Immune-mediated Pathogenesis and Therapies for Inflammatory Autoimmune Diseases

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Both central and peripheral immune tolerance mechanisms are crucial in maintaining the non-self-reactivity of T and B-cells and in eliminating self-reactive T and B-cells, respectively. Failure of such immune tolerance mechanisms results in autoimmunity leading to the development of autoimmune diseases (ADs). ADs are a heterogeneous set of chronic diseases with shared aetiology in which the immune system may mistakenly attack an individual’s own organ or affect the whole body. The increasing incidence and prevalence make ADs as one of the leading causes of disability and mortality worldwide. To date, there are more than 100 ADs collectively reported, affecting almost 4.5% individuals globally [1, 2].

Genetic and environmental factors are believed to play important roles in the development of ADs [3, 4]. Shared immunogenic pathogenesis are frequently observed making the differentiation of two unlike ADs a challenge. Different cytokines, pro-inflammatory cytokines, chemokines, enzymes, cicosanoids, immune cells and inflammatory mediators take part in the inflammatory pathogenesis of ADs, ultimately leading to devastating and life-threatening clinical manifestations [5, 6].

The main therapeutic goals in treating ADs are to maintain autoimmunity-derived different clinical manifestations and to sustain immunological homeostasis. Using the current knowledge of immune-mediated pathogenesis of ADs, several novel strategies for the therapeutic intervention of the said disease have been conducted. Administration of immunosuppressive drugs is one of such strategies. Specific-targeted treatments involving administration of inflammatory mediator-specific monoclonal antibodies have also been tested and found to be potentially promising in both preclinical and clinical applications. Recently, researches on immunosuppressive or regulatory immune cell-based therapy and exploration of new biomarkers including autoantibodies in ADs have gained much attention [7-9].

In this special issue of Current Pharmaceutical Design (CPD), Giemza-Stokłosa et al. [10] represented an update on the role of ferritin in inflammatory and autoimmune diseases including adult onset Still’s diseases, macrophage activation syndrome, catastrophic antiphospholipid syndrome and sepsis with an emphasis to hyperferritinaemic syndrome. Cavestro et al. [11] provided an insightful review focusing on pathogenesis of migraine from the origin of the neuro-inflammatory theory, to the modern pathophysiological model and the latest therapies. In an interesting review, Khan et al. [12] summarized the role of chemokines and their receptors in pathogenesis of rheumatoid arthritis (RA) and also indicated the possible interactions of chemokines or receptors with various synthetic and natural compounds that might be used as potential therapeutic targets in treatment of RA. Kechida [13] critically reviewed the common factors in the pathogenesis of autoimmune diseases aiming towards developing better treatment strategies.

To sum up, we would like to complete this editorial by thanking Dr. William A. Banks, the Editor-in-Chief, as well as Mr. Kazim Baig, the director of CPD, along with all the contributing authors who have enthusiastically responded to our request by contributing to this special issue of CPD. In addition, we would like to thank all the reviewers who evaluated the submitted manuscripts for this special issue and provided their evaluation based on novelty and scientific contribution in the fields of autoimmunity.

REFERENCES


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