

Anaphylaxis Management in Dental Practice

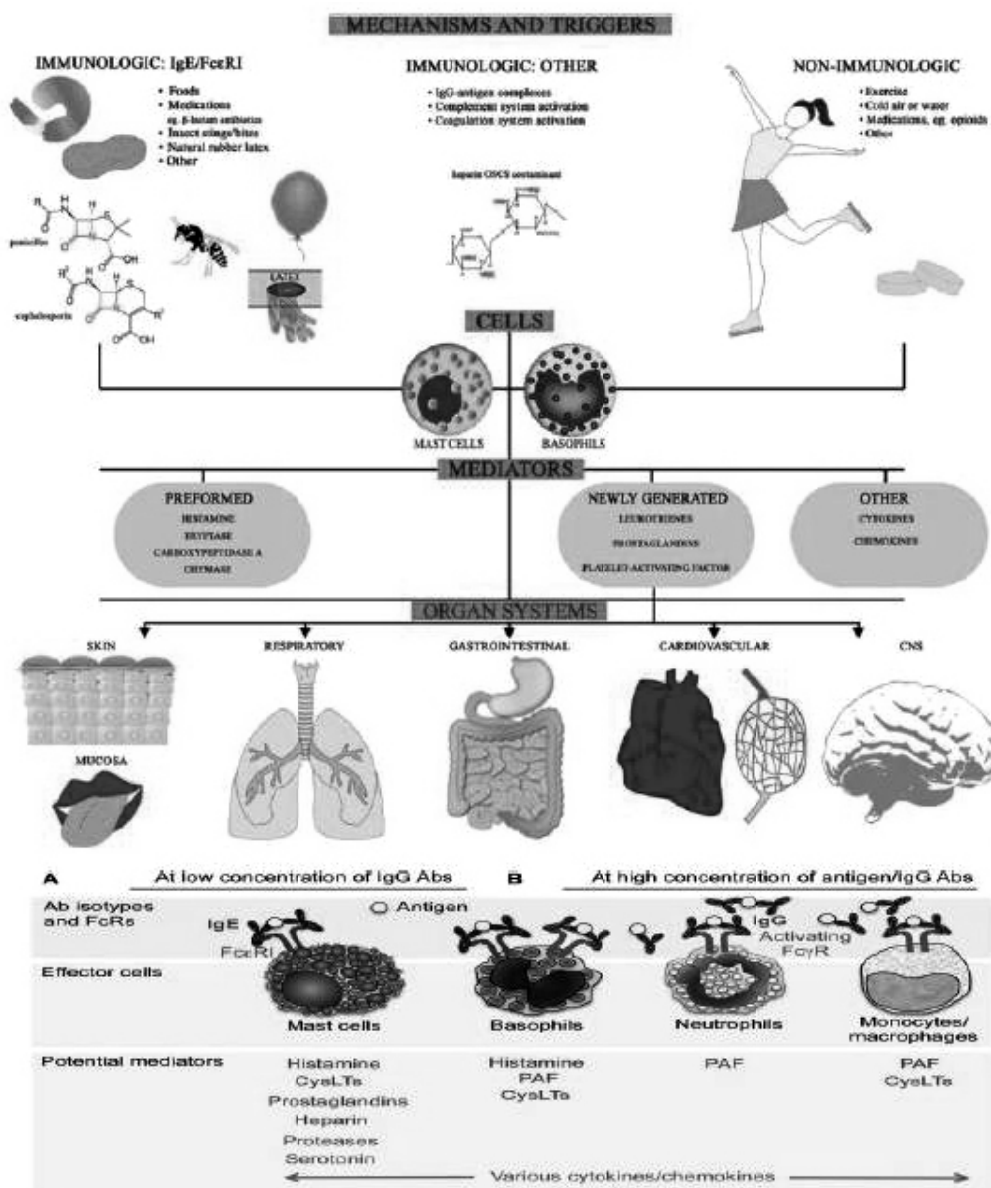
Dr. S. Malathi¹, Dr. C. Ravindranath¹, Dr. M. Vaishnavi²

¹Associate Professor, Department of Medicine, ²Junior Resident,
Tamil-Nadu Government Dental College and Hospital, Chennai.

Introduction:

Anaphylaxis is defined as “an acute potentially life-threatening hypersensitivity reaction, involving the release of mediators from mast cells, basophils, and recruited inflammatory cells.”

Vasoactive mediators actively released by mast cells, immunoglobulin E-mediated, cause systemic anaphylaxis. If an administered drug causes an immune-mediated hypersensitive reaction, such as an anaphylactic reaction, it can even be a threat to life. Management of risk factors and careful monitoring to avoid allergens





and triggers can greatly help prevent anaphylaxis. It can potentially be precipitated by various commonly used or prescribed therapeutic agents encountered in dental practice. Dentists must be capable of recognizing and initiating the management of anaphylaxis. This article will shed some light on anaphylaxis recognition and treatment by dentists.

Pathophysiology:

IgE – Mediated Anaphylaxis:

In IgE-mediated anaphylaxis, the first contact of the allergen with the host results in the production of specific IgE antibodies by plasma cells – a process called **sensitization**. The Fc portion of these IgE antibodies becomes fixed to high-affinity cell surface receptors on mast cells and basophils. Subsequent exposure to the allergen causes cross-linking of the IgE antibodies and aggregation of their receptors. This results in the release of **preformed mediators** such as histamine, tryptase, carboxypeptidase A, proteoglycans, chymase, and TNF-alpha and **newly synthesized mediators** such as leukotrienes, prostaglandins, TNF-a, platelet-activating factor. Collectively, these mediators are responsible for the clinical manifestations of anaphylaxis.

Anaphylactoid reactions:

Anaphylactoid reactions (non-allergic anaphylaxis) do not require previous exposure to the allergen. There are a number of mechanisms

implicated in the process, and these vary depending on the agent. Mechanisms include direct activation of mast cells and basophils to cause histamine release and activation of the kallikrein-kinin system and complement and clotting cascades.

Anaphylaxis types based on major causative agents:

1. Drug induced anaphylaxis – medications, local anaesthetic agents, vaccines.
2. Latex induced anaphylaxis
3. Food induced anaphylaxis
4. Insect sting induced anaphylaxis
5. Exercise induced anaphylaxis
6. Idiopathic anaphylaxis

Causes of anaphylaxis in dental practice:

1. Antibiotics
2. Latex
3. Chlorhexidine
4. Local Anaesthetic
5. General Anaesthetic
6. Iodoform
7. Toothpaste

Allergy to local anaesthetic agents:

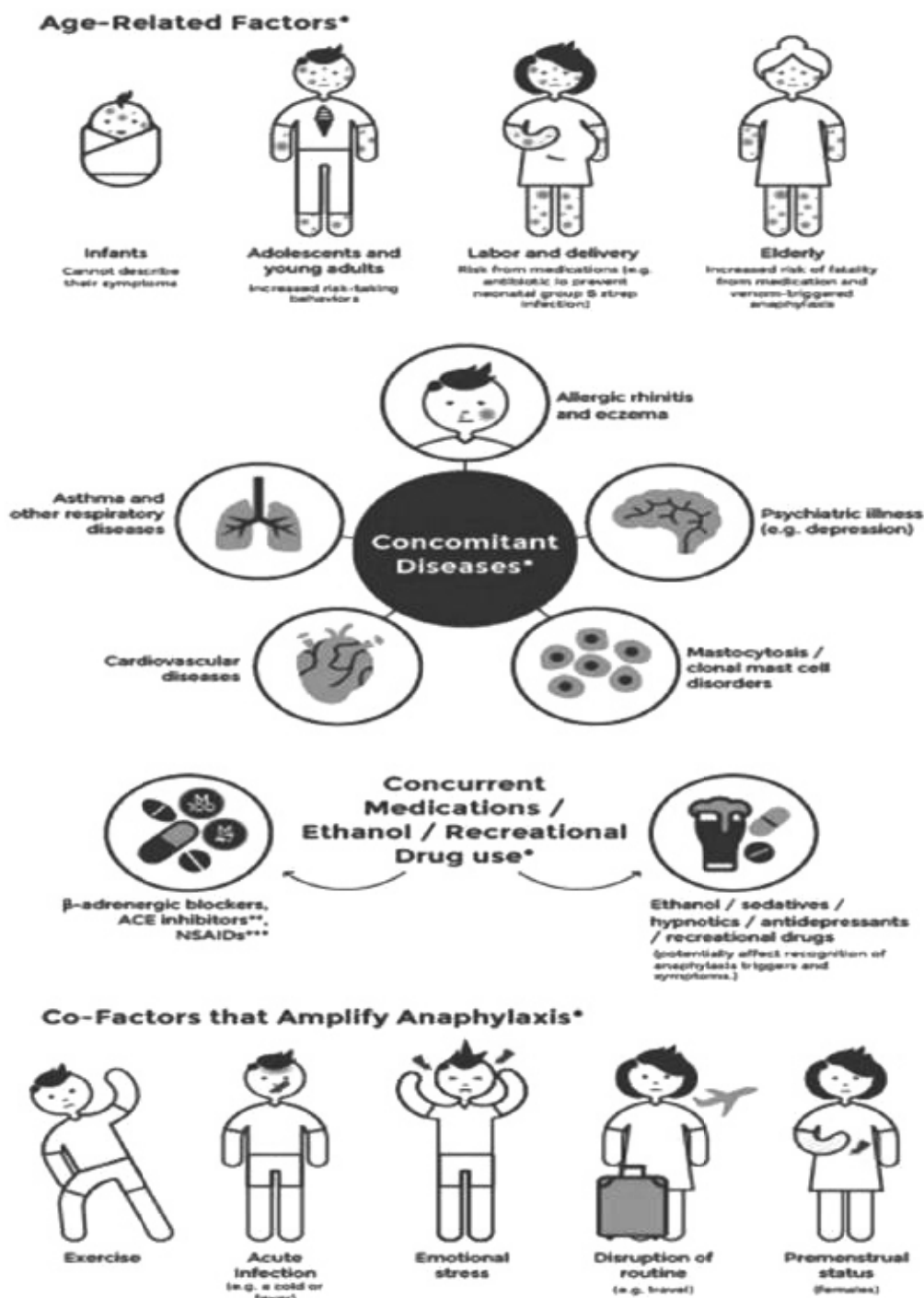
Allergic reactions to local anesthetic agents may occur due to sensitivity to:

1. Either the ester or amide component.
2. The methylparaben is used as a preservative in the multiple-dose vials.

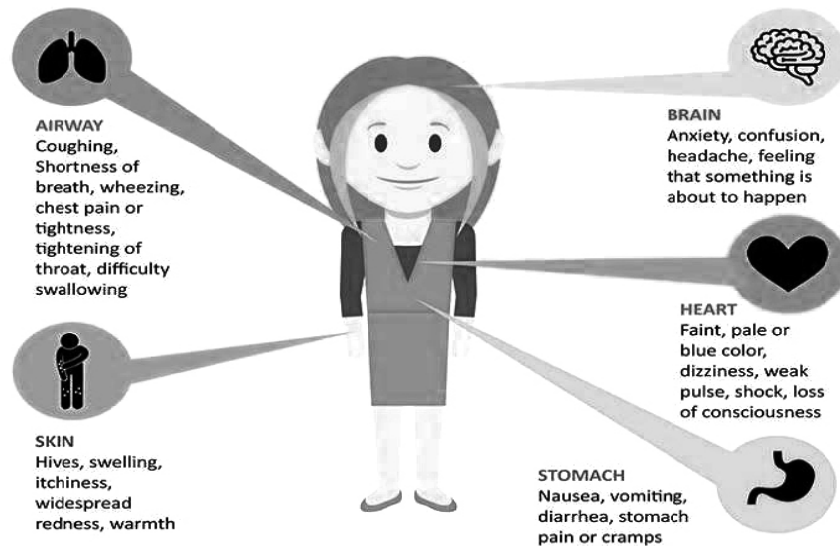
- The antioxidants are used in some formulations.

Ester local anesthetics are associated with a higher incidence of allergic reactions due to one of their metabolites, para-aminobenzoic acid (PABA). PABA is structurally similar to

methylparaben. Amide local anesthetics do not undergo metabolism to PABA, so hypersensitivity to amide local anesthetics is rare. Because of these possible hypersensitivity reactions, many manufacturers have reformulated some of their products to eliminate methylparaben. Antioxidants (sodium bisulfite, metabisulfite) are



SIGN AND SYMPTOMS OF ANAPHYLAXIS



added to local anesthetic products that contain vasoconstrictors (epinephrine, levonordefrin) to prevent biodegradation by oxygen. Some patients may also be allergic to sulfites, so the antioxidants should be avoided. A patient would rarely be allergic to an ester and amide local anesthetic.

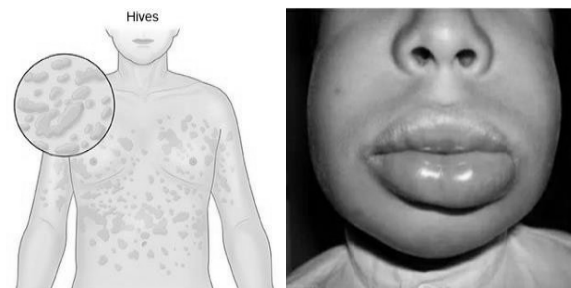
Clinical Signs And Symptoms:

Anaphylaxis symptoms arise rapidly and can progress to life-threatening airway obstruction and convulsions, which may result in death within minutes. In anaphylaxis, hypotension is when the systolic blood pressure is less than 90 mm Hg or a 30% reduction in the systolic pressure from baseline. Gastrointestinal symptoms are more prevalent in food-induced anaphylaxis.

For a diagnosis of anaphylaxis, there should be an acute onset (minutes to hours) of two or more skin-mucosal involvement, respiratory compromise; hypotension and associated symptoms; and persistent gastrointestinal symptoms.

Biphasic Reactions: In these situations, recurrence of anaphylaxis can occur up to 72 hours after the resolution of the initial reaction,

although more commonly, it occurs within eight hours. Therefore, it is important for the patient who has experienced an anaphylactic episode to have a period of observation.



Management of Anaphylaxis in Dental Practice:

Emergency Management Of Anaphylaxis In The Clinic:

1. Evaluate airway, breathing, circulation, and consciousness
2. Review cutaneous and gastrointestinal system for signs and symptoms of anaphylaxis
3. Suspected anaphylaxis
4. Cease procedure, clear airway of materials, remove the potential triggering agent from the patient

5. Position patient supinely or sitting with legs elevated if there is difficulty breathing
6. Administer IM adrenaline (0.3 mg for adults and children greater than five years, 0.15 mg for children less than five years)
7. Call an ambulance
8. Supply supplemental oxygen via facemask at a rate of at least 6-8L/min. Administer asthma medication (e.g., salbutamol) if respiratory symptoms
9. Continue to monitor vital signs. Commence basic life support if breathing ceases. Readminister IM adrenaline every 5 minutes if symptoms persist
10. Await the arrival of the ambulance and transfer to the hospital

Drugs Used in the Management of Anaphylaxis:

Vasoactive Substances

Adrenaline: The most important drug in the acute therapy of anaphylaxis is adrenaline (epinephrine). Through the activation of α - and β -adrenergic receptors, adrenaline functionally antagonizes all of the important path mechanisms of anaphylaxis by vasoconstriction, reduction of vascular permeability, bronchodilation, edema reduction, and positive inotropy in the heart.

Adrenaline should be administered intramuscularly, preferably into the anterolateral aspect of the middle third of the thigh (vastuslateralis muscle). Adrenaline should be administered for anaphylaxis by intravenous (IV) route only if the patient becomes profoundly hypotensive or develops a cardiopulmonary arrest or those who fail to respond to multiple doses of IM adrenaline.

In a patient not in need of resuscitation, immediate intramuscular application of a dose of 0.3 to 0.5 mg adrenaline (body weight range 30 to 50 kg) to the outer upper thigh is the drug therapy of the first choice. Compared with the intravenous application, the risk of severe cardiac side effects is considerably lower. In case of no response, the

injection can be repeated every 5–10 minutes, depending on side effects. The adrenaline auto-injector could also be considered, available online in two different color coding.

0.15 mg (green-labeled device) – 10-20 kg

0.3 mg (yellow-labeled device) >20 kg

Adrenaline auto-injector has an added advantage of an increased shelf life than conventional adrenaline ampoule but is expensive. Allergy to adrenaline is rare and is usually due to hypersensitivity to sodium metabisulphite, a preservative found in adrenaline 1:1,000 solution and all adrenaline self-injector devices (Epipen). Adrenaline should still be administered to a patient in anaphylaxis who is hypersensitive to sodium metabisulphite. The need for the life-saving drug far outweighs any theoretical risk from sulphites.

Other Vasoactive Substances

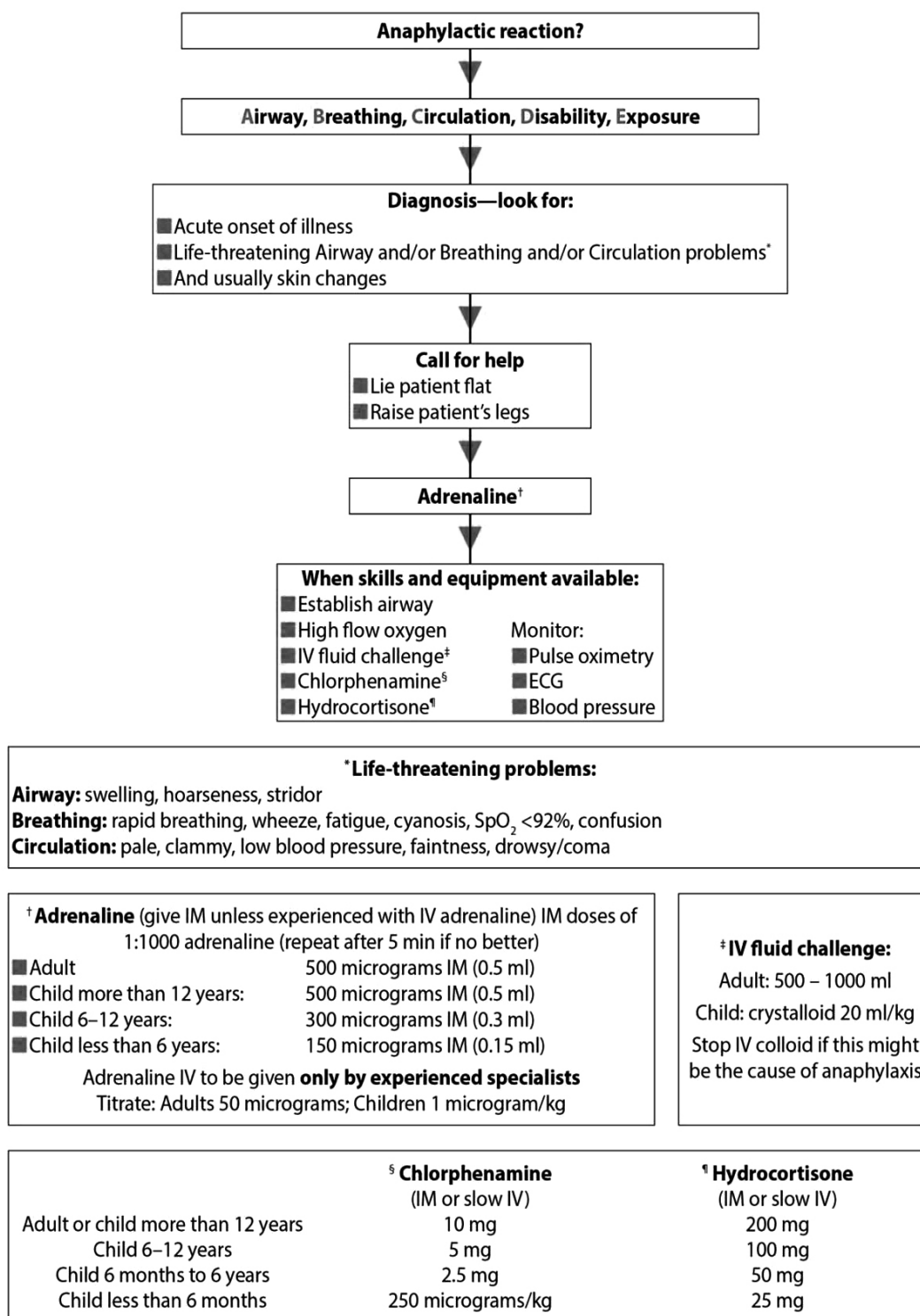
Dopamine, noradrenaline, vasopressin

Oxygen

In case of cardiovascular or pulmonary reactions, an immediate supply of oxygen via an oxygen mask with a reservoir bag is recommended. Administration of high flow 100 % oxygen is recommended. A laryngeal mask or a laryngeal tube can be helpful. In rare cases, endotracheal intubation by an experienced physician (usually an emergency physician or anesthesiologist) becomes necessary.

Volume Substitution

An important pathophysiological aspect of anaphylaxis is the resulting hypovolemia treated with adequate volume substitution. In case of anaphylactic shock, a supply of 0.5–1 liter, and possibly up to 2–3 liters of fluid — depending on the response — in a very short time is required for adults; for children, initially 20 ml/kg body weight. Primarily, normal saline (NaCl 0.9 %) or balanced electrolyte solutions should be used. When large quantities of electrolyte solutions are given, they remain in the intravascular space for a short time only.



Antihistamines H1 Receptor Antagonists

The central role of histamine as a mediator of allergic reactions and the efficacy of H1 antagonists in acute urticaria or

rhinoconjunctivitis is evident. Their effects on circulatory parameters and bronchoconstriction, however, are poorly documented. Compared to adrenaline, antihistamines show a slower onset of action; however, they show a favorable

benefit/side effect profile in a broad range of indications. An effect upon the allergic reaction can be assumed, and therefore, antihistamines should be given early in all anaphylactic responses to block the effects of histamine.

Corticosteroids

Due to their slow onset of action, corticosteroids play a minor role in the acute phase of anaphylaxis treatment. Hydrocortisone (sodium succinate) should be administered after severe anaphylactic attacks to help avert the late sequelae. Hydrocortisone may be administered by intravenous injection by intramuscular injection. The preferred method for initial emergency use is intravenous injection. Current resuscitation council (UK) guidelines for treating anaphylaxis (anaphylaxis UK guidelines) recommend giving hydrocortisone 200 mg by intramuscular injection or slow intravenous injection after initial resuscitation for treatment of adults or children more than ten years of age.

Bronchodilators:

For persistent wheeze salbutamol, 8-12 puffs of 100 micrograms (spacer) or 5 mg (nebulizer) can be given. Bronchodilators must not be used as a first-line medication for anaphylaxis as they do not prevent or relieve upper airway obstruction, hypotension, or shock. In patients taking non-cardio selective beta-blockers, adrenaline may not work. Such patients would require IV salbutamol in a hospital setting. If there is concurrent bronchospasm, salbutamol should be administered.

Conclusion:

The occurrence of anaphylaxis during dental procedures is rare; however, it could lead to adverse consequences when it occurs. There are a number of dental-related causes, including mouthwashes, local anesthetics, latex, and antibiotics for anaphylaxis. Awareness of dentists in basic life support and management of dental emergencies is crucial. The dental team must

Discharge Management:



respond effectively and manage the life-threatening situation appropriately. The timely administration of adrenaline is life-saving; any delays can lead to a poor outcome.

References:

1. Adrenaline in the treatment of anaphylaxis: what is the evidence? Andrew P C McLean-Tooke, Claire A Bethune, Ann C Fay, Gavin P Spickett
2. Knowledge and attitude toward anaphylaxis during local anesthesia among dental practitioners in Chennai – a cross-sectional study Madhuram Krishnamurthy Naveen

Kumar Venugopal Ashok
LeburuSelvendranKasiswamyElangovan Praveen
Nehrudhas

3. Dentists' knowledge about anaphylaxis caused by local anaesthetics F. C, etinkayaa*, G. Sezginb, O. MertAslan
4. Management of anaphylaxis in the dental practice: an update Phil Jevon*1 and Shaam Shamsi2
5. Systemic Anaphylaxis Following Local Lidocaine Administration During a Dental Procedure Chih-Yung Chiu, MD, Tzou-Yien Lin, MD, Shao-Hsuan Hsia, MD, Shen-Hao Lai, MD, and Kin-Sun Wong, MD