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Perspective

A Note on Immune Complex

Julien Caumartin*

Department of Virology, Molecular Virology and Vaccinology Unit, Pasteur Institute, Paris, Île-de-France, France

DESCRIPTION

Immunological-complex illness is a condition in which circulating antigen–antibody complexes, which are produced by co-existing immune reactants, cause vascular damage. In this talk, I'll go over the evidence that immune complexes play a role in the development of glomerulonephritis in systemic lupus erythematous (SLE), as well as the factors that influence the pathogenesis of an experimental model that resembles SLE nephritis in many ways. Clinical Symptoms of Systemic Lupus Erythematosus It is necessary to comment on the clinical and pathological aspects of the illness at the beginning.

A molecule produced by the binding of multiple antigens to antibodies is known as an immunological complex, also known as an antigen-antibody complex or antigen-bound antibody. The bound antigen and antibody function as a single entity, effectively acting as an antigen with its own epitope. The immune complexes can be subjected to a variety of responses after an antigen-antibody reaction, including complement deposition, opsonisation, phagocytosis, or protease processing. Red blood cells with CR1 receptors on their surfaces can bind C3b-coated immune complexes and transport them to phagocytes that are usually found in the liver and spleen, before returning to the general circulation.

The size and form of an immunological complex are determined by the antigen-antibody ratio. This, in turn, defines the immune complex's effect. FcRs, membrane-bound receptors that bind the constant sections of antibodies, are found in many innate immune cells. Most FcRs on innate immunity cells have a poor affinity for a single antibody and must instead attach to an immune complex containing many antibodies to initiate their intracellular signalling pathway and transmit a message from the outside to the inside of the cell.

Additionally, by combining and joining several immune complexes, the avidity, or strength of binding, of the FcRs can be increased. This allows innate immune cells to receive many inputs at the same time, preventing them from becoming activated too soon.

Functions

Regulation of antibody production: Immune complexes may also play a function in antibody production control. B lymphocytes have BCRs on their surfaces, and antigen interaction to these receptors activates a signalling cascade that results in activation. FcRIIb, low affinity receptors unique to the constant region of IgG, are also expressed on the surfaces of B cells. The ligand for these receptors is IgG immune complexes, and immune complex attachment to these receptors causes apoptosis, or cell death. B cells differentiate into plasma cells after activation, and they stop expressing BCR but keep expressing FcRIIb, which permits IgG immune complexes to regulate IgG production via negative feedback and avoid uncontrolled IgG synthesis.

Activation of dendritic cells and macrophages: Immune complexes, particularly those formed of IgG, also have a role in the activation and control of phagocytes, such as DCs and macrophages. Immune complexes are more effective than antigens alone in promoting DC maturation. Because many FcR have a poor affinity for IgG, only immune complexes, not single antibodies, can activate the FcR signalling cascade. Immune complexes binding to FcRs produce considerable alterations in antigen uptake and processing, maturation of the vesicles containing the internalised antigen, and activation in DCs and macrophages as compared to single antibodies binding to FcRs. Different FcRs are expressed by varied types of macrophages and DCs, and they have different affinity for single antibodies and immune complexes.

This allows the DC or macrophage's response to be fine-tuned, and the level of IgG to be adjusted as a result. Distinct FcRs elicit different responses in DCs or macrophages by activating different signalling pathways that can activate or inhibit cellular processes. Antigen presentation begins when the immune complex binds to the DC's membrane-bound receptor and the immune complex and receptor are internalised, allowing the DC to activate T cells. Immune complexes increase T cell activation by this method.

Elimination of opsonized immune complexes: Another form of IgG constant region receptor, Type I FcRs, can attach to IgG immune complexes and cause the opsonized complex to be

Correspondence to: Julien Caumartin, Department of Virology, Molecular Virology and Vaccinology Unit, Pasteur Institute, Paris, Île-de-France, France, E-mail: CaumartinJulien@ ctys.com

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eliminated. Multiple type I FcRs attach to immune complexes, which cluster on the cell surface and initiate the ITAM signalling cascade. The phosphorylation of certain amino acids within a protein sequence initiates this signalling cascade, which leads to the removal of the opsonized immunological complex.