoms. IBD, including Crohn's disease and ulcerative