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Immunological Disorders and Immunotherapy

Perspective

Risk Factors of Progression in Systemic Lupus Erythematosus

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DESCRIPTION

Systemic Lupus Erythematosus (SLE) is an immune-mediated disease whose pathogenesis results in autoantigen exposure and the generation of numerous autoreactive antibodies with various antigenic specificities, which are thought to be the disease's primary effectors along with a wide range of cytokines. Environmental factors and genetic predisposition to disease play significant roles in disease development. Systemic autoimmunity was developed in mice that were not predisposed to autoimmune illness as a result of repeated vaccination, according to studies. When macrophages are chemically depleted, mice, which are not prone to autoimmunity, have a comparable autoimmune response. Macrophages are necessary to eliminate apoptotic cells and stop the subsequent necrosis of apoptotic cells from exposing autoantigens.

This suggests that increased autoantigenic exposure via increased autoantigenic load or decreased removal is an early event in the pathogenesis of SLE. When exposed to excessive apoptotic loads, macrophages are speculated to undergo enhanced apoptosis, which increases autoantigen exposure and lowers excision. We can reasonably hypothesise that one significant role of environmental factors in the pathogenesis of SLE is to facilitate autoantigenic exposure to the adaptive immune system in light of an SLE monozygotic concordance rate as low as 24%. This indicates that early concomitants in the aetiology of SLE include increased autoantigenic exposure and reduced autoantigen removal. There are numerous distinct cell death mechanisms that could expose intracellular autoantigens to the immune system.

However, due to the enormous quantity of cellular mass that is normally undergoing apoptosis, which amounts to 150 billion cells every day or more than 10% of total cellular body mass per month, it is thought to have a substantial role in the presentation of pathogenic autoantigens. Professional phagocytes, such as macrophages, generally phagocytoze cells going through apoptosis. However, people with SLE reveal an increase in the frequency of cells going through apoptosis along with decreased phagocytosis. An important step in the pathogenesis of SLE is thought to be the simultaneous occurrence of increased apoptosis and impaired phagocytosis, which can result in a buildup of autoantigen exposure, autoantibody synthesis, and autoimmunity.

The presence of a systemic drug that can improve apoptosis while impairing phagocytosis. Depleted glutathione has been related to increased apoptosis in lymphocytes from SLE patients. Since glutathione is the main reducing agent involved in cellular hydrogen peroxide (H_2O_2) neutralization, a decrease in cellular glutathione will cause an increase in cellular H_2O_2. Being one of the most susceptible cells in the body to the apoptotic action of hydrogen peroxide, lymphocytes have been shown in experiments to undergo apoptosis when exposed to H_2O_2 concentrations as low as 0.7 m. additionally, hydrogen peroxide can inhibit macrophage phagocytosis.

The pathophysiology of SLE is a realistic consideration when both effects of H_2O_2 are taken into consideration. Studies indicating a considerable rise in blood H_2O_2 of up to 220 mM related to anti-dsDNA antibodies and tissue damage in a murine model. Simultaneous lymphocyte apoptosis and decreased macrophage phagocytosis have been found to significantly correlate with disease activity in people with SLE, providing more evidence that H_2O_2 plays a role in the aetiology of the disease. Lymphocytes and macrophages have surpassed their functional limit of redox equilibrium; permitting the intracellular accumulation of hazardous quantities of H_2O_2, apoptosis and decreased phagocytosis take effect. A steady equilibrium between the formation of harmful reactive oxygen species and their ongoing elimination by the cell is known as redox homeostasis. Inefficient redox homeostasis causes H_2O_2 to accumulate within cells. H_2O_2 mediates the majority of the biological effects of reactive oxidant species.

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