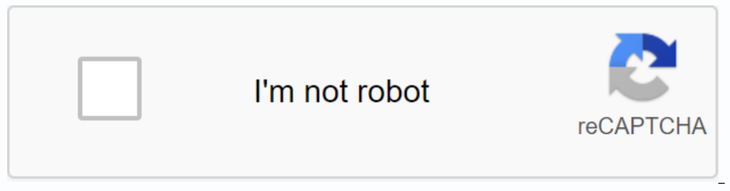

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Anaphylactic shock pathophysiology pdf

IgE-mediaated anaphylacsy is the classic form of anaphylacsy, which reveals an IgE antibody response in a sensitive antigen-sensitive individual, therefore. Antigen-specific IgE antibodies are then linked to mast cells and basophils. Later exposure to the sensitizing antigen causes cross-bonding of cell-bound IgE and causes mast cell (and/or basophil) degranulation. Other types of immunological anaphylacsy do not contain IgE. For example, anaphylacsy caused by the application of blood products, including intravenous immunoglobulin or animal antiserum, is at least partially due to complete activation. Immune complexes formed as in vivo or in vitro can activate complement steps. Some by-products of cascading plasma-activated complement 3 (C3a), plasma activated complement 4 (C4a) and plasma-activated complement 5 (C5a) are called anaphylatoxins and can be the main cell/basophyll degranulation. Mast cells and basophils are released by IgE- or non-IgE mediations degranulate, pre-educenine and newly created leucotrien, prostaglandins, and platelet activation factor (PAF). In classical form, mediator release occurs when the antigen (allergen) is linked to IgE, which previously sings antigens linked to sensitive basophils and mast cells. Mediators are released immediately when the antigen is connected. Some agents are thought to cause direct non-immunological release of mediators from mast cells, this process is not mediation by IgE. These include opioids, dextrans, protamine and vancomysin. The mechanisms underlying these reactions are not fully understood but may include specific receptors (e.g., opioids) or non-receptor-mediator mast cell activation (e.g., hyperosmolarity). Foods, Hymenoptera stings and intravenous (IV) contrast agents are the most common provocative substances in anaphylacside. Anaphylactic can also be idiopathic. Typical examples of IgE-median anaphylacsy include reactions to many foods, medications and insect bites. Hypersensitivity to food is a problem encountered in the industrialized world. [8] In the United States, nearly 4 million Americans have proven good food allergies. A study in Australia has shown that more than 10% of 12-month-olds challenge ige-median food allergies. [9] In Montreal, 1.5% of elementary school students were found to be susceptible to peanuts. Reactions to foods are thought to be the most common pre-hospital (outpatience) cause of anaphylacy. Some foods are more likely to reveal an IgE antibody response and lead to anaphylacticia than others. Possible foods that reveal an IgE antibody response in all age groups include peanuts, tree nuts, fish, and shellfish. It is likely to reveal an IgE antibody response in children also contains milk, eggs, wheat and soy. Over 32 deaths thought to be caused by foodborne anaphylacsy an analysis found that peanuts are responsible food in 62% of cases. Placebo controlled difficulties, peanut sensitive patients may react as little as 100 µg of peanut protein. [10] In harmony with previous studies, the Rochester Epidemiology Project found that food ingestion was the leading cause of anaphylacy, making up a third of all cases. [11] In the past, a history of IgE-mediated egg allergy has been a contraindication to receive the annual flu vaccine. A few years ago, individuals with egg allergies received the flu vaccine, but usually with a graded multi-dose protocol or based on the test to sew the skin for the vaccine itself. Given the recent evidence that individuals with egg allergies can safely receive the flu vaccine without the risk of systemic reactions according to the general population, the latest guidelines have now recommended that individuals with all egg allergies be vaccinated with a single dose flu vaccine. Furthermore, there is no role in the skin test because no evidence suggests that it identifies individuals at risk of a reliable systemic reaction. [12, 13] Scombroid fish poisoning can sometimes mimic foodborne anaphylacsy. Bacteria in degraded fish produce enzymes capable of decarboxing histidine to produce biogenic amines, including histamine and fog-uroconic acid, which are capable of mast cell degranulation. Most cases of IgE-median drug anaphylacsi in the United States are caused by penicillin and other beta-lactam antibiotics. Approximately 1 5000 exposure to the parenteral dose of penicillin or cephalosporin antibiotic causes anaphylacsy. Penicillin is metabolized into a large determinant, benzylpenicilloyl, and multiple small determinants. Penicillin and its metabolites are small molecules that reveal only one immune response when haptens conjuge with carrier proteins. Other beta-lactam antibiotics may cross-react with penicillins or may also have unique structures that act as haptens. Reactions to cephalosporins can occur in patients with penicillin allergies. In these patients, older agents such as cephalothhin, cephaloxin, sefoliyl, and cephazolin are more likely to collapse an allergic reaction than cefprozil, cefuroxime, ceftaziidime, or ceftriacone. This increased reactivity with older agents is due to the more antigenic similarity of the side chain not present with new second and third generation agents. A report suggested that the actual indentation of anaphylactics for cephalosporins in penicillin-anaphylactic patients was much lower than 10% frequently quoted-perhaps 1%, with most reactions considered mild. [14] A retrospective study evaluated 606 hospitalized patients with a history of penicillin allergies who were given cephalosporin. Only one patient (0.17%) had a reaction and was small. [15] In another article, patients with penicillin allergies seem to have a higher risk of subsequent reaction to any drug (about 3 factors) and an allergic risk Cephalosporins in patients with a history of penicillin allergy may be up to 8 percent higher in those with a history of penicillin allergies (i.e., at least part of the observed cross-reactivity may represent a general state of immune hypersupame, rather than actual cross-reactivity). [16] Pichichero reviewed complex literature and offered specific guidance for the use of cephalosporins in patients with a igE-median reaction to penicillin. [17] Patients with a history of positive skin tests for penicillin allergy are at high risk of subsequent reactions to penicillins. However, approximately 95% of patients with a history of penicillin allergy have negative skin tests and a low risk of reaction. Very low risk (1-2%) development of anaphylacsis for cephalosporins in patients with less defined reactions to penicillin. The rate of skin test reactivity in patients with known penicillin allergy is almost 50% of imipenem. In contrast, there is no known in vitro or clinical cross-reactivity between penicillins and aztreonam. When either penicillin or cephalosporin is the drug of choice for a life-threatening emergency patient, there are a number of options. When the date is unclear, the drug can be applied under close observation; however, where possible, obtain informed consent from the patient. Emergency treatment measures should be taken for anaphylacsi. Alternatively, when history is more persuasive, an alternative agent should be selected if it provides similar activity or follow a desensitization protocol. Many other drugs have been impliced in IgE-median anaphylacsy, albeit less frequently. In the surgical environment, anaphylactic reactions may most often be due to muscle relaxants but also due to hypnotics, antibiotics, opioids, colloids, and other agents. The prevalence of latex allergy was higher in the 1980s (due to hiv and hepatitis B and C outbreaks and universal measures taken), but the incidence of latex-free materials has decreased significantly since widespread use. If latex is responsible for anaphylacsia in the perioperative environment, reactions tend to occur during maintenance anesthesia, while other agents tend to cause reactions during anesthesia induction. Volatile anesthetic agents can cause immune-mediaked hepatic toxicity, but they are not involved in anaphylactic reactions. [18] Hymenoptera stings are a common cause of allergic reaction and anaphylactic. 0.5-3% of the U.S. population had a systemic reaction after being stung. [19] Hymenoptera poisoning in the United States results in fewer than 100 deaths a year. It is much more common than full-developed anaphylacsia after local reaction and urticaria Hymenoptera stings without other symptoms of anaphylacsia. Adults with generalized urticaria have a high risk of anaphylacsia with future stings, but a local reaction, severity is not a risk factor for anaphylacsy. Beware to treat patients and release them from the emergency department (ED) to prevent future exposure as much as possible after a section of generalized urticaria from anaphylacsy or hymenoptera envenomation. Consider contacting an allergist for desensitization, especially when more exposure is likely. Also, consider prescribing a treatment kit with an epinephrine autoinjector and oral antihistamine. Both are effective measures for preventing or improving future reactions. Alergen-

specific subcuttant immunotherapy (SCIT) can cause IgE-median anaphylacsy. Allergy injections are a common trigger for anaphylacy. This is not unexpected because the treatment is based on injecting an allergen that the patient is sensitive to. However, life-threatening reactions are rare. Three studies show that deaths from SCIT occurred at a mortality rate of about 1 per 2,500,000 injections. [20, 21, 22] A total of 104 deaths were reported due to SCIT and skin tests between 1945 and 2001. Risk factors for immunotherapy-related severe anaphylacsis include poorly controlled asthma, simultaneous use of beta-blockers, high dose of allergen, errors in the application and lack of adequate observation time after injection. Fatal reactions close to subcuttant immunotherapy (NVR) were also retrospectively examined. Of the 646 allergist-immunologists who responded to a survey on reactions, 273 reported NVR. Researchers have identified an NFR as respiratory reconciliation, hypotension or both and require immediate epinephrine. Hypotension was reported in 80% and respiratory failure in 10% of NDRs was seen only in individuals with asthma. Epinephrine was delayed or not applied in 6% of these cases. Reactions to aspirin and non-steroidal anti-inflammatory drugs (NSAIDs) in the past have been classified as IgE-independent because they are thought to be caused by abnormal metabolism of arachidonic acid. Aspirin/NSaid and bronchospasm isolated cutaneous reactions in aspirin-sensitive asthmatics (usually in association with nasal polyposis) are really done through IgE-independent mechanisms. With these drugs, the closure of cyclooxygenase causes the prostanoid road to be closed and causes over-production of leucotriens through the 5-lipoxygenase pathway. These patients have marked cross-reactivity between aspirin and most NSaid. Anaphylaccia occurs after taking these drugs, however, apparently through a different mechanism that is more consistent with igE-median anaphylacsi. With real anaphylactic acid, different cyclooxygenase inhibitors do not appear to react diagonally. Anaphylacsy occurs only after exposure to 2 or more implied drugs, suggesting a need for previous sensitivity. Finally, patients with real anaphylacsia usually do not have underlying asthma, nasal polyposia, or urticaria. In a study of about 52,000 people taking NSaid, 35 developed anaphylactic shock. Conversion of angiotensin Commonly used (ACE) inhibitors in the treatment of hypertension are associated with angioedema in 0.5-1.0% of patients who receive them. Systemic anaphylacsy is rarely associated with these agents. Anaphylacsy can be caused by the application of blood products, including iv immunoglobulin or animal antiserum, at least partly as a result of complementary casing adaptation. Cascading is capable of causing some side mast cell/basophyll degranulation. (See Pathophysiology.) Exercise-related anaphylacsis is a rare syndrome that can take 1 out of 2 form. The first form is food dependent, requiring exercise and last inging of certain foods (e.g., wheat, celery) or medications (eg, NSaid) caused by a section of anaphylacy. In these patients, they do not produce a section of exercise alone, and, similarly, the culprit does not cause a section of digesting food or medication alone. The second form is characterized by intermittent episodes of anaphylacy during exercise, independent of swallowing any food. Anaphylacsi does not necessarily occur during each episode of physical exertion. Anaphylacsis can be a manifestation of systemic mastocytsis, a disease characterized by excessive mast cell load in multiple organs. Such patients seem to be at high risk for food and poison reactions. Alcohol, vancomysin, opioids, radiocontrast media and other biological agents that can directly separate mast cells are generally not recommended in these patients. Opioids are thought to cause direct, non-immunological release of mediators from mast cells of some agents such as desharsan, protamine and vancomysin. There is also evidence that dextrans and protamine can activate various inflammatory pathways, including complement, clotting, and vasoactive (kallikrein-kinin) systems. Intravenously administered radiocontrast media causes an anaphylactic reaction that is clinically similar to actual anaphylacsy and treated in the same way. The reaction is not related to prior exposure. Approximately 1-3% of patients with hyperosmolar IV contrast can react. Reactions to radiocontrast are usually mild (most commonly urticaria), only rare deaths have been reported. The risk of a fatal reaction was estimated at 0.9 cases per 100,000 exposures. Pre-treatment with the use of antihistamines or corticosteroids and low molecular weight (LMW) contrast substances leads to lower rates of anaphylactic reactions to the IV radiocontrast medium (about 0.5%). Since the recurrence rate is estimated at 17-60%, consider these measures for patients with a history of reactions before. Some agencies only use LMW agents. Personnel, medications, and equipment necessary for the treatment of allergic reactions should always be present when these agents are administered. Get approval before management. Patients with atopic and/or asthma also have an increased risk of reaction. In addition, taking this allergic reaction is more difficult to treat Shellfish or iodine allergy iv is not a contraindication for contrast use and a pre-treatment regimen is not mandatory. As with any allergic patient, consider the use of LMW contrast materials. In fact, term iodine allergy is a misnam. Iodine is an important trace element found in the body. No one's allergic to iody. Patients who reported iodine allergy often had either a previous contrast reaction, a shellfish allergy, or a contact reaction to povison-iodine (Betadine). Mucosal exposure to radiocontrast agents (e.g., GI, geniourinary [GU]) has not been reported to cause anaphylacsis; Therefore, the previous reaction history is not a contraindication for the use of GI or GU of these agents. Idiopathic anaphylactic syndrome is a recursing anaphylacsi syndrome in which consistent triggers can be determined despite extensive research. [23] This recurrencing syndrome should be distinguished from a single ancestor of anaphylacsis, where its etiology may be uncertain. Idiopathic anaphylacsia can be classified as sparse (6 attacks <per year) or frequent (≥6 attacks per year or 2 or more episodes in the last 2 months). [23] An approach is a long-term conical prednion for individuals with sparse episodes, expected treatment with epinephrine, antihistamines and prednizone and prednizone, and for those with frequent episodes. Most of these patients are women, and atopia seems to be an underlying risk factor. Two-thirds of patients have 5 or fewer attacks a year, and one-third have more than 5 attacks a year. A sub-population of women develops anaphylaccy in relation to the menstrual cycle; This phenomenon is known as catamenial anaphylacsi. [24, 25] In severe cases, these patients require medical pituitary suppression and even manipulation of hormonal levels with oofhorektomy. Most of these patients react to shifts in progesterone levels, and diagnosis can be confirmed by provoking an anaphylactic event through the administration of low doses of progesterone. The indentency of bifhasic anaphylacsi varies from 1% to a further 23%. In addition, the initial time of the late phase can range from 1 to 72 hours (most occur within 8-10 hours). Potential risk factors include severity of the first phase, delayed or suboptimal doses of epinephrine during initial treatment, laryngeal edema or hypotension in the first stage, delayed onset of symptoms after exposure to the culprit antigen (usually a food or insect sting), or a history of biphasic anaphysics. [26] Permanent anaphylactic, anaphylactic, which can last 5-32 hours, occurred in 7 25 cases (28%), stark and sullivan report, 2 dead. [27] Three (23%) of the 13 cows analyzed in a report on deadly or fatal anaphylacsy to food had similarly permanent anaphylaks. [28] However, retrospective data from other researchers show that persistent anaphylacsy is rare. Neither biphasik nor biphasik Anaphylactic is predictable from the severity of the first phase of an anaphylactic reaction. Since life-threatening symptoms of anaphylactic may recur, patients may need to be monitored 24 hours or more after significant improvement from the first stage. [26] When prescribing epinephrine, all patients should always be taught to have 2 injectors at hand. As mentioned above, atopia is a risk factor for anaphylacsi. The Rochester Epidemiology Project found that 53% of anaphylactic patients had a history of atopic disease (e.g., allergic rhinitis, asthma, atopic dermatitis). [11] Atopia was detected in 37% of patients in the Memphis study. [29] Other studies have shown to be a risk factor for anaphylacsy from foods, exercise-induced anaphylacsy, idiopathic anaphylacsy, radiocontrast reactions, and latex reactions. The underlying atopy does not appear to be a risk factor for penicillin or insect sting reactions. The route and timing of the application affects anaphylactic potential. The oral route of the application is less likely to cause a reaction, and although such reactions usually occur after the ingesting of foods by someone with an allergy to less severe, fatal reactions. The longer the interval between exposures, the less likely it is to repeat an IgE-median reaction. This is thought to be due to catabolism and decreased igE synthesis specific to the allerge over time. This doesn't seem to be the case for IgE-independent reactions. Retrospective emergency department work on 302 patients admitted with anaphylacsy found that antihypertensive pharmacotherapy, which was taking at least 1 antihypertensive drug from 87 (29%) of them, increased the risk of intervention and hospitaly in the organ system. [30, 31] When ACE inhibitors, beta blockers, diuretices or any antihypertensive drugs were used, the risk of involvement in 3 or more organ systems increased more than 2-fold. Many of these agents were also associated with an increased risk for in-bed admissions. [30, 31] 31]