Types Of Hypersensitivity Reactions

== History == f296685ccb Toxic epidermal necrolysis (TEN); f296685ccb In 1963, Philip George Houthem Gell and Robin Coombs introduced a systematic classification of the different types of hypersensitivity based on the types of antigens and immune responses involved. According to this system, known as the Gell and Coombs classification or Gell-Coombs's classification, there are four types of hypersensitivity: f296685ccb Acute generalized exanthematous pustulosis (AGEP). f296685ccb The Arthus reaction was discovered by Nicolas Maurice Arthus in 1903. Arthus repeatedly injected horse serum subcutaneously into rabbits. After four injections, he found that there was edema and that the serum was absorbed slowly. Further injections resulted in stronger reactions with faster onset: the fifth resulted in a longer-lasting edema and more severe infiltration, the sixth the formation of a sterile thick white mass that lasted weeks, and on the seventh a rapid reddening followed by blanching and drying, ending with a gangrenous plaque. The rest of the animal remained healthy. f296685ccb Type I, which is an Immunoglobulin E (IgE)-mediated immediate reaction. f296685ccb Drug reaction with eosinophilia and systemic symptoms (i.e. DRESS syndrome), also termed drug-induced hypersensitivity syndrome (DIHS); f296685ccb The activation of the complement system results in opsonization, the agglutination of red blood cells, cell lysis, and cell death. f296685ccb == Pathophysiology == f296685ccb In type I hypersensitivity, B cells are stimulated (by CD4+ Th2 cells) to produce IgE antibodies specific to an antigen. The difference between a normal infectious immune response and a type 1 hypersensitivity response is that in type 1 hypersensitivity, the antibody is IgE instead of IgA, IgG, or IgM. During sensitization, the IgE antibodies bind to FcεRI receptors on the surface of tissue mast cells and blood basophils. Mast cells and basophils coated by IgE antibodies are "sensitized". Later exposure to the same allergen cross-links the bound IgE on sensitized cells, resulting in anaphylactic degranulation, which... f296685ccb == Classification == f296685ccb

Type II hypersensitivity This subsequently leads to cell lysis, tissue damage or loss of function through mechanisms such as. Type II hypersensitivity, in the Gell and Coombs classification of allergic reactions, is an antibody-mediated process in which IgG and IgM antibodies Type II hypersensitivity, in the Gell and Coombs classification of allergic reactions, is an antibody-mediated process in which IgG and IgM antibodies are directed against antigens on cells (such as circulating red blood cells) or extracellular material (such as basement membrane) f296685ccb

complement activation via the classical complement pathway f296685ccb Early exposure of children to potential allergens may be protective. Treatments for allergies include avoidance of known... f296685ccb This reaction is caused when CD4+ Th1 cells recognize foreign antigen in a complex with the MHC class II on the surface of antigen-presenting cells. These can be macrophages that secrete IL-12, which stimulates the proliferation of further CD4+ Th1 cells. CD4+ T cells secrete IL-2 and interferon gamma (IFNγ), inducing the further release of other Th1 cytokines, thus mediating the immune response. Activated CD8+ T cells destroy target cells on contact, whereas activated macrophages produce hydrolytic enzymes and, on presentation with certain intracellular pathogens, transform into multinucleated giant cells. f296685ccb

Arthus reaction Type III hypersensitivity reactions are immune complex-mediated In immunology, the Arthus reaction () is a type of local type III hypersensitivity reaction. immunology, the Arthus reaction (/ˌɑːr'tjuːs/) is a type of local type III hypersensitivity reaction. Type III hypersensitivity reactions are immune complex-mediated, and involve the deposition of antigen/antibody complexes mainly in the vascular walls, serosa (pleura, pericardium, synovium), and glomeruli. This reaction is usually encountered in experimental settings following the injection of antigens that an animal has already developed antibodies against. It rarely happens in humans f296685ccb

Adverse drug reactions... f296685ccb The classification organizes the hypersensitivity reactions to NSAIDs into the following five categories: f296685ccb Some clinical examples: f296685ccb Stevens-Johnson/toxic epidermal necrolysis overlap syndrome (SJS/TEN); and f296685ccb

Severe cutaneous adverse reactions reactions initiated by CD8+ T cells and natural Severe cutaneous adverse reactions (SCARs) are a group of potentially lethal adverse drug reactions that involve the skin and mucous membranes of various body openings such as the eyes, ears, and inside the nose, mouth, and lips. In more severe cases, SCARs also involves serious damage to internal organs. necrolysis overlap syndrome are a spectrum of Type IV, Subtype IVc, delayed hypersensitivity reactions, i. e f296685ccb

Allergy and Robin Coombs that described four types of hypersensitivity reactions, known as Type I to Type IV hypersensitivity. Allergic reactions give rise to allergic diseases such as hay fever, allergic conjunctivitis, allergic asthma, atopic dermatitis, food allergies, and anaphylaxis. With this new classification, the An allergy is an exaggerated immune response where the body mistakenly identifies an ordinarily harmless allergen as a threat. Symptoms of allergic diseases may include red eyes, an itchy rash, sneezing, coughing, a runny nose, shortness of breath, or swelling f296685ccb

Type I hypersensitivity Type I hypersensitivity (or immediate hypersensitivity), in the Gell and Coombs classification of allergic reactions, is an allergic reaction provoked Type I hypersensitivity (or immediate hypersensitivity), in the Gell and Coombs classification of allergic reactions, is an allergic reaction provoked by re-exposure to a specific type of antigen referred to as an allergen. Type I is distinct from type II, type III and type IV hypersensitivities. The relevance of the Gell and Coombs classification of allergic reactions has been questioned in the modern-day understanding of allergy, and it has limited utility in clinical practice f296685ccb

Hypersensitivity pneumonitis (HP) can be categorized as acute, subacute, and chronic based on the duration of the illness. f296685ccb Exposure may be by ingestion, inhalation, injection, or direct contact. f296685ccb SCARs includes five syndromes: f296685ccb Type II hypersensitivity reactions are also known as antibody-mediated cytotoxic reactions. These reactions usually take between 2 and 24 hours to develop. f296685ccb == Signs and symptoms == f296685ccb Stevens–Johnson syndrome (SJS); f296685ccb anti-receptor activity. f296685ccb The overreaction of the helper T cells and overproduction of cytokines damage tissues, cause inflammation, and cell death. Type IV hypersensitivity can usually be resolved with topical... f296685ccb Type II, an antibody-mediated reaction canonically... f296685ccb == Types == f296685ccb

Hypersensitivity pneumonitis The inhaled antigens produce a hypersensitivity immune reaction causing inflammation of the airspaces (alveoli) and small airways

(bronchioles) within the lung. a hypersensitivity immune reaction causing inflammation of the airspaces (alveoli) and small airways (bronchioles) within the lung. Hypersensitivity pneumonitis Hypersensitivity pneumonitis (HP) or extrinsic allergic alveolitis (EAA) is a syndrome caused by the repetitive inhalation of antigens from the environment in susceptible or sensitized people. Hypersensitivity pneumonitis may eventually lead to interstitial lung disease. Common antigens include molds, bacteria, bird droppings, bird feathers, agricultural dusts, bioaerosols and chemicals from paints or plastics. People affected by this type of lung inflammation (pneumonitis) are commonly exposed to the antigens by their occupations, hobbies, the environment and animals f296685ccb

Type III hypersensitivity Finally, the third step is the inflammatory reaction, during which the classical pathway is activated and macrophages and neutrophils are recruited to the affected tissues. Type III hypersensitivity, in the Gell and Coombs classification of allergic reactions, occurs when there is accumulation of immune complexes (antigen-antibody Type III hypersensitivity, in the Gell and Coombs classification of allergic reactions, occurs when there is accumulation of immune complexes (antigen-antibody complexes) that have not been adequately cleared by innate immune cells, giving rise to an inflammatory response and attraction of leukocytes. The second step is immune complex deposition, during which the complexes leave the plasma and are deposited into tissues. The first step is immune complex formation, which involves the binding of antigens to antibodies to form mobile immune complexes. Such reactions may progress to immune complex diseases. There are three steps that lead to this response f296685ccb

NSAID hypersensitivity reactions These syndromes have recently been classified by the European Academy of Allergy and Clinical Immunology Task Force on NSAIDs Hypersensitivity. Hypersensitivity drug reactions differ from drug toxicity reactions in that drug toxicity reactions result from the pharmacological action of a drug, are dose-related, and can occur in any treated individual. Hypersensitivity drug reactions differ from drug toxicity reactions in that drug toxicity reactions result from the pharmacological action of a drug NSAID (or nonsteroidal anti-inflammatory drug) hypersensitivity reactions encompass a broad range of allergic or allergic-like symptoms that occur within minutes to hours after ingesting aspirin or other NSAID nonsteroidal anti-inflammatory drugs. Hypersensitivity reactions are idiosyncratic reactions to a drug. Although the term NSAID was introduced to signal a comparatively low risk of adverse effects, NSAIDs do evoke a broad range of hypersensitivity syndromes. drugs f296685ccb

Type IV hypersensitivity This response involves the interaction of T cells, monocytes, and macrophages. Type IV hypersensitivity, in the Gell and Coombs classification of allergic reactions, often called delayed-type hypersensitivity, is a type of hypersensitivity Type IV hypersensitivity, in the Gell and Coombs classification of allergic reactions, often called delayed-type hypersensitivity, is a type of hypersensitivity reaction that can take a day or more to develop. Unlike the other types, it is not humoral (not antibody-mediated) but rather is a type of cell-mediated response f296685ccb

Hypersensitivity Hypersensitivity (also called hypersensitivity reaction) is an immune response characterized by mechanisms that cause significant tissue damage or physiological Hypersensitivity (also called hypersensitivity reaction) is an immune response characterized by mechanisms that cause significant tissue damage or physiological dysfunction, whether directed against pathogens, harmless environmental antigens, or self-antigens that is reproducible upon re-exposure to the antigen. Collectively, hypersensitivities are extremely common: hay fever affects about 1 in 10 people worldwide, asthma affects hundreds of millions, and about 1 in 12 people have an autoimmune disease. While hypersensitivity mechanisms can sometimes serve protective functions (such as control of infectious diseases), they are

distinguished by their capacity to cause collateral tissue damage that may exceed any protective benefit f296685ccb

Antibody-dependent cellular cytotoxicity or f296685ccb The five disorders have similar pathophysiologies, i.e. disease-causing mechanisms, for which new strategies are in use or development to identify individuals predisposed to develop the SCARs-inducing effects of specific drugs and thereby avoid treatment with them. Maculopapular rash (MPR) is a less-well defined and benign form of drug-induced adverse skin reactions; while not classified in the SCARs group, it shares a similar pathophysiology with SCARs and is caused by some of the same drugs which cause SCARs. f296685ccb Common allergens include pollen, certain foods, metals, insect stings, medications, and materials, such as latex. The development of allergies is due to genetic and environmental factors. The mechanism of allergic reactions involves immunoglobulin E antibodies (IgE) binding to an allergen and then to a receptor on mast cells or basophils, where they trigger the release of inflammatory chemicals such as histamine. Diagnosis is typically based on a person's medical history. Further testing of the skin or blood may be useful in certain cases. Positive tests, however, may not necessarily mean there is a significant allergy to the substance in question. f296685ccb