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Review Article

Perioperative Anaphylaxis: Mini Review

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Abstract

Perioperative Anaphylaxis (PA) occurs during the operation. It is a life-threatening systemic allergic reaction. Although rare, it is most severe and has a high mortality and morbidity rate. In the development of PA, the leading causes are antibiotics and neuromuscular blocking agents (NMBAs). Diagnosis is difficult due to the physiological changes of many drugs administered during anesthesia. Immunoglobulin E (IgE) or non-IgE mechanisms are responsible for the primary mechanism of PA. The most common symptoms of PA are hypotension, hypoxemia, high airway pressures, and urticaria. PA usually discontinues surgery, prolonged hospital stays, and unexpected intensive care unit (ICU) hospitalizations, which may increase morbidity, mortality, and hospital costs. The most common causes of PA include NMBAs, beta-lactam antibiotics, latex, and chlorhexidine. In the perioperative period, the first step in the treatment is eliminating the causative agent, epinephrine, and adequate fluid resuscitation. In the postoperative period, serial serum tryptase measurements, skin tests, in vitro tests, and several tests must be performed to identify the responsible agent. Recognition, management, and prevention of PA are significant for all anesthesiologists, and knowledge of this subject is necessary. This review aims to emphasize PA's importance in our anesthesia practice.

INTRODUCTION

Perioperative Anaphylaxis (PA) is a severe, life-threatening, and fatal systemic hypersensitivity reaction. PA has a broad clinical spectrum and is a generally fatal complication, although its frequency is low [1]. The ability, knowledge, and management to recognize and treat this clinical picture in the perioperative period are crucial for every anesthetist [1,2]. The incidence of PA is 1:10000–1:11000, and the mortality rate is 1.4–4.75% [3,4]. In the development of PA, The leading causes are antibiotics and NMBAs. Diagnosis is challenging due to the physiological changes of many drugs administered during anesthesia. The most common symptoms of PA are hypotension, hypoxemia, high airway pressures, and urticaria. Immunoglobulin E (IgE) or non-IgE mechanisms are responsible for the primary mechanism of PA [5,6]. PA usually discontinues surgery, prolonged hospital stays, and unexpected ICU hospitalizations, which may increase morbidity, mortality, and hospital costs. The most common causes of PA include NMBAs (mainly succinylcholine and rocuronium), beta-lactam antibiotics (mainly penicillins, cephalosporins, and glycopeptides), chlorhexidine, and latex. When PA develops due to many substances and drugs that patients are exposed to in the perioperative period, it may be challenging to detect triggering agents (Table 1) [3,4]. Immunological tests are essential in diagnosing, but the findings should be carefully evaluated because of high false positive and negative rates. Epinephrine is the most effective first-line treatment for PA. In the perioperative period, the first step in the treatment is eliminating the causative agent, epinephrine, and adequate fluid resuscitation [5,6]. This review aims to emphasize PA's importance in our anesthesia practice.

ETIOLOGY

Anesthesiologists routinely provide patients with procedural requirements in many clinical settings, such as the operating room (Under general and regional anesthesia), ICU, emergency room, and non-operating room environment they intervene. In these settings, many drugs, blood products, and contrast agents for imaging are given to patients, causing adverse reactions, including immediate hypersensitivity reactions, even PA may develop due to these. The causes of PA are quite broad in the spectrum. While specifying the etiological factors, reference can be made to the pathophysiological mechanisms. According to this, the primary mechanism of PA is mediated by immunoglobulin E (IgE) or non-IgE mechanisms [5,6].

1-Immunoglobulin E (IgE) dependent immunological mechanisms:

IgE-mediated anaphylaxis accounts for approximately 60% of PA and has a more severe course [5]. IgE-mediated anaphylaxis possible agents include latex, hapten-forming antibiotics (β-lactams, sulfonamides, streptomycin, vancomycin), NMBAs,

protamine, chlorhexidine and hypnotic agents (barbiturates, propofol, etomidate), allergen extracts (pollen, dust, mold), foods, vaccines, poisons, heterologous serums, heparin, hormones (insulin, progesterone, calcitonin), disinfectants and local anesthetics (Table 1)[5-7].

2- Immunoglobulin E (IgE) non-dependent immunological mechanisms:

Non-IgE-bound direct mast cell and basophil non-immunological mechanisms with degranulation. Considering the causes of non-IgE-mediated anaphylaxis, NMBAs, hypnotics, non-steroidal anti-inflammatory agents, blood transfusion in those with IgA deficiency, radiographic contrast agents, protamine, dextran, local anesthetics, ketamine, opioid narcotics, amphotericin-B, aspirin, indomethacin, antineoplastic agents, and exercise, it is seen that many causes such as idiopathic recurrent PA, are listed (Table 1) [5-7].

Pathophysiology of Perioperative Anaphylaxis

Anaphylaxis, as stated in the etiological causes. It occurs by two main mechanisms:

1-IgE-mediated anaphylaxis

2-Non-IgE mediated anaphylaxis

Both mechanisms result in tissue mast cells, and it causes the release of chemical mediators that initiate anaphylactic reactions from basophils. The IgE-mediated mechanism, the first encounter with antigen/allergen macrophage-initiated process IgE by the range of B-cells while providing the formation of IgE, basophil, and mast they start sensitization by binding on their cells, binds to the antigen on the second encounter with the antigen from these previously sensitized cells by binding of two IgE molecules to basophil and mast cells [8-14]. Releasing chemical mediators, mainly histamine, leukotrienes (LTC4, LTD4, LTE4), inflammatory cells release many mediators, bradykinin, nitric oxide, and platelet-activating factor (PAF) [8]. The clinical consequences of anaphylactic reactions occur with the release of these mediators. The acute shock that develops during anaphylaxis is classically known as disruptive shock. This table originated from the effects of vasoactive mediators on the cardiopulmonary system associated with profound hypovolemia. Vasoplegia and shock cause many physiological changes with the effect of inflammatory mediators released [8,10]. These results:

- Increase in vascular permeability
- Increased secretion from bronchial glands
- Increased smooth muscle contraction in bronchioles, gastro-intestinal tract, uterus, and blood vessels
- Increased migration of eosinophils, neutrophils
- Increased platelet aggregation and degranulation
- It can be listed as an increase in kallikrein/bradykinin release (Figure 1).

Table 1: Agents that Trigger Perioperative Anaphylaxis

Frequently	Less commonly
• Beta-lactam antibiotics • NMBAs: Mainly succinylcholine and rocuronium • Chlorhexidine • Dyes (especially patent blue) • Sugammadex	• Latex • A-Galactosidase; gelatins • Allogeneic blood components • Hypnotics • Opioids • Radiocontrast agents • Local anesthetics

NMBAs: Neuromuscular blocking agents

In non-IgE mediated Anaphylaxis, IgG/IgA, and antigen, the complex it forms create complement system activation (C3a, C5a), and anaphylatoxic complement mediators activate basophil and mast cells. A second way is complement non-immunologically, the allergen can directly stimulate basophil and mast cells [8-10].

Clinical Findings

Detecting and diagnosing the clinical signs of PA as patients are under general anesthesia is quite challenging. PA's typical clinical manifestations include cardiovascular, respiratory, and cutaneous findings (Table 2). The most common findings are hypotension and bronchospasm [7,8,11]. PA is challenging to recognize and diagnose when under general anesthesia and in the ongoing surgical process. Many clinical pictures in the differential diagnosis can mimic PA in this period. Physiological disturbances associated with cardiovascular and respiratory effects and anesthesia are similar to the PA reaction. They may obscure the clinical presentation of PA, which may complicate the initial diagnosis of PA in the perioperative period [14]. 25% of PA is caused by air embolus, CO_2 embolism, pulmonary edema, tension pneumothorax, pulmonary embolism, arrhythmia, sympathectomy due to high neuraxial block, hemorrhage, and primary cardiac event (Cardiac tamponade, cardiogenic shock, and cardiac arrest). The differential diagnosis was delayed due to extensive conditions (Table 3) [3].

The pharmacological effects of drugs given routinely during general anesthesia may cause hypotension, tachycardia, bradycardia, and various rhythm disturbances that may delay the diagnosis of PA. Identifying urticaria and other cutaneous findings that develop in a fully occluded patient during general anesthesia is difficult. However, symptoms such as nausea, itching, and rash are impossible to detect when the patient is unconscious. Therefore, according to international guidelines, a group of anesthesiologists, allergists, and immunologists has suggested a scoring system to classify an immediate hypersensitivity reaction in the perioperative period. This scoring calculates a numerical score based on the timing of the suspected reaction to cardiovascular, respiratory, and dermal/mucosal features and possible intravenous (IV) triggers, with the option to include tryptase measures in this scoring. Perioperative hypersensitivity reactions are then classified as:

- Unlikely
- Possible

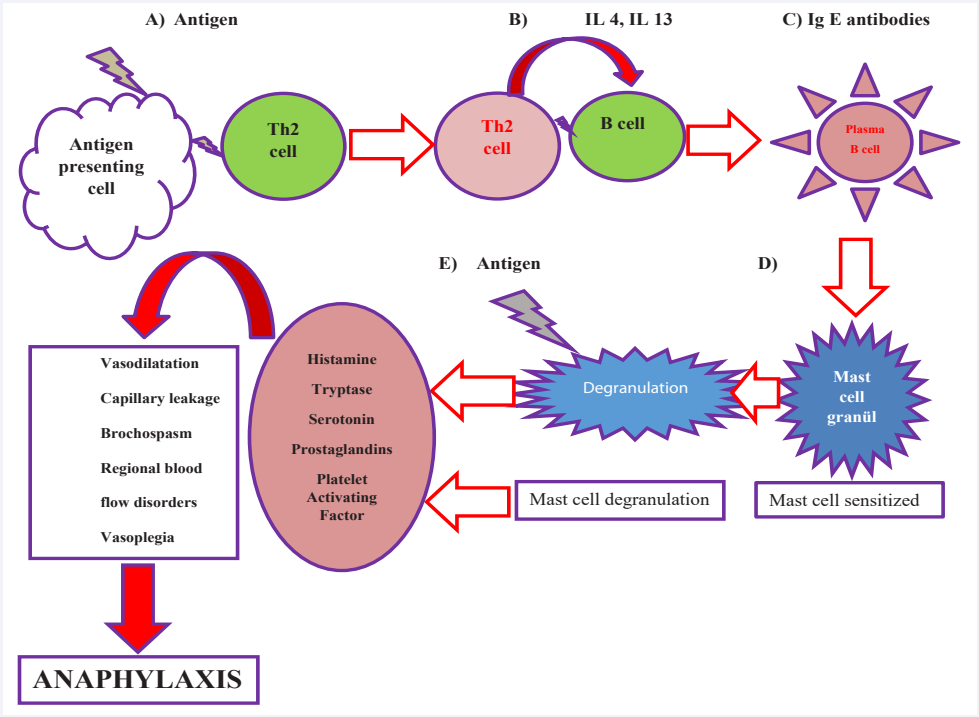

Figure 1 Pathophslogi of Perioperative Anaphylaxis.

Table 2: Symptoms and Signs of Perioperative Anaphylaxis

System	Symptoms	Signs
• Cardiovascular	• Diaphoresis • Dizziness • Palpitations	• Cardiac arrest • Hypotension or cardiovascular collapse • End-tidal carbon dioxide ↓ • Tachycardia/Bradycardia • Dysrhythmias
• Respiratory	• Acute hoarseness • Chest discomfort • Short of breath • Wheezing	• Acute respiratory failure • Bronchospasm/Raised inspiratory pressures during ventilation • Reduced pulmonary compliance • Laryngeal edema • Stridor
• Mucosa/skin	• Burning • Tingling • Itching	• Flushing • Diffuse erythema • Cutaneous/Mucosal edema • Urticaria
• Neurologic	• Malaise • Sense of impending doom	• Loss of consciousness • Confusion
• Gastrointestinal	• Abdominal cramps • Nausea	• Diarrhea • Vomiting

Table 3: Differential Diagnosis in Perioperative Anaphylaxis

Other causes of the airway and respiratory signs	Other causes of hypotension or vasoplegia
• Acute bronchospasm/asthmatic crisis • Aspiration • Endotracheal tube malposition • Air embolus • Posextubation stridor • Pulmonary edema • Tension pneumothorax • Transfusion-related acute lung injury (TRALI)	• Arrhythmia • Hemorrhage • Vasovagal reaction • Venous air embolism • Cardiac tamponade • Cardiogenic shock • Overdose of vasoactive drugs • Sympathectomy due to spinal/Epidural anesthesia • Pulmonary embolus • Sepsis

*There are many reactions and physiological events in the differential diagnosis of perioperative anaphylaxis during or after general anesthesia. Tryptase levels should be normal in all disorders noted to exclude perioperative anaphylaxis.

- Likely
- Very likely
- Almost certainly, an immediate [14].

Triggers Factors

Antibiotics: In the United States, antibiotics are responsible for roughly half of the identified events of PA as the cause [2,3,5,7]. Beta-lactams account for 90% of PA due to antibiotics, and among these, the most common were cefazolin and co-amoxiclav [2,3,5].

Neuromuscular blocking agents: Between 60 and 70% of PA cases are NMBAs. Rates of PA associated with NMBAs are lower in the USA. It constitutes 20-30% of the identified triggers of NMBAs succinylcholine and atracurium are also commonly implicated in NMBAs [2,3,5]. The most significant risk in PA with all NMBAs is short-acting succinylcholine, a depolarizing agent. NMBAs are an IgE-mediated immunological pathway and non-immunological direct mast cell activation that may cause PA.

Interestingly, atracurium is a particularly potent inducer of mast cell activation and histamine release, although many NMBAs have been shown to induce mast cell activation via non-IgE mechanisms [15]. Suxamethonium, atracurium, and rocuronium are the most commonly accused NMBAs. The patient's IgE-mediated previous exposure to NMBA for an allergic reaction staying is not a prerequisite. Quaternary ammonium ions to certain other drugs and environmental chemicals that have (e.g., cosmetic) exposure appears to be cross-sensitive to NMBAs. Histamine release mivacurium, which could be more pronounced with some NMBAs such as atracurium. The high reaction rate is because NMBAs are non-false positive, causing specific mast cell release skin test results [2,3,5,7].

Latex: In most studies, PA is caused by natural rubber latex second, after NMBAs. It has been reported to be in the rank [16-18]. It is derived from the natural rubber latex Hevea brasilensis, broadly used in many medical devices and drugs [19]. The most common causes of latex exposure in the perioperative setting are skin or gloves with prolonged contact with mucosal surfaces (sterile and examination), drains, and catheters. Latex reactions with surgical intervention 30 minutes from the start. or it tends to occur later. Wearing latex gloves on visceral surfaces symptoms with manipulation by clinicians emerge with latex anaphylaxis from forming specific IgE against latex proteins. It is an IgE-mediated reaction [18,19].

Sugammadex: Sugammadex, a cyclodextrin molecule, acts through the neuromuscular binding of ammonium and is mainly effective in antagonizing NMBAs such as rocuronium and vecuronium. The literature indicates that sugammadex is one of the three leading triggers of PA [2,17].

Hypnotics: Hypnotic induction agents in PA constitute approximately 2% of cases. Barbiturates are responsible for most of the reactions. Direct mast cell activation has also been reported. However, most reactions from induction agents are IgE-mediate [9]. Non-barbiturate anaphylaxis against agents is very rare. Propofol may cause IgE-mediated reactions, but the most common are non-immunological reactions. Propofol can directly stimulate the release of histamine, and this effect may be more substantial when applied together with NMBAs. Propofol soybean oil, egg lecithin, and formulated in a lipid vehicle containing glycerol. The product information lists allergy to eggs or soybeans as a contraindication. Although studies have shown that these individuals, the vast majority tolerated propofol. Benzodiazepines non-barbiturate induction are agents. Hypotension following IV administration: These agents have known side effects, but anaphylactic reactions are rare. Diazepam causes more reactions than midazolam. In addition, midazolam is administered to patients with drug allergies. It is used safely for the induction of anesthesia. Opioids in anesthesia/analgesia cause frequent flushing following IV administration and urticaria. However, angioedema and bronchospasm rarely cause life-threatening reactions, such as causes opioids rather than an IgE-mediated reaction causing non-specific mast cell activation. Morphine and codeine by direct mast cell activation with opioids, as this may cause false positive results. Specific dilutions in skin tests should be [2-5,7,9,17].

Chlorhexidine: Chlorhexidine has been related to rates of PA and leading third as the cause of PA [3]. While some patients progress with mild skin reactions, fatal anaphylactic reactions may occur in some patients [21].

Risk Factors

Risk factors for PA include previous unexplained life-threatening during anesthesia, any history of an allergic reaction, female gender (specific for drugs), mast cell disorders, other allergic conditions (such as asthma, eczema or hay fever, multiple past surgeries (especially latex and for ethylene oxide), drug allergy and atopy [1-3,10,11].

Management of Perioperative Anaphylaxis

PA is a fatal clinical condition that requires immediate aggressive, effective, and accurate intervention in a multidisciplinary manner when its diagnosis is suspected. The first thing to do is discontinue the suspected triggering agent and emergency resuscitation measures, including administration of 100% oxygen, establishing a decisive airway, epinephrine, and adequate fluid resuscitation (It should be initiated according to the Advanced Cardiovascular Life Support guidelines (ACLS) [22-24]. To correctly manage the emergency when PA develops under general anesthesia, calling for early help is a known step in treating hemodynamically unstable patients. It is crucial to apply resuscitation according to the guideline. First, IV administration of epinephrine, followed by fluid resuscitation and appropriate administration of other treatments, is essential. The rapid management of PA is summarised in Figure 2 [23,24].

Epinephrine: Epinephrine should be administered promptly, based on the severity of the reaction, and the dose should be titrated and repeated as necessary, depending on the response to

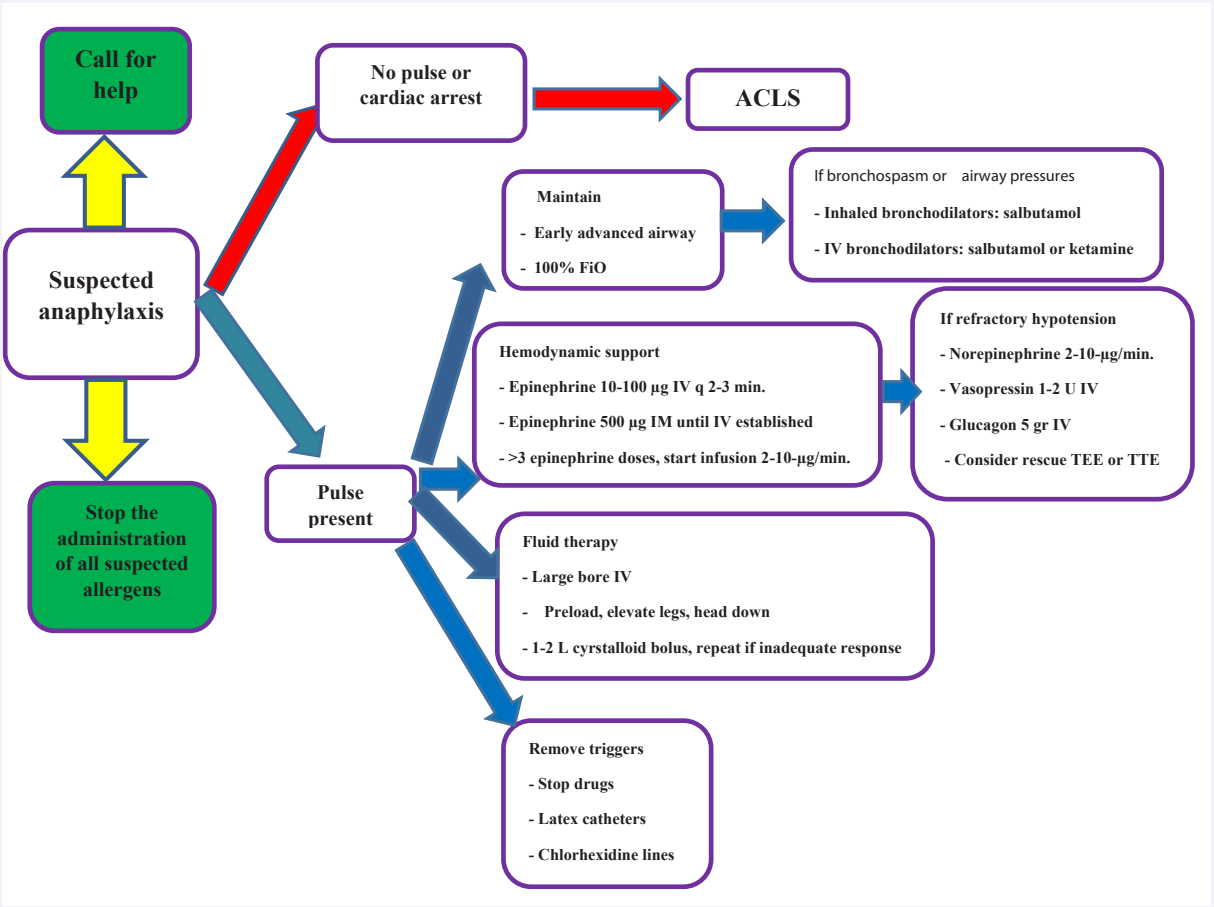

Figure 2 Rapidly management of Perioperative Anaphylaxis
ACLS: Advanced cardiopulmonary life support, TEE: Transesophageal echocardiography, TTE: Transthoracic echocardiography, IV: Intravenous

treatment. The severity of PA is graded from I to IV according to the Ring and Messmer classification as specified in the guidelines: Classification accordingly

1- Mild reactions: Grade I

2- Moderate: Grade II

3- Life-threatening: Grade III

4- Cardiac and/or respiratory arrest: Grade IV (Table 4)[23].

ACLS guidelines have recommended initial epinephrine doses as follows:

Grade II: 10-30 µg IV bolus for reactions

Grade III: 50-200 µg IV boluses for reactions

Grade IV: Recommend an initial epinephrine dose of 1 mg IV with repeated 1 mg [22-24].

If **accelerating** doses of epinephrine are necessary, defined as three repeated doses:

a) IV infusion starting at 0.05-0.1 µg/kg/min.

b) Epinephrine can be administered intramuscularly if IV access is unavailable

c) The suggested dosing ranges from 0.3-0.5 mg to 0.5-0.8 mg [22-24].

Volume Treatment: Adequate IV fluid therapy is a cornerstone of PA management due to leaky capillaries, vasodilation, and pooling of splanchnic circulation, which results in significant fluid shifts and hypovolemia [22,23].

According to guidelines have recommended base initial crystalloid bolus volumes by reaction severity as follows:

Grade II: 0.5 L

Grade III: 1 L

Grade IV: as per advanced life support standards considering [22].

Generally, guidelines recommend 20-30 mL/kg of crystalloid

Table 4: Grading Perioperative Anaphylaxis (arranged from the Ring and Messmer grading scale)

Grade	Basic	Specific symptoms
Grade 1	• Generalized mucocutaneous signs	• Erythema • Urticaria • Angioedema
Grade 2	• Multi-organ manifestations	• Mucocutaneous signs • Bronchospasm • Hypotension
Grade 3	• Severe life-threatening multi-organ manifestation	• Tachycardia/Bradycardia • Arrhythmias • Cardiovascular collapse • Bronchospasm • May have cutaneous signs
Grade 4	• Cardiopulmonary arrest	• Cardiac or respiratory arrest

fluid, and fluid therapy is tailored to clinical response and underlying pathophysiology [22,23].

To more accurately and precisely assess fluid response, to determine the ventricular function and left ventricular end-diastolic volumes and inferior vena cava diameter, and to evaluate any confounding cause of hemodynamic instability, transthoracic or transesophageal echocardiography should be considered [22].

Other treatment modalities: Epinephrine-resistant hypotension may require inotropes such as norepinephrine and vasopressin [22-24]. In patients using beta-blockers, glucagon can be used in cases unresponsive to epinephrine [23,24]. Salbutamol can be administered as an inhaled or IV infusion in the case of bronchospasm without hypotension or unresponsive to epinephrine [22-24]. Treatment of PA in pregnant patients has additional measures such as left uterine displacement and consideration of cesarean section if indicated [23].

Intravenous antihistamines such as dexchlorpheniramine are applicable for the symptomatic treatment of pruritus, urticaria, angioedema, and mild grade I reactions or to prevent the recurrence of symptoms after the initial reaction [22,23]. The literature has reported no improvement in the results after using antihistamines or no adverse effect on antihistamines (2,3,22). Although the evidence is limited, corticosteroids help prevent or shorten prolonged reactions it could be [22-24].

In conclusion, administering antihistamines and corticosteroids is not a priority and should only be given after the patient has undergone essential therapy and has been adequately resuscitated. The decision to continue or cancel the surgery should be based on the patient's clinical condition after the patient has been stabilized. Patients who develop PA should be followed closely in the ICU for at least the first 24 hours due to possible complications and the risk of PA recurrence [23,24].

Postoperative Period

In general, in suspected PA reactions, an anesthesiologist must be consulted and should contact the allergist [24]. In addition to notifying pharmacovigilance centers for all grade II-IV reactions, they should be evaluated with the anesthetic allergy testing center (if any) for further investigation of the reaction [22-24]. Anaphylaxis interrogation, typically anesthesia charts, tryptase measurements, skin testing, in vitro testing, and drug provocation, consists of an examination that includes testing [22]. After determining the causative and triggering agent(s), preventive and protective strategies are considered and in line with the recommendations and guidelines. Future surgery should be performed with appropriate preparations [24].

Serum tryptase test: Serum tryptase is the test that should be sampled acutely as it is a marker of mast cell degranulation when PA develops. Normal tryptase values are below 11.4 μg/L. Tryptase values greater than 9.8 μg/L are associated with positive allergy, reasonable sensitivity, and specificity. A peak tryptase value greater than 33 μg/L is significant in detecting an agent responsible for the reaction. Serum tryptase levels above baseline should suggest PA, mainly due to mast cell degranulation. However, normal levels of tryptase do not exclude the possibility of PA or false negatives [22-25]. Although elevated serum tryptase in PA shows that it is more IgE-mediated [26], it should be noted that it can also be elevated in both IgE and non-IgE-mediated reactions [22,25,27].

Serum tryptase values peak approximately 1 hour after a PA pattern develops [25]. According to ANZAAG guidelines, blood samples should be taken immediately after the first treatment, at 1 hour, 4 hours, and >24 hours. [23]. In our clinic, we routinely collect samples of a baseline tryptase 30 minutes to 2 hours after the PA and 24 hours after the PA event.

Skin testing: IgE-mediated allergic reactions and almost all skin testing are recommended for diagnosing PA reactions and these tests [22-25]. It should be done 4-6 weeks after the PA reaction in a specialized center [25-27]. Skin tests are helpful for NMBAs, antibiotics, and latex [23,27]. Although the specific IgE antibodies can be measured for latex, succinylcholine, chlorhexidine, and some selected antibiotics, the sensitivity and specificity of these assays may vary according to the clinical picture [22–25]. When the decision to perform a provocation test is taken, a careful risk-benefit analysis should be made, as informed consent and a team and environment that can manage cardiopulmonary events that may occur [22-25].

Key Points

- Early recognition and recognition of PA are significant.
- Administering adequate and appropriate doses of epinephrine is the cornerstone of PA therapy.

- Antihistamines and steroids in the initial treatment of PA should not be used.
- PA is a clinical diagnosis, and the plasma tryptase test only supports the diagnosis.
- When a PA develops, the anesthesiologist should have adequate knowledge and play a key role in patient management.

CONCLUSION

Knowledge of PA recognition, management, and prevention is essential and challenging for all anesthesiologists. Clinical symptoms and signs of PA early recognition and prompt treatment rapid initiation can reduce mortality and morbidity. With the subject relevant, up-to-date algorithms should be followed; epinephrine administration, fluid replacement, hemodynamic and respiratory treatment consisting of close monitoring of the parameters protocol by all healthcare practitioners should be well known. Acute and primary tryptase measurements are vital for post-event diagnosis. The triggering agent must be identified, and it is crucial to make preventive and preventive recommendations for possible future perioperative exposures.

Author Contribution Statement

Cekmen N conceived and designed the review. Cekmen N and Yazar Ç wrote the manuscript. I read the manuscript and approved the final version.

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