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Immune Overreaction and Human Health: A Detailed View of Hypersensitivity Reactions

Elian Morveau*

Department of Clinical Immunology, Northlake University, Wellington, New Zealand

DESCRIPTION

The human immune system acts as a defense network that protects the body from infectious organisms, toxic substances, and abnormal cells. In many individuals, this defense process works efficiently and removes harmful agents with minimal tissue injury. In some situations, however, the immune response becomes excessive or misdirected, leading to tissue damage instead of protection. These abnormal immune responses are known as hypersensitivity reactions. They arise when the immune system reacts strongly to substances that are harmless for most people or when it attacks the body's own tissues after exposure to specific antigens. Such reactions may appear within minutes or may develop gradually over several days depending on the type of immune mechanism involved.

Hypersensitivity reactions are traditionally grouped into four categories each category differs in mechanism, immune mediators, timing, and clinical appearance. Understanding these reactions has improved diagnostic methods and therapeutic approaches used in hospitals and research centers across the world.

Type I hypersensitivity is also known as immediate hypersensitivity. It occurs rapidly after exposure to an allergen and involves immunoglobulin E antibodies, commonly called IgE. During the first encounter with an allergen such as pollen, dust mites, animal dander, or certain foods, antigen-presenting cells stimulate helper T lymphocytes. These lymphocytes encourage B cells to produce IgE antibodies. The IgE molecules then attach to mast cells and basophils. During later exposure to the same allergen, the allergen binds to the IgE molecules attached to these cells, causing the release of histamine, leukotrienes, and other inflammatory chemicals. These mediators produce symptoms such as itching, sneezing, skin rash, bronchial constriction, and swelling.

Allergic rhinitis, asthma, urticaria, and anaphylaxis are common examples of Type I hypersensitivity. Anaphylaxis is among the most severe allergic conditions because it can lead to airway obstruction, severe hypotension, and circulatory collapse within a short period. Foods such as peanuts and shellfish, insect venom, and medications including penicillin may trigger this reaction. Immediate medical treatment with epinephrine is often necessary to prevent fatal outcomes.

Type II hypersensitivity involves antibody-mediated destruction of cells. In this reaction, immunoglobulin G or immunoglobulin M antibodies bind directly to antigens located on cell surfaces or extracellular tissues. The binding activates complement proteins or attracts immune cells that damage the targeted tissues. Unlike Type I reactions, Type II reactions are not linked to IgE antibodies or mast cell degranulation.

Several diseases demonstrate this mechanism. Autoimmune hemolytic anemia develops when antibodies attack red blood cells, resulting in hemolysis and anemia. In Goodpasture syndrome, antibodies target proteins in the basement membranes of the lungs and kidneys, leading to pulmonary bleeding and renal dysfunction. Another well-known example is hemolytic disease of the newborn, which may occur when an Rh-negative mother produces antibodies against Rh-positive fetal red blood cells. Modern prenatal screening and preventive treatment with Rh immunoglobulin have greatly reduced the frequency of this condition.

Type III hypersensitivity is caused by immune complex deposition. Immune complexes form when antibodies bind to soluble antigens in circulation. Under normal conditions these complexes are removed efficiently by phagocytic cells. When produced in excessive amounts or when clearance mechanisms fail, the complexes accumulate in blood vessels, joints, kidneys, and other tissues.

CONCLUSION

Hypersensitivity reactions represent an important aspect of clinical medicine because they involve multiple organ systems and affect millions of individuals globally. These reactions demonstrate that the immune system, while protective under

Correspondence to: Elian Morveau, Department of Clinical Immunology, Northlake University, Wellington, New Zealand, E-mail: elian.morveau@northlake-medresearch.nz

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normal circumstances, can also become a source of significant tissue injury. Their deposition activates complement proteins and inflammatory pathways, causing tissue injury. Careful evaluation of symptoms, immune mechanisms, and patient history allows clinicians to identify the underlying category of hypersensitivity and select appropriate treatment strategies. Continued scientific investigation will likely improve prevention, diagnosis, and therapy for these complex immune disorders in the coming years.