South American Publishing

SAP Dentistry. 2025; 3:199
doi: 10.62486/dent2025199

REVIEW

Anaphylactic shock: a review of the pathophysiology and therapeutic management

Shock anafiláctico: revisión sobre la fisiopatogenia y el manejo terapéutico

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Cite as
De María A, Verdú S, Vela Ferreira A, Zunini G, Baggini E, Brusca MI. Anaphylactic shock: a review of the pathophysiology and therapeutic management. SAP Dentistry. 2025; 3:199. https://doi.org/10.62486/dent2025199

Publication History
Submitted: 24-03-2024
Revised: 25-06-2024
Accepted: 15-10-2024
Published: 01-01-2025

ABSTRACT

Introduction: anaphylactic shock is a severe allergic reaction that can occur rapidly and be life-threatening. Understanding it is crucial for medical care, especially in settings where medications are administered or procedures are performed that can trigger allergic reactions.
Objective: to review the etiological, pathophysiological, clinical, and therapeutic features of anaphylactic shock.
Development: the most common causes of anaphylactic shock include allergies to food, insect stings, medications, and latex. Pathophysiologically, it is characterized by massive release of inflammatory mediators such as histamine, leading to vasodilation, increased vascular permeability, and bronchoconstriction. Clinically, it presents with symptoms such as urticaria, edema, respiratory distress, and hypotension. Diagnosis is based on clinical history and symptom presentation. Immediate treatment includes the administration of adrenaline, which acts as a vasoconstrictor and bronchodilator, and may be followed by antihistamines and corticosteroids.
Conclusions: anaphylactic shock is a medical emergency that requires immediate recognition and treatment. Education and preparation of medical personnel are essential to ensure an appropriate response to this condition. Early identification of triggers can prevent future episodes and improve patient outcomes.

Keywords: Shock; Anaphylaxis; Treatment; First Aid; Allergy.

RESUMEN

Introducción: el shock anafiláctico es una reacción alérgica severa que puede ocurrir de forma rápida y potencialmente mortal. Su comprensión es crucial para la atención médica, especialmente en entornos donde se administran medicamentos o se realizan procedimientos que pueden desencadenar reacciones alérgicas.
Objetivo: revisar las características etiológicas, fisiopatológicas, clínicas y terapéuticas del shock anafiláctico.
Desarrollo: las causas más comunes del shock anafiláctico incluyen alergias a alimentos, picaduras de insectos, medicamentos y látex. Fisiopatológicamente, se caracteriza por la liberación masiva de mediadores inflamatorios como histamina, lo que provoca vasodilatación, aumento de la permeabilidad vascular y broncoconstricción. Clínicamente, se presenta con síntomas como urticaria, edema, dificultad respiratoria y hipotensión. El diagnóstico se basa en la historia clínica y la presentación de síntomas. El tratamiento inmediato incluye la administración de adrenalina, que actúa como un vasoconstrictor y broncodilatador, y puede ser seguido por antihistamínicos y corticosteroides.
Conclusiones: el shock anafiláctico es una emergencia médica que requiere reconocimiento y tratamiento inmediatos. La educación y preparación del personal médico son fundamentales para garantizar una respuesta adecuada ante esta condición. La identificación temprana de los desencadenantes puede prevenir episodios futuros y mejorar los resultados del paciente.

Palabras clave: Shock; Anafilaxia; Tratamiento; Primeros Auxilios; Alergia.

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INTRODUCCIÓN

Anaphylactic shock is a severe and unexpected allergic reaction that occurs rapidly and can be life-threatening. Its immediate clinical diagnosis is crucial, as it affects several vital body systems, including the cutaneous, respiratory, gastrointestinal, and cardiovascular systems.[1]

In recent years, the incidence of anaphylactic shock has increased, highlighting the need for dental professionals to understand its signs and symptoms. These usually manifest immediately after antigen administration. The main clinical manifestations include skin pruritus, edema, and urticaria, as well as marked hypotension and respiratory distress. In some cases, gastrointestinal symptoms such as abdominal pain, vomiting, and diarrhea may also occur. It is important to note that anaphylactic shock can be biphasic, meaning that even if the initial episode is controlled, it can recur up to 72 hours later.[2]

Adrenaline is the drug of choice for the treatment of anaphylactic shock. Its α-adrenergic properties induce vasoconstriction, which improves peripheral vasodilation and relieves hypotension, erythema, urticaria, and angioedema. In addition, their β-adrenergic effect causes bronchodilation, increases cardiac output and contractility, and prevents the release of mediators from mast cells and basophils, thus improving coronary blood flow.[3]

Dentists need to be familiar with the preparation and presentations of adrenaline. Ampoules of adrenaline that must be loaded into a syringe, as well as auto-injection devices, are available on the market.[3]

Anaphylaxis is a condition that requires immediate assistance in a variety of settings, such as doctors' offices, hospitals, schools, homes, or public spaces. Therefore, dentists must recognize that anaphylactic shock is a severe allergic reaction that can be fatal and act without hesitation in its treatment.

Therefore, it is of utmost importance that students, as future health professionals, know anaphylactic shock and the proper use of medications to treat it.[4]

Refractive work was carried out to review the etiological, pathophysiological, clinical, and therapeutic characteristics of anaphylactic shock.

METHOD

A search for information was conducted in Redalyc, Elsevier Science Direct, PubMed/Medline, and SciELO, as well as in the ClinicalKeys services and the Google Scholar search engine. Advanced search strategies were used to retrieve the information by structuring search formulas using the terms "shock," "anaphylaxis," "treatment," "first aid," "allergy," etc., as well as their equivalents in English. Those providing theoretical and empirical information in Spanish or English language were selected from the resulting documents.

DEVELOPMENT

Anaphylaxis is an allergic reaction that occurs unexpectedly and rapidly, constituting a true medical emergency that will compromise the patient's life. It differs from other allergic reactions because it will affect the whole organism; that is, it is systemic and presents symptoms in several systems, such as the respiratory system producing respiratory difficulty and the cardiovascular system producing hypotension.[1,2,5,6] It is an acute immunoglobulin E (IgE)-mediated reaction that will occur in patients previously sensitized to the antigen and whose clinical picture presents immediately after contact with the allergen, ranging from mild manifestations such as a skin reaction to an anaphylactic shock reaction. Recognition of the milder signs and symptoms is important as these can progress to a fatal outcome.[6] Anaphylactic shock represents a type I hypersensitivity reaction in which three components are involved:

- The allergen - antigen.
- Antibodies - immunoglobulin E (IgE).
- Mediators - mast cells and basophils.

Causes

Anaphylaxis is multifactorial, and its etiology varies according to regions, cultural and ethnic differences, age groups, diet, and way of food preparation. Food is the main trigger of anaphylaxis in children and adolescents, while it is medication and insect venoms in adults. Cases that, after a complete allergological study, fail to identify the cause and there is no other triggering factor, are classified as idiopathic. Latex can be implicated in health personnel and hospital environments, besides being responsible for several cases of perioperative anaphylaxis. Pharmaceutical formulations, cosmetic products, and processed foods should also be considered possible triggers. The most common causes are as follows:

- Drug-induced: the most common causes of an anaphylactic reaction are drugs, among which we have antibiotics, the most important of which are beta-lactams such as penicillins and, to a lesser extent, anti-inflammatory drugs such as aspirin.
- Induced by insect bites: the second cause of anaphylactic shock is insect stings such as bees, wasps, and ants that affect 3 % of the population. This type of reaction, in the worst case, can cause death in 30 minutes if not treated in time with the use of self-injectable adrenaline.
- Induced by food and others: other causes of vital importance are dust, pollen, and certain foods such as nuts and shellfish. Food allergy is more frequent in children and young people.
- Latex induced: material present in gloves or rubber dam used for isolation of the operative field in patients who already know they are allergic to this material should be treated in environments free of this material, including gloves.
- Induced by anesthesia: the causal agent of the allergy is sodium bisulfite, which is the preservative of local anesthetics with vasoconstrictor; up to 4 % of these reactions are fatal.

Table 1. Etiology anaphylactic shock	
Medicines and diagnostic 46,7 to 62 %	Beta-lactam antibiotics, non-steroidal anti-inflammatory drugs, contrast media.
Food 22 to 24 %	Fruits, nuts, seafood and fish, egg, milk.
Insect bites 8 % to 13 %	
Physical factors 3 % to 4 %	
Latex 7 %	
Idiopathic 3 % to 5 %	
Source: [7]	

Physiopathology

Hypersensitivity reactions can be described by the Gell and Coombs classification, which systematizes them depending on their time scale and etiology in four groups, but they are not necessarily independent. Between 60 % and 70 % of allergic anaphylaxis during anesthesia are mediated by IgE and correspond to a type I immediate hypersensitivity reaction according to this classification. The remainder is mediated by IgG and complement-related immune complexes.[6]

IgE-mediated anaphylaxis

To be at risk for type I hypersensitivity reactions, an individual must have been sensitized to an antigen through a previous exposure. The first exposure must have produced an antigen-specific immunoglobulin E (IgE) and the ability to produce that IgE maintained in plasma cell memory. Hypersensitivity occurs when a subsequent exposure to the same antigen induces large-scale IgE production, which binds to receptors on the surface of mast cells and basophils. The interaction of IgE bound to mast cells and basophils with their antigen induces degranulation and release of inflammatory mediators, which cause altered capillary permeability (urticaria, edema), vasodilatation, bronchoconstriction, hypotension with tachycardia and other signs and symptoms. Among the preformed mediators are those of the protease family and proteoglycans: tryptase, kinase, carboxypeptidase, and heparin. Among the neoformed mediators, the stimulation of the synthesis of lipid metabolites occurs mainly in prostaglandins and leukotrienes. Platelet-activating factors, serotonin, bradykinin, calcitonin, and nitric oxide have also been implicated in shaping the clinical picture of anaphylaxis. The effects of histamine, associated with those of the other preformed and neoformed mediators, can lead to anaphylactic shock, the most severe clinical manifestation of anaphylaxis.[2,5,6]

Hypersensitivity

Hypersensitivity refers to excessive or inappropriate immune response to environmental antigens, usually nonpathogenic, causing tissue inflammation and organ malfunction. Gell and Coombs classified the immunopathogenic mechanisms or hypersensitivity reactions into four types, each of which sequentially involves different cell types and soluble mediators.[2,5,6]

- Type I hypersensitivity: these are reactions in which Ag (antigen) combine with specific immunoglobulins (Ig) E that are fixed by their Fc end (crystallizable fraction) to receptors of the membrane of mast cells and basophils of peripheral blood.
- Type II hypersensitivity: these reactions are mediated by the interaction of Ig G and Ig M performed with Ag on the cell surface and other tissue components.
- Type III hypersensitivity: these are reactions produced by circulating immunocomplexes (IC) of Ag-Ag, which, when deposited in tissues, cause phagocyte activation and tissue damage.
- Type IV hypersensitivity: these are cellular or cell-mediated hypersensitivity reactions caused by sensitized T lymphocytes upon contact with the specific Ag. These can produce an immunological lesion by direct toxic effect or by releasing soluble substances (lymphokines).

Hypersensitivity Type I

Corresponds to immediate hypersensitivity reactions that occur within 15 minutes from the interaction of Ag with preformed Ig E in persons previously sensitized to that antigen.

First, the Ag enters through the skin or mucous membranes of the respiratory tree or gastrointestinal tract and is captured by Ag-presenting cells, which stimulate Th2 lymphocytes to secrete a pattern of cytokines that in turn stimulate Ag-specific B-lymphocytes to produce specific Ig E; this Ig E binds to mast cell and basophil receptors.

Sensitization to the allergen occurs in this first stage; when again exposed to the Ag, binding the Ag to the specific Ig E attached to the membrane of these cells occurs and leads to degranulation. This results in the release of vasoactive and inflammatory mediators (histamines, chemotactic factors, leukotrienes, platelet-activating factor) that cause vasodilatation, increased capillary permeability, glandular hypersecretion, smooth muscle spasm, and tissue infiltration of eosinophils and other inflammatory cells, responsible for the symptomatology.

These early reactions are accompanied within 2 to 4 hours by a late phase reaction that occurs after contact with Ag, with infiltration of inflammatory cells.[2,5,6]

Clinical manifestations

Ig E-mediated inflammation is responsible for reactions that are usually localized, affecting a particular organ, e.g., respiratory

tract, digestive tract, or skin, or cause systemic reactions such as anaphylactic shock.[2,5,6]

Clinical picture

The main manifestations of anaphylactic shock occur in places with a higher concentration of mast cells, such as skin and mucous membranes, lungs, heart, and gastrointestinal tract. He states that the clinical picture is variable and difficult to differentiate from other adverse reactions, which will complicate its diagnosis and, therefore, delay its treatment, which may result in unfortunate consequences.[2,8]

When the allergen is administered parenterally, this type of allergic reaction occurs within seconds or minutes, whereas when administered orally or topically, these symptoms take longer to manifest. Among the most important are those involving the respiratory and cardiovascular systems, which is why they should be treated earlier).[2,8,9,10]

The symptomatology of an allergic event is neither uniform nor constant; sudden life-threatening attacks may occur or manifest with more or less overlapping pictures. Signs and symptoms can be classified according to the severity based on the type of system affected. Thus, we have:

Grade I: presents mucocutaneous manifestations.

Grade II: mucocutaneous manifestations accompanied by respiratory and cardiovascular symptoms.

Grade III: cardiovascular collapse.

Grade IV: cardiac arrest.

Grade V: death.[2,8,9,10]

Signs and symptoms of anaphylactic shock:

In addition, it should be taken into account that these reactions are biphasic, i.e., they can occur up to 72 hours after successful resolution of the initial reaction. However, the common occurrence is within the next 8 hours. Therefore, it is considered important that the patient who has suffered from an anaphylactic reaction spend a period of observation under appropriate medical personnel.[5,8]

Diagnosis

The dentist and his auxiliary team should gather a series of theoretical and practical knowledge to resolve emergencies that may occur in a dental office. Untrained personnel, lack of equipment, and limited physical space add to the lack of knowledge of each dentist about the basic maneuvers and critical pharmacology to be used in the face of these complications.[11]

Immediately, there is no medical test that can help us recognize anaphylactic shock; we will only base ourselves on the clinical suspicion presented by the patient.[8,12]

The most important step in the dental consultation to avoid such an emergency is prevention. This is achieved using a physical evaluation of the patient before dental treatment, a clinical history questionnaire, a history of dialogue, a physical examination, and a risk assessment.[13]

The main manifestations presented by the patient are the beginnings of anaphylactic shock, such as pruritus.[14]

There are laboratory tests that can help us diagnose the causative agent of the immune response, but these would take too long and would not be of great use at the moment since we must act quickly. The laboratory test that can help us once the situation has been managed is tryptase, which is the most commonly used. Increased tryptase values (>25 ug/L) indicate a diagnosis of anaphylaxis; tryptase reaches its maximum value after 30 minutes and remains elevated for up to 6 hours.[2,8,9,10]

Anaphylactic shock may be considered to be present if the patient presents within minutes or hours after administration of a drug with one of the three criteria present:

- Skin and mucosal involvement: pruritus, generalized urticaria, redness or swelling of the lips, tongue, or uvula.
- Respiratory involvement: dyspnea, wheezing, stridor, decreased airflow, hypoxemia.
- Hypotension or evidence of target organ dysfunction, hypotonia, syncope, and incontinence.

Treatment

Intramuscular adrenaline is the recommended first-line medication for the treatment of anaphylactic shock. Worldwide, there is alarming data on the lack of knowledge and incorrect use of rescue measures in anaphylaxis. In Argentina, a study conducted by Lucini et al. in 2006 has shown that 51 % of emergency physicians do not know the dosage of adrenaline in situations of anaphylactic shock. The availability of adequate rescue medication in emergency rooms in our country should also be studied. In Latin America, the use of adrenaline seems to be below adequate standards, with use in only 36 % of patients. In Australia, Braganza et al., in a sample of patients captive of the health system, showed that only 39,3 % of patients with anaphylactic shock received adrenaline before or during hospital admission. Confirming these data, Brown et al.[1] indicate that less than 60 % of patients with anaphylactic shock receive adrenaline as the first treatment measure. In another study conducted in Great Britain, it has been shown that, in 142 fatal cases of anaphylaxis, only 14 % of the patients received adrenaline before cardiorespiratory arrest. Furthermore, in recent years, there has been a considerable increase in the prevalence of anaphylactic shock in countries such as the United Kingdom and better identification of patients under one year of age.[2,15]

Route of administration: currently, the recommended route of administration is intramuscular. Individuals with anaphylaxis who received subcutaneous adrenaline achieved plasma concentrations of 1802 ± 214 pg/ml in a mean time of 34 ± 14 minutes, while in those who received it intramuscularly, the concentration obtained was 2136 ± 351 pg/ml in a mean time of 8 ± 2 minutes.

The appropriate sector for administration is the area of the vastus externus of the quadriceps between the anterior midline and the external lateral line. For reasons not yet established, this sector is associated with the best adrenaline absorption and is the most comfortable for the patient.[1,8,16,17]

Preparation of adrenaline: adrenaline can be used by loading the ampoule into a tuberculin syringe. With the syringe preloaded manually, there is a risk of accelerated inactivation of the adrenaline, the main cause of which is heat. The ampoules' " caramel " color delays light-induced drug degradation. An ampoule/syringe is inexpensive. However, under controlled conditions, the filling of a syringe with an adrenaline ampoule differs greatly between trained and untrained persons, such as the patients themselves. This point is crucial because a delay in the application of adrenaline can be life-threatening for the patient. In addition, accidents related to falling ampoules, syringes, finger pricks, etc., are frequent and constitute a risk for patients. However, in highly trained people, especially in emergency rooms, intensive care areas, or operating rooms, this system has the advantage of being low-cost and easy to administer.[3,13,17]

In recent years, adrenaline auto-injection devices have appeared in the Argentine market. They still need to be widely available; they have the disadvantage of having a short expiration period and a very high cost. However, this medication allows a simple and safe application in patients over 10 kg of weight. It does not require filling any syringe and is ready to use. Doses are available in 0,15 mg and 0,30 mg of adrenaline. Although the auto-injector has limitations and its design could be improved for better handling, its use is useful for the immediate rescue of patients suffering from anaphylactic shock. Current devices are safer, with longer needles to ensure intramuscular application in people with more abundant subcutaneous cellular tissue. When the device is removed after application, the needle retracts and avoids accidental punctures.[3,13,17]

Use of the adrenaline autoinjector:

1. Grasp the unit with the orange tip pointing down.
2. Form a fist around the unit.
3. With your other hand, remove the blue activation cap.
4. Place the orange tip near the anterolateral region of the thigh.
5. With a quick movement, push the autoinjector firmly against the thigh so that the unit is at 90° (perpendicular) to the thigh.
6. Hold the applicator for 10 seconds.
7. Remove the device. Massage the area. The needle will retract into the orange tip of the device.
8. If the medication was correctly administered, the clear window will darken.
9. Place the device in its plastic case and take it to the hospital.

Each ampoule of adrenaline contains a concentration of 1 mg/ml. The correct dose per kilogram of body weight is 0,01 mg/kg body weight. It is also possible to dilute 1/10 and use a dose of 0,1 ml per kilogram of body weight, which makes the dosage more easily calculated and managed for administration. However, making dilutions delays the application of the medication and can lead to errors in preparation.[3,8,13,16,17]

Early recognition of initial symptoms is paramount, as early medication administration can make a major difference in the further development of the condition. Severe cutaneous symptoms are usually of very rapid onset, with the spread of hives, the presence of angioedema, and, characteristically, pruritus of palms and soles.

Respiratory symptoms may be associated with the occurrence of bronchospasm and asphyxia. Hypotension, tachycardia, and infarction symptoms are the predominant cardiovascular symptoms.[3,13,17]

The need for post-treatment clinical observation of the patient with anaphylactic shock is imperative because of the possibility of delayed reaction and biphasic anaphylaxis.

Adrenaline does not present any contraindication for its use against a precise indication. There are some conditions in which auto-injectors are an absolute indication if appropriate to the patient's weight and availability.[3,13,17]

Education of students and professionals. Knowledge of anaphylactic shock treatment and dissemination of treatment guidelines should be directed toward students and professionals. Thus, dissemination of knowledge through all available means should be considered.

The essential objectives for the successful rescue of a patient with an episode of anaphylaxis are:

1) early recognition of the severity of the clinical picture;
2) early request for specialized help;
3) immediate initiation of treatment with adrenaline.

To achieve these objectives, a triad is proposed, consisting of an alert phase, an alarm phase and an action phase.

- Alert phase: corresponds to the period before a serious situation to take general precautions to avoid possible misfortunes. Incorporation of the clinical history with precise patient information (identification, allergological diagnosis, therapeutic recommendations, etc.) Preparation of the professional and clinical recognition of anaphylactic shock and the appropriate protocols for its rescue.
- Alarm phase: these are the signals that inform about a threat's real or imminent presence.
- Action phase: once the alarm phase has been triggered, the established protocol should be followed.

Care in the use of adrenaline:

As mentioned above, intramuscular adrenaline is the drug of first choice in the treatment of anaphylactic shock. Therefore, studies carried out in different countries have shown that most dentists do not know how to use it or have not used it as the first-line drug to treat anaphylactic shock. Therefore, it is important to review the correct and up-to-date use of adrenaline in the dental office.[13]

First aid kit

It is important and necessary to have a first aid kit for medical emergencies in the dental office, but more important is that the dentist knows how to handle the different components of the kit, especially the drugs.

It is recommended that the emergency kit should be mobile and transportable with easy access to oxygen apparatus and should include a manual with emergency phone numbers, names of people responsible for updating the emergency kit, and emergency protocols. The dentist can choose only those materials with which they are familiar and ready for use; it is not recommended that there be numerous injectable medications since, in the dental office, most emergencies can be controlled without the use of these, except anaphylactic shock in this case if we would need an injectable solution of epinephrine, corticosteroid, and antihistamine.

Drugs

The following table will detail the essential drugs to treat anaphylactic shock within the dental office:

Table 2. Medications for anaphylactic shock and their dosages

Medication	Dosis
Epinephrine	0,3 mg 1:1000 adults 0,25 mg 1:1000 6-12 years old 0,12 mg 1:1000 6 months-6 years
Diphenhydramine	Mild: adults: 25-50 mg every six to eight hours Children > 10 kg: 12,5-25 mg three to four times / day Moderate: 25-50 mg IM
Saline	20 ml/kg in the first 5 to 10 minutes
Albuterol	Children: one spray 90 mcg/spray Adult: two sprays 90 mcg/spray
Methylprednisolone	50-100 mg IV
Inhaled salbutamol	100-200 ug
Hydrocortisone	100-500 mg IM

Source: [5]

CONCLUSIONS

The topic review highlights the importance of continuing education in managing anaphylactic shock, given its increasing incidence in diverse populations. Identifying early symptoms and acting quickly can make the difference between life and death. In addition, healthcare professionals must be familiar with available treatments and understand the underlying pathophysiology to address better complications that may arise. Implementing standardized protocols in clinics and hospitals can facilitate a rapid and effective response. Likewise, patient education on avoidance of known triggers and appropriate use of adrenaline auto-injectors is crucial for the long-term management of those with a history of anaphylaxis. Finally, research into new treatments and preventive approaches that can reduce the incidence of anaphylactic shock, thereby improving the quality of life of affected patients, should be encouraged.

FINANCING

The authors did not receive funding for the development of this research.

CONFLICT OF INTEREST

The authors declare that there is no conflict of interest.

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