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Chapter 1 : Anaphylaxis - Wikipedia

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Patch test Patch testing is a method used to determine if a specific substance causes allergic inflammation of the skin. It tests for delayed reactions. It is used to help ascertain the cause of skin contact allergy, or contact dermatitis. Adhesive patches, usually treated with a number of common allergic chemicals or skin sensitizers, are applied to the back. The skin is then examined for possible local reactions at least twice, usually at 48 hours after application of the patch, and again two or three days later. Blood testing[edit] An allergy blood test is quick and simple, and can be ordered by a licensed health care provider e. Unlike skin-prick testing, a blood test can be performed irrespective of age, skin condition, medication, symptom, disease activity, and pregnancy. Adults and children of any age can get an allergy blood test. For babies and very young children, a single needle stick for allergy blood testing is often more gentle than several skin pricks. An allergy blood test is available through most laboratories. Multiple allergens can be detected with a single blood sample. Allergy blood tests are very safe, since the person is not exposed to any allergens during the testing procedure. The test measures the concentration of specific IgE antibodies in the blood. Quantitative IgE test results increase the possibility of ranking how different substances may affect symptoms. A rule of thumb is that the higher the IgE antibody value, the greater the likelihood of symptoms. Allergens found at low levels that today do not result in symptoms can not help predict future symptom development. The quantitative allergy blood result can help determine what a patient is allergic to, help predict and follow the disease development, estimate the risk of a severe reaction, and explain cross-reactivity. These methods have shown that patients with a high total IgE have a high probability of allergic sensitization, but further investigation with allergy tests for specific IgE antibodies for a carefully chosen of allergens is often warranted. Challenge testing is when small amounts of a suspected allergen are introduced to the body orally, through inhalation, or via other routes. Except for testing food and medication allergies, challenges are rarely performed. When this type of testing is chosen, it must be closely supervised by an allergist. This testing method is used most often with foods or medicines. A patient with a suspected allergen is instructed to modify his diet to totally avoid that allergen for a set time. If the patient experiences significant improvement, he may then be "challenged" by reintroducing the allergen, to see if symptoms are reproduced. There are other types of allergy testing methods that are unreliable, including applied kinesiology allergy testing through muscle relaxation , cytotoxicity testing, urine autoinjection, skin titration Rinkel method , and provocative and neutralization subcutaneous testing or sublingual provocation. Allergy prevention in children Dietary avoidance is not effective as a preventative measure for allergies. Vegetable oil, nuts and fast food may increase the risk while fruits, vegetables and fish may decrease it. These include antihistamines , glucocorticoids , epinephrine adrenaline , mast cell stabilizers , and antileukotriene agents are common treatments of allergic diseases. Although rare, the severity of anaphylaxis often requires epinephrine injection, and where medical care is unavailable, a device known as an epinephrine autoinjector may be used. Allergen immunotherapy Anti-allergy immunotherapy Allergen immunotherapy is useful for environmental allergies, allergies to insect bites, and asthma. EPD has also been tried for the treatment of autoimmune diseases but evidence does not show effectiveness. The authors concluded that, based on rigorous clinical trials of all types of homeopathy for childhood and adolescence ailments, there is no convincing evidence that supports the use of homeopathic treatments.

Chapter 2 : Inflammation - Wikipedia

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Received Jun 2; Accepted Nov This article has been cited by other articles in PMC. Abstract Background Outbreaks of acute Chagas disease associated with oral transmission are easily detected nowadays with trained health personnel in areas of low endemicity, or in which the vector transmission has been interrupted. Given the biological and genetic diversity of Trypanosoma cruzi, the high morbidity, mortality, and the observed therapeutic failure, new characteristics of these outbreaks need to be addressed at different levels, both in Trypanosoma cruzi as in patient response. Methods The clinical, pathological and epidemiological aspects of outbreaks were analyzed. In addition, Trypanosoma cruzi clones were biologically characterized both in vitro and in vivo, and the susceptibility to the classical trypanocidal drugs nifurtimox and benznidazole was evaluated. Trypanosoma cruzi clones were genotyped by means of mini-exon intergenic spacer and cytochrome b genes sequencing. Results All clones were DTU I, and based on the mini-exon intergenic spacer, belong to two genotypes: G2 related with sub-urban, and G11 with rural outbreaks. The cardiac tissue showed intense inflammatory infiltrate, myocardial necrosis and abundant amastigote nests. However, although the gastrointestinal tissue was congestive, no inflammation or parasites were observed. Conclusions Although all clones belong to DTU I, two intra-DTU genotypes were found with the sequencing of the mini-exon intergenic spacer, however there is no strict correlation between genetic groups, the cycles of the parasite or the clinical forms of the disease. When the diagnosis was early, the patients responded well to antichagasic treatment, which highlights the importance of diagnosis and treatment early to prevent fatal outcomes associated with these acute episodes. Electronic supplementary material The online version of this article doi: In Colombia, it is estimated that 1. CD has two clinical phases: The digestive forms of CD occur almost exclusively in Argentina, Brazil, Chile and Bolivia, although they have also been reported in Mexico, and Colombia [3 â€” 5]. Differences in biological characteristics among T. In this regard, efforts to analyze the relevance of these differences in pathogenesis of CD are necessary. A new subdivision within TcI parasites has been reported using nuclear and mitochondrial molecular markers as miniexon and cytochrome b gene sequencing, respectively [16 â€” 18]. Some of these TcI variants seem to be associated with humans and peridomestic and sylvatic transmission cycles [18]. However, recent reviews identified that although there are genetic and geographical structures, these are not strictly associated with cycle and host origins [19]. No vaccines are available so far, and there are only two registered drugs, the nitrofuran derivative, nifurtimox Lampit, Bayer and 2-nitroimidazole benznidazole Radanil, Roche , being especially effective in newborns, and in the acute phase [1]. However, these drugs have severe limitations of long protocols of treatment and potential harmful side-effects. Subsequently, in the period â€”, other cases were reported from several geographic regions of Colombia, including Santander Department [36]. In , nine new acute CD cases were reported [37]. Between and outbreaks of probable oral transmission were reported in Santander [33 , 38]. The purpose of this study was to evaluate the clinical and pathological features of patients involved in six outbreaks from Santander, between to , and the biological and genetic characteristics of T. These genotypes had differential distribution in urban and rural areas, and the G2 genotype was more susceptible to drugs analysed. Methods Ethics, consent and permissions Patients were included in the study after written informed consent, according to the declaration of Helsinki. For children enrolled in the study, written informed consent was given by parents. Patients diagnosed with CD were treated according to the guidelines of the Colombian Health Ministry. Santander is one of the 32 departments of Colombia located in the northeastern part of the country. Santander Department is considered the third most endemic department for CD in Colombia [39]. According to the Santander Health authorities, six municipalities are considered highly endemic, some of

which had improvement of rural housing and vector control program [40]. The first outbreak occurred in December in the town of Lebrija, in there were two outbreaks, one in a peri-urban area of the Bucaramanga city Barrio Bucaramanga , and another in Piedecuesta.

Chapter 3 : Molecular and Biological Aspects of the Acute Allergic Reaction : S. Johansson :

Molecular and Biological Aspects of the Acute Allergic Reaction (Nobel Foundation Symposia) Softcover reprint of the original 1st ed. Edition.

The activation and degranulation of eosinophils is strictly regulated, as their inappropriate activation would be very harmful to the host. The first level of control acts on the production of eosinophils by the bone marrow. Few eosinophils are produced in the absence of infection or other immune stimulation. But when TH2 cells are activated, cytokines such as IL -5 are released that increase the production of eosinophils in the bone marrow and their release into the circulation. However, transgenic animals overexpressing IL-5 have increased numbers of eosinophils eosinophilia in the circulation but not in their tissues, indicating that migration of eosinophils from the circulation into tissues is regulated separately, by a second set of controls. The key molecules in this case are CC chemokines see Section Most of these cause chemotaxis of several types of leukocyte, but two are specific for eosinophils and have been named eotaxin 1 and eotaxin 2. The eotaxin receptor on eosinophils, CCR3, is a member of the chemokine family of receptors see Section The eotaxins and these other CC chemokines also activate eosinophils. Identical or similar chemokines also stimulate mast cells and basophils. For example, eotaxin attracts basophils and causes their degranulation, and MCP-1, which binds to CCR2, similarly activates mast cells in both the presence or absence of antigen. These findings show that families of chemokines, as well as cytokines, can coordinate certain kinds of immune response. A third set of controls regulates the state of eosinophil activation. In their nonactivated state, eosinophils do not express high-affinity IgE receptors and have a high threshold for release of their granule contents. The potential of eosinophils to cause tissue injury is illustrated by rare syndromes due to abnormally large numbers of eosinophils in the blood hypereosinophilia. These syndromes are sometimes seen in association with T-cell lymphomas, in which unregulated IL -5 secretion drives a marked increase in the numbers of circulating eosinophils. The clinical manifestations of hypereosinophilia are damage to the endocardium Fig. Hypereosinophilia can cause injury to the endocardium. The top panel shows a section of the endocardium from a patient with hyper-eosinophilic syndrome. There is an organized fibrous exudate and the underlying endocardium is thickened by fibrous tissue. Eosinophils and basophils cause inflammation and tissue damage in allergic reactions In a local allergic reaction , mast-cell degranulation and TH2 activation cause eosinophils to accumulate in large numbers and to become activated. Their continued presence is characteristic of chronic allergic inflammation and they are thought to be major contributors to tissue damage. Basophils are also present at the site of an inflammatory reaction. Basophils share a common stem-cell precursor with eosinophils; growth factors for basophils are very similar to those for eosinophils and include IL -3, IL-5, and GM-CSF. There is evidence for reciprocal control of the maturation of the stem-cell population into basophils or eosinophils. Basophils are normally present in very low numbers in the circulation and seem to have a similar role to eosinophils in defense against pathogens. Like eosinophils, they are recruited to the sites of allergic reactions. Eosinophils , mast cells, and basophils can interact with each other. Eosinophil degranulation releases major basic protein, which in turn causes degranulation of mast cells and basophils. An allergic reaction is divided into an immediate response and a late-phase response The inflammatory response after IgE -mediated mast-cell activation occurs as an immediate reaction , starting within seconds, and a late reaction, which takes up to 8â€”12 hours to develop. These reactions can be distinguished clinically Fig. The immediate reaction is due to the activity of histamine, prostaglandins, and other preformed or rapidly synthesized mediators that cause a rapid increase in vascular permeability and the contraction of smooth muscle. The late-phase reaction is caused by the induced synthesis and release of mediators including leukotrienes, chemokines, and cytokines from the activated mast cells see Fig. These recruit other leukocytes, including eosinophils and TH2 lymphocytes , to the site of inflammation. Although the late-phase reaction is clinically less marked than the immediate response, it is associated with a second phase of smooth muscle

contraction, sustained edema , and the development of one of the cardinal features of allergic asthma: A wheal-and-flare allergic reaction develops within a minute or two of superficial injection of antigen into the epidermis and lasts for up to 30 minutes. The late-phase reaction is an important cause of much serious long-term illness, as for example in chronic asthma. This is because the late reaction induces the recruitment of inflammatory leukocytes, especially eosinophils and TH2 lymphocytes , to the site of the allergen-triggered mast-cell response. This late response can easily convert into a chronic inflammatory response if antigen persists and stimulates allergen-specific TH2 cells , which in turn promote eosinophilia and further IgE production. The clinical effects of allergic reactions vary according to the site of mast-cell activation When reexposure to allergen triggers an allergic reaction , the effects are focused on the site at which mast-cell degranulation occurs. In the immediate response, the preformed mediators released are short-lived, and their potent effects on blood vessels and smooth muscles are therefore confined to the vicinity of the activated mast cell. The more sustained effects of the late-phase response are also focused on the site of initial allergen-triggered activation, and the particular anatomy of this site may determine how readily the inflammation can be resolved. Thus, the clinical syndrome produced by an allergic reaction depends critically on three variables: There are two main anatomical distributions of mast cells: If an allergen is introduced directly into the bloodstream or is rapidly absorbed from the gut, the connective tissue mast cells associated with all blood vessels can become activated. This activation causes a very dangerous syndrome called systemic anaphylaxis Acute Systemic Anaphylaxis, in Case Studies in Immunology , see Preface for details. Disseminated mast-cell activation has a variety of potentially fatal effects: This potentially fatal syndrome is called anaphylactic shock. It can occur if drugs are administered to people who have IgE specific for that drug, or after an insect bite in individuals allergic to insect venom. Some foods, for example peanuts or brazil nuts, can cause systemic anaphylaxis in susceptible individuals. This syndrome can be rapidly fatal but can usually be controlled by the immediate injection of epinephrine, which relaxes the smooth muscle and inhibits the cardiovascular effects of anaphylaxis. The most frequent allergic reactions to drugs occur with penicillin and its relatives. In people with IgE antibodies against penicillin, administration of the drug by injection can cause anaphylaxis and even death. Great care should be taken to avoid giving a drug to patients with a past history of allergy to that drug or one that is closely related structurally. This ring reacts with amino groups on host proteins to form covalent conjugates. When penicillin is ingested or injected, it forms conjugates with self proteins, and the penicillin-modified self peptides can provoke a TH2 response in some individuals. These TH2 cells then activate penicillin-binding B cells to produce IgE antibody to the penicillin hapten. Thus, penicillin acts both as the B-cell antigen and, by modifying self peptides, as the T-cell antigen. When penicillin is injected intravenously into an allergic individual, the penicillin-modified proteins can cross-link IgE molecules on the mast cells and cause anaphylaxis. Allergen inhalation is associated with the development of rhinitis and asthma Inhalation is the most common route of allergen entry. Many people have mild allergies to inhaled antigens, manifesting as sneezing and a runny nose. This is called allergic rhinitis, and results from the activation of mucosal mast cells beneath the nasal epithelium by allergens such as pollens that release their protein contents, which can then diffuse across the mucus membranes of the nasal passages. Allergic rhinitis is characterized by intense itching and sneezing, local edema leading to blocked nasal passages, a nasal discharge, which is typically rich in eosinophils, and irritation of the nose as a result of histamine release. A similar reaction to airborne allergens deposited on the conjunctiva of the eye is called allergic conjunctivitis. Allergic rhinitis and conjunctivitis are commonly caused by environmental allergens that are only present during certain seasons of the year. For example, hay fever is caused by a variety of allergens, including certain grass and tree pollens. Autumnal symptoms may be caused by weed pollen, such as that of ragweed. These reactions are annoying but cause little lasting damage. A more serious syndrome is allergic asthma, which is triggered by allergen-induced activation of submucosal mast cells in the lower airways Fig. This leads within seconds to bronchial constriction and increased secretion of fluid and mucus, making breathing more difficult by trapping inhaled air in the lungs. Patients with allergic asthma often need treatment, and asthmatic attacks

can be life-threatening. An important feature of asthma is chronic inflammation of the airways, which is characterized by the continued presence of increased numbers of TH2 lymphocytes , eosinophils, neutrophils, and other leukocytes Fig.

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Molecular and Biological Aspects of the Acute Allergic Reaction Molecular Events in Membrane Fusion Occurring During Mast Cell Degranulation Molecular and.

Atherosclerosis Atherosclerosis, formerly considered a bland lipid storage disease, actually involves an ongoing inflammatory response. Recent advances in basic science have established a fundamental role for inflammation in mediating all stages of this disease from initiation through progression and, ultimately, the thrombotic complications of atherosclerosis. These new findings provide important links between risk factors and the mechanisms of atherogenesis. Clinical studies have shown that this emerging biology of inflammation in atherosclerosis applies directly to human patients. Elevation in markers of inflammation predicts outcomes of patients with acute coronary syndromes, independently of myocardial damage. In addition, low-grade chronic inflammation, as indicated by levels of the inflammatory marker C-reactive protein , prospectively defines risk of atherosclerotic complications, thus adding to prognostic information provided by traditional risk factors. Moreover, certain treatments that reduce coronary risk also limit inflammation. In the case of lipid lowering with statins, this anti-inflammatory effect does not appear to correlate with reduction in low-density lipoprotein levels. These new insights into inflammation in atherosclerosis not only increase our understanding of this disease but also have practical clinical applications in risk stratification and targeting of therapy for this scourge of growing worldwide importance. A common example is hay fever , which is caused by a hypersensitive response by mast cells to allergens. Pre-sensitised mast cells respond by degranulating , releasing vasoactive chemicals such as histamine. These chemicals propagate an excessive inflammatory response characterised by blood vessel dilation, production of pro-inflammatory molecules, cytokine release, and recruitment of leukocytes. Myopathies[edit] Inflammatory myopathies are caused by the immune system inappropriately attacking components of muscle, leading to signs of muscle inflammation. They may occur in conjunction with other immune disorders, such as systemic sclerosis , and include dermatomyositis , polymyositis , and inclusion body myositis. In addition, diseases affecting the bone marrow may result in abnormal or few leukocytes. Pharmacological[edit] Certain drugs or exogenous chemical compounds are known to affect inflammation. Vitamin A deficiency causes an increase in inflammatory responses, [20] and anti-inflammatory drugs work specifically by inhibiting the enzymes that produce inflammatory eicosanoids. Certain illicit drugs such as cocaine and ecstasy may exert some of their detrimental effects by activating transcription factors intimately involved with inflammation e. Such an approach may limit side effects that are unrelated to the tumor of interest, and may help preserve vital homeostatic functions and developmental processes in the organism. According to a review of , recent data suggests that cancer-related inflammation CRI may lead to accumulation of random genetic alterations in cancer cells. DNA damages may cause genetic mutations due to inaccurate repair. In addition, mistakes in the DNA repair process may cause epigenetic alterations. Typically, several hundreds to thousands of genes are methylated in a cancer cell see DNA methylation in cancer. DNA repair genes, in particular, are frequently inactivated by methylation in various cancers see hypermethylation of DNA repair genes in cancer. A report [41] evaluated the relative importance of mutations and epigenetic alterations in progression to two different types of cancer. This report showed that epigenetic alterations were much more important than mutations in generating gastric cancers associated with inflammation. HIV and AIDS[edit] It has long been recognized that infection with HIV is characterized not only by development of profound immunodeficiency but also by sustained inflammation and immune activation. Animal studies also support the relationship between immune activation and progressive cellular immune deficiency: SIV sm infection of its natural nonhuman primate hosts, the sooty mangabey , causes high-level viral replication but limited evidence of disease. Recent studies demonstrated that caspase-1 -mediated pyroptosis , a highly inflammatory form of programmed cell death, drives CD4 T-cell depletion and inflammation by HIV. Pyroptosis appears to create a pathogenic vicious cycle in which dying CD4 T cells and

other immune cells including macrophages and neutrophils release inflammatory signals that recruit more cells into the infected lymphoid tissues to die. The feed-forward nature of this inflammatory response produces chronic inflammation and tissue injury. Such agents would almost certainly be used in combination with ART. Resolution of inflammation[edit] The inflammatory response must be actively terminated when no longer needed to prevent unnecessary "bystander" damage to tissues. Resolution of inflammation occurs by different mechanisms in different tissues. Mechanisms that serve to terminate inflammation include:

Chapter 5 : H2O - The Mystery, Art, and Science of Water: The Chemistry of Water

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Chapter 6 : Allergy - Wikipedia

Molecular and Biological Aspects of the Acute Allergic Reaction Modes of Action of Antigen-Antibody Reaction and Compound 48/80 in Histamine Release.