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Mechanisms of Processes Which Make the Organism Hypersensibilized and Type I Hypersensitive Reaction

Raxmonov Shoxzodbek Oybek o'g'li
Assistant of the Department of Pathology and Forensic Medicine, Central Asian Medical University
Andijan State Medical Institute, 2 nd year clinical supervisor, "Laboratory work" course

Annotation. A hypersensitivity reaction is an altered immunologic response to an antigen that results in disease or damage to the individual. Hypersensitivity reactions can be classified in two ways: by the immunologic mechanism that causes disease (types I, II, II, and IV), and by the source of the antigen that the immune system is attacking (allergy, autoimmunity, and alloimmunity (also termed isoimmunity). The mechanism that initiates the onset of hypersensitivity reactions is not completely understood. It is generally accepted that genetic, infectious, and environmental agents are contributing factors.

Keywords: Mechanisms, Organism, initiates

Introduction

Hypersensitivity reactions can be characterized by the particular immune mechanism that results in the disease. These mechanisms are apparent in most hypersensitivity reactions and have been divided into four distinct types: type I (Ig E mediated reactions), type II (tissue-specific reactions), type III (immune complex–mediated reactions), and type IV (cell-mediated reactions). This classification is an artificial one, and a particular disease is seldom associated with only a single mechanism. The four mechanisms are interrelated, and in most hypersensitivity reactions, several mechanisms can be functioning simultaneously or sequentially.

Immunologic Mechanisms of Hypersensitivity Reactions

As with all immune responses, hypersensitivity reactions require sensitization against a particular antigen that results in a primary immune response. This occurs when the immune system first encounters an antigen and forms antigen-specific memory B cells and T cells (immunologic memory). Disease symptoms appear after secondary exposure to the offending antigen when memory cells are rapidly activated against the same antigen. Hypersensitivity reactions are immediate or delayed, depending on the time required to elicit clinical symptoms after reexposure to the antigen. Reactions that occur within minutes to a few hours after exposure to antigen are termed immediate hypersensitivity reactions. Delayed hypersensitivity reactions may take several hours to appear and are at maximal severity days after reexposure to the antigen. Generally, immediate reactions are caused by antibody, whereas delayed reactions are caused by cells (e.g., T cells, natural killer [NK] cells, macrophages).

The most rapid and severe immediate hypersensitivity reaction is anaphylaxis. Anaphylaxis occurs within minutes of reexposure to the antigen and can be either systemic (generalized) or cutaneous (localized). Symptoms of systemic anaphylaxis include pruritus, erythema, vomiting, abdominal cramps, diarrhea, and breathing difficulties, and the most severe reactions may include contraction of bronchial smooth muscle, edema of the throat, and decreased blood pressure that can lead to shock and death. Examples of systemic anaphylaxis are allergic reactions to bee stings, peanuts, shellfish, or eggs. Cutaneous anaphylaxis results in local symptoms related to inflammation, such as pain, swelling, and redness, which occur at the site of

exposure to an antigen (e.g., a painful local reaction to an injected vaccine or drug).

Mechanisms of Hypersensitivity Reactions. Type I: IgE-Mediated Hypersensitivity Reactions Type I hypersensitivity reactions are mediated by antigen-specific immunoglobulin E (IgE) and the products of tissue mast cells (Fig. 8.1). Most common allergic reactions are type I reactions against environmental antigens. Because of this strong association, many health care professionals use the term allergy to indicate only IgE-mediated reactions. However, IgE can contribute to some autoimmune and alloimmune diseases.

Mechanisms of type I, IgE-mediated hypersensitivity reactions. IgE has a relatively short life span in blood because it rapidly binds to Fc receptors (antibody receptors) on mast cells. The Fc receptors on mast cells specifically bind Ig E that has not previously interacted with antigen. After a large amount of IgE has bound to the mast cells, an individual is considered sensitized. When there is a secondary or reexposure of a sensitized individual to the allergen, the IgE antibodies signal the mast cells to release mediators. Histamine is the most potent preformed mediator of IgE-mediated hypersensitivity. Histamine acts immediately (within 15-30 minutes) and affects several key target cells. The tissues most commonly affected by type I responses contain large numbers of mast cells and are sensitive to the effects of histamine released from them. These tissues are found in the gastrointestinal tract, the skin, and the respiratory tract. Acting through histamine 1 (H1) receptors, histamine contracts bronchial smooth muscles (bronchial constriction), increases vascular permeability (edema), and causes vasodilation (increased blood flow). The interaction of histamine with H2 receptors results in increased gastric acid secretion.

Mast cells also synthesize secondary mediators, such as leukotrienes, prostaglandins, and platelet activating factor, which act more slowly (within hours) and have effects similar to that of histamine. These newly formed mediators also attract other immune cells (e.g., eosinophils, neutrophils, basophils, monocytes) and activate kinins and complement. These mediators are responsible for a late phase reaction that sets in 2 to 24 hours later even without additional exposure to antigen and may last for several days. Gastrointestinal allergy is caused by allergens that enter through the mouth—usually foods or medicines—and results in increased fluid secretion and increased peristalsis. Symptoms include vomiting, diarrhea, or abdominal pain. Foods most often implicated in gastrointestinal allergies are milk, chocolate, citrus fruits, eggs, wheat, nuts, peanut butter, and fish. The most common food allergy in adults is a reaction to shellfish, which may initiate an anaphylactic response and death. When food is the source of an allergen, the active immunogen may be an unidentifiable product of how the food is processed during manufacture or broken down by digestive enzymes. Sometimes the allergen is a drug, an additive, or a preservative in the food. For example, cows treated for mastitis with penicillin yield milk containing trace amounts of this antibiotic. Thus hypersensitivity apparently caused by milk proteins may, instead, be the result of an allergy to penicillin.

Urticaria, or hives, is a dermal (skin) manifestation of allergic reactions. The underlying mechanism is the localized release of histamine and increased vascular permeability, resulting in limited areas of edema. Urticaria is characterized by white fluid-filled blisters (wheals) surrounded by areas of redness (flares). This wheal and flare reaction is usually accompanied by pruritus. Not all urticarial symptoms are caused by immunologic reactions. Some, termed nonimmunologic urticaria, result from exposure to cold temperatures, emotional stress, medications, systemic diseases, or malignancies (e.g., lymphomas). Effects of allergens on the mucosa of the eyes, nose, and respiratory tract include conjunctivitis (inflammation of the membranes lining the eyelids), rhinitis (inflammation of the mucous membranes of the nose), and asthma (constriction of the bronchi). Symptoms are caused by vasodilation, hypersecretion of mucus, edema, and swelling of the respiratory mucosa. Because the mucous membranes lining the respiratory tract are continuous, they are all adversely affected. The degree to which each is affected determines the symptoms of the disease. One of the most common type I reactions is asthma. The central problem in allergic diseases of the lung is obstruction of the large and small airways (bronchi) of the lower respiratory tract by bronchospasm (constriction of smooth muscle in airway walls), edema, and thick secretions. This leads to ventilatory insufficiency, wheezing, and difficult or labored breathing. Certain individuals are

genetically predisposed to develop allergies and are described as being atopic. In families in which one parent has an allergy, allergies develop in about 40% of the offspring. If both parents have allergies, the incidence may be as high as 80%. Atopic individuals tend to produce higher quantities of IgE and have more Fc receptors for IgE on their mast cells. The airways and the skin of atopic individuals have increased responsiveness to a wide variety of both specific and nonspecific stimuli.

All in all, hypersensitivity processes are a process caused by the protective forces of the body, and the result is a pathological condition, which is considered a disease. It has several manifestations in the human organ system and is named after various diseases. Above, we focused only on the mechanism of type I hypersensitivity, in addition, there are three more types..

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