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A Critical Approach: Timely Diagnosis and Management of Anaphylactic Shock in the Operating Room

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ABSTRACT

Introduction: *Perioperative anaphylaxis is a critical emergency that is difficult to diagnose under general anesthesia due to the inability to assess subjective symptoms and the frequent absence of cutaneous signs.*

Case Report: *A 56-year-old female patient with a history of anaphylactic shock caused by ceftriaxone three weeks prior was scheduled for breast-conserving surgery. Following anesthetic induction and 10 minutes after the administration of methylene blue, she developed severe hypotension (50/26 mmHg) and an abrupt drop in $ETCO_2$ (28 mmHg) without any cutaneous manifestations.*

Intervention or Treatment: *Given the lack of response to the administration and increased infusion rate of norepinephrine, anaphylactic shock was suspected, and titrated IV epinephrine boluses (50 mcg) were initiated, followed by a continuous infusion, in conjunction with aggressive volume replacement with crystalloids and 100% oxygen therapy, resulting in an immediate and adequate response.*

Course and Outcomes: *The patient achieved hemodynamic stabilization with epinephrine, allowing for the gradual withdrawal of vasopressors, uncomplicated extubation in the operating room, and hospital discharge on the third postoperative day.*

Conclusions: *Sudden cardiovascular collapse without cutaneous signs should raise suspicion of transanesthetic anaphylaxis. Epinephrine is the indispensable first-line medication; a recent history of anaphylaxis requires prior allergy evaluation to identify risk factors before a new procedure. Although the acute event was resolved according to standard protocol and consultation with an allergist was pending for follow-up, the absence of such an evaluation following the first event in our case is undeniable.*

Keywords

General Anesthesia, Anaphylactic Shock, Hypersensitivity, Methylene Blue, Epinephrine.

Introduction

We present the case of a cancer patient scheduled for breast-conserving surgery plus sentinel lymph node biopsy who experienced a drop in cardiac output during anesthesia following the administration of anesthetic drugs and methylene blue. As a result, crystalloids, oxygen therapy, and vasoactive drug support were administered, and anaphylaxis was suspected based on the patient's clinical features and medical history.

Anaphylaxis is one of the most serious emergencies that can occur

during the perioperative period and can have a fatal outcome. Its pathophysiological severity and the difficulty in identifying it in a patient under general anesthesia—who cannot report symptoms, has a limited visual field that hinders clinical assessment, is receiving multiple medications, and may exhibit hemodynamic and ventilatory changes typical of surgery and anesthesia—present a significant anesthesiological challenge. The sixth National Audit Project revealed an incidence of one case per 10,000 anesthetics administered across 266 episodes of severe perioperative anaphylaxis, in which the most significant agents were antibiotics, neuromuscular blockers, chlorhexidine, and dyes—notably Patent Blue. Furthermore, hypotension was identified clinically as one of the most prominent manifestations of the event, followed by desaturation, bronchospasm, cardiac rhythm disturbances, and a reduction or disappearance of the capnographic waveform. Forty cardiac arrests and 10 deaths were recorded [1-5].

Clinical Case Presentation

We present the clinical case of a 56-year-old female patient with a mass in her left breast measuring approximately 6×7×5 mm, classified as BIRADS 6, with a histopathological diagnosis of grade 2 ductal carcinoma in situ. She is scheduled for harpoon-guided breast-conserving surgery and sentinel lymph node biopsy. During the preanesthetic evaluation, vital signs were as follows: blood pressure 124/66 mmHg, heart rate 69 bpm, respiratory rate 18 bpm, and SpO_2 95%; weight 60.6 kg, height 1.60 m, and BMI 23.43 kg/m². Relevant medical history: multiple trauma 26 years ago requiring 3 weeks of intensive care and intubation; critical condition with cardiorespiratory arrest; multiple blood transfusions; surgery for a right femur fracture, rib fractures, and clavicle fracture; four cesarean sections; trigger finger surgery with no reported complications; hypothyroidism for the past 4 years, currently treated with levothyroxine; anaphylactic shock associated with intravenous administration of ceftriaxone during the preoperative period 3 weeks prior to the current scheduled procedure, which required administration of epinephrine, hydrocortisone, and norepinephrine; did not require advanced airway management; denies substance abuse. Currently, there are no indications of a difficult airway; she is clinically stable. Preoperative workup: laboratory values within normal ranges; electrocardiogram shows sinus rhythm without abnormalities. Based on this, her anesthetic-surgical risk was classified as ASA III, ARISCAT score 43 points, and a general anesthesia plan was established.

The patient was admitted to the operating room with a 20-gauge peripheral venous access in the right upper limb; a second 14-gauge peripheral venous access was established in the same limb. Noninvasive monitoring showed blood pressure of 146/67 mmHg, HR 73 bpm, and RR 16 bpm; SpO_2 89% on room air; preoxygenation with a face mask at 6 L/min and FiO_2 80%. Intravenous induction was initiated with fentanyl 180 mcg, lidocaine 60 mg, propofol 100 mg, and rocuronium 40 mg. following pharmacological hysteresis, direct laryngoscopy was performed (Cormack-Lehane grade II), and atraumatic orotracheal intubation was achieved on the first attempt with a 7.0 mm ID Murphy tube; tube placement was confirmed via continuous capnography, bilateral chest expansion, and symmetrical pulmonary auscultation; the tube was secured 19 cm from the dental arch with 7 mL of air; the patient was placed on volume-controlled mechanical ventilation with a tidal volume of 350 mL, respiratory rate of 14 breaths per minute, I:E ratio of 1:2, PEEP of 5 cmH_2O, and peak pressure of 35 cmH_2O; Ppeak 13 cmH_2O; sevoflurane CAM 0.8 with FiO_2 30%; vital signs: BP 116/53 (80) mmHg, heart rate 60 bpm, SpO_2 95%, $ETCO_2$ 33 cmH_2O, temperature 36.4°C. Due to the patient's history of airway hyperresponsiveness, it was decided to administer 8 mg of dexamethasone and 200 mg of hydrocortisone intravenously. During the transanesthetic period, a sudden desaturation to 89% and an $ETCO_2$ of 28 cmH_2O were observed, so the FiO_2 was increased to 100%. while blood pressure (BP) was measured, revealing arterial hypotension at 89/36 (61) mmHg; a loading dose of Hartman's solution at 10 ml/kg was immediately administered while an infusion of norepinephrine at 0.1 mcg/kg/min was initiated, with a poor response (BP 69/35 (45)), HR 71 bpm, SpO_2 91%, Ppeak 19 cmH_2O. The patient was partially uncovered with no evidence of skin reactions; norepinephrine was increased to 0.12 mcg/kg/min, but there was no response, with BP at 50/26 (34) mmHg. The event occurred approximately 10 minutes after the administration of methylene blue; a severe hypersensitivity reaction/anaphylaxis was suspected, so a 50 mcg intravenous bolus of epinephrine was administered, resulting in an immediate response: BP 85/47 (60) mmHg, heart rate 59 bpm, SpO_2 92%, $ETCO_2$ 30 cmH_2O. The same bolus dose was repeated while initiating an infusion at 0.03 mcg/kg/min, resulting in a good response: blood pressure 120/61 (84) mmHg, HR 107 bpm, sinus rhythm, O_2 sat 93%, $ETCO_2$ 35 cmH_2O. Norepinephrine was gradually tapered until discontinued, achieving patient stability with epinephrine at 0.02 mcg/kg/min and a mean arterial pressure above 70 mmHg. Anesthetic maintenance with sevoflurane at 0.6–1.0 CAM, fentanyl and rocuronium administered in divided doses with total doses of 330 mcg and 55 mg, respectively, and a lidocaine infusion at 10 mcg/kg/min with a total dose of 106 mg, intravenous adjuvants: calcium gluconate 1 g, acetaminophen 1 g, ketorolac 30 mg, ondansetron 4 mg, and tramadol 100 mg. The surgical procedure concluded with a positive fluid balance of 1,280 mL and estimated blood loss of 40 mL. The patient was extubated following gentle suctioning of oropharyngeal secretions, with epinephrine administered at a dose of 0.01 mcg/kg/min; blood pressure was 116/63 (77) mmHg, heart rate was 62 bpm, respiratory rate 14 breaths per minute, and $SatO_2$ 95%. Six puffs of salbutamol were administered prophylactically; given adequate ventilatory mechanics and hemodynamic stability, it was decided to discontinue epinephrine. Protective airway reflexes and adequate spontaneous ventilatory mechanics were observed, so extubation was performed without complications. A reservoir mask was placed with supplemental oxygen at 10 L/min, and the patient was closely monitored in the operating room, where he remained stable; it was therefore decided to transfer him to the post-anesthesia care unit. He was admitted with BP 108/55 (72) mmHg, HR 68 bpm, SpO_2 95%, Aldrete score 9, and reported no pain. She remained hemodynamically and ventilatorily stable, met the criteria for discharge, and was transferred to the inpatient ward without further complications. She was discharged from the

hospital on the third postoperative day, reporting mild pain in the surgical area but denying any other symptoms.



Figure 1: Patient in the immediate postoperative period.

Discussion

One of the most serious emergencies that can occur during the anesthetic-surgical procedure is anaphylaxis. Both its pathophysiological severity and the challenge of identifying it in a patient under general anesthesia—who is exposed to multiple medications simultaneously and experiencing hemodynamic and ventilatory changes typical of surgery and anesthesia—cannot be assessed conventionally, making its identification a challenge, In contrast to anaphylaxis in conscious individuals, subjective symptoms—such as pruritus, a sense of impending doom, dyspnea, or gastrointestinal symptoms—cannot be assessed conventionally during general anesthesia; cutaneous signs may go unnoticed due to the patient's position, surgical drapes, and the location of the surgical team. Therefore, intraoperative diagnosis relies heavily on the detection of sudden physiological changes, particularly desaturation, hypotension, capnographic abnormalities, cardiovascular collapse, and bronchospasm, among others [1,2].

The 6th National Audit Project (NAP6) analyzed 266 episodes of severe perioperative anaphylaxis (grades 3 to 5), estimating an incidence of approximately 1 case per 10,000 anesthesiologists. Among the main agents identified were antibiotics, neuromuscular blockers, chlorhexidine, and dyes—particularly Patent Blue. Clinically, hypotension was a central manifestation of the event, followed by bronchospasm, changes in heart rate, desaturation, and a decrease and/or loss of the capnographic trace. Additionally, 40 cardiac arrests were documented, resulting in 10 deaths, thus demonstrating this condition's ability to rapidly progress from an apparently sudden hemodynamic disturbance to severe shock and ultimately cardiac arrest [2,3].

The significance of the case presented here lies in the fact that it involves multiple factors that increase the difficulty of diagnosis and treatment: a recent history of an anaphylactic reaction, new perioperative exposure to various medications, the use of a dye, and the subsequent cardiovascular collapse. Therefore, the aim is not only to establish a temporal relationship between the administration of methylene blue and hemodynamic deterioration, but also to analyze and review the pathophysiology, differential diagnoses in cases of suspected anaphylaxis, the response to vasopressor medications, and the implications for future medical procedures.

A recent history of anaphylaxis as the primary clinical risk factor

A key finding in this case is that, three weeks prior to the current procedure, the patient experienced an anaphylactic reaction to ceftriaxone. This history should have been considered a high-risk indicator requiring an allergy evaluation prior to further exposure to anesthesia and surgery. Perioperative reactions pose a diagnostic challenge that is particularly difficult, as multiple drugs may be administered during anesthesia within a short period of time, which hinders the retrospective identification of the causative agent [4,5].

The European Academy of Allergy and Clinical Immunology (EAACI) recommends that patients with suspected perioperative immediate reactions undergo specialized evaluation, which should include a detailed chronological review of all exposures, assessment of available biomarkers, and testing to detect specific allergies. The goal is not only to confirm a reaction but also to identify the causative agent and establish safe alternatives for future procedures, especially when the patient needs to undergo another surgery within a short period of time [4].

The interval of only three weeks between the episode related to ceftriaxone and the subsequent anesthesia is an aspect that warrants particular attention in this case—not because this history is considered proof that ceftriaxone caused cross-sensitization to methylene blue (since they are molecules with different chemical structures), but because the two reactions must be studied separately. The history of the first reaction is considered important, as it demonstrates that the patient had previously exhibited a severe hypersensitivity reaction and that there was an opportunity to conduct an etiological study prior to the subsequent surgical procedure.

The investigation of a perioperative reaction should not be viewed solely as a procedure conducted after the event has occurred, but rather as an intervention aimed at reducing the risk of recurrence. Developing a personalized anesthesia plan, identifying suspected agents, and properly documenting the event are essential elements for ensuring patient safety [4,5].

Premedication and Limitations of Glucocorticoids

For decades, corticosteroids were used to prevent the progression of hypersensitivity reactions and the onset of biphasic reactions. However, current evidence does not consider them a safe strategy for preventing the onset of anaphylaxis. Current guidelines identify epinephrine as the primary treatment and state that corticosteroids play a secondary role and should not delay the administration of epinephrine [1,6].

The administration of glucocorticoids should not be interpreted as evidence that adequate prophylaxis was provided, since an anaphylactic reaction is driven by a cascade of mediators that begins rapidly, whereas the genomic effects of glucocorticoids take longer to manifest. Therefore, from a pathophysiological standpoint, it is unlikely that a dose administered immediately before exposure will reliably halt mast cell activation, which is nearly immediate.

For patients with a history of perioperative anaphylaxis, the primary goal should be to avoid exposure to suspected triggers and to identify safe pharmacological alternatives, while always ensuring that rescue therapy—which cannot be replaced by premedication—is readily available [1,4,6].

Debunking Cross-Reactivities and Clinical Positions

- A detailed analysis of the current evidence requires dispelling deeply entrenched myths in everyday perioperative practice:
- **Propofol and Food Allergies:** A documented allergy to eggs or soy is not a contraindication for the administration of propofol, since the lipid fraction of the drug lacks the allergenic proteins responsible for sensitization.
- **Povidone-Iodine and Seafood:** An allergy to seafood (mediated by tropomyosin) does not imply reactivity to or contraindicate the use of iodine-based antiseptics.
- **Use of Sugammadex as a Rescue Agent:** The use of sugammadex as a therapeutic measure to reverse acute rocuronium-induced anaphylaxis is not recommended, due to the documented risk that sugammadex itself may act as an inducer of IgE-mediated anaphylaxis.
- **Systematic Screening for Hidden Triggers:** Given the lack of documentation regarding certain supplies on standard surgical records, post-event allergy evaluation should, as a matter of protocol, include testing for chlorhexidine, latex, and antiseptics in all patients under investigation [7].

Possible triggers of the reaction

In the perioperative setting, dyes used to identify anatomical structures—such as sentinel lymph nodes in this case—are less likely to cause hypersensitivity than antibiotics and neuromuscular blockers, but they can still trigger severe systemic reactions [2,9].

The NAP6 reported nine cases related to Patent Blue and emphasized that reactions to certain dyes may begin later than those associated with some antibiotics or neuromuscular blockers. This timing has important diagnostic implications, since if the reaction occurs several minutes after induction, there is a risk of mistakenly attributing it to the last drug administered, thereby overlooking agents administered prior to the onset of hemodynamic deterioration [2].

Severe hypersensitivity reactions have been associated with methylene blue; although their frequency is difficult to determine because there are only a few case reports, their existence is sufficiently well-documented to warrant their inclusion in the differential diagnosis when there is a compatible temporal relationship. Patients with cross-reactions to Patent Blue who later showed reactivity to methylene blue have been reported; this highlights the need for allergy testing that takes into account not only the agent administered during the procedure but also any other related dyes, if present. Confirmation of the cause requires integrating the timeline, biomarkers, and allergy testing [9,10].

Pathophysiological considerations

The pathophysiology of perioperative anaphylaxis is varied and can result from immunological or non-immunological mechanisms. The activation of mast cells and basophils leads to the release of leukotrienes, tryptase, prostaglandins, histamine, and other mediators that cause increased vascular permeability, vasodilation, bronchoconstriction, and cardiovascular abnormalities. A reaction that is clinically consistent with anaphylaxis does not necessarily require the demonstration of agent-specific IgE, since there are processes linked to the direct activation of mast cells (the MRGPRX2 receptor, expressed in human mast cells, has the capacity to mediate non-IgE-mediated hypersensitivity reactions to certain anesthetic drugs), complement, and other immunological or pharmacological pathways [11,12].

Clinical manifestations of anaphylaxis during anesthesia

In this particular case, we must emphasize that the "absence of skin manifestations does not rule out the disease." The diagnosis can be quickly oriented if the patient is awake and presents with pruritus, urticaria, erythema, or angioedema; however, during general anesthesia, these clinical findings may not be visible or may go unnoticed due to the patient being covered by surgical drapes, the position of the patient and surgical team, as well as the simultaneous administration of medications that can alter clinical presentation. Therefore, sudden cardiovascular deterioration is significant in the differential diagnosis. The NAP6 study revealed that hypotension was present in severe episodes of anaphylaxis, and that cardiovascular compromise may be accompanied by bronchospasm, desaturation, and changes in capnography. A sudden decrease in $ETCO_2$ may indicate a drop in cardiac output, manifesting before a noninvasive blood pressure measurement (broad differential diagnosis: pulmonary embolism, severe hemorrhage, ventilation problems, and anaphylaxis; anaphylaxis takes priority when there is a temporal relationship with a potentially implicated drug) [2,3].

The Ring and Messmer classification was published in 1977 by German researchers Johannes Ring and Klaus Messmer in the medical journal *The Lancet* and remains effective for identifying the severity of reactions; it should be applied based on documented manifestations, where severe cardiovascular involvement may be classified as Grade III, and Grade IV corresponds to cardiac arrest [13].

Grade I: Limited to purely mucocutaneous manifestations, such as localized erythema or urticaria.
Grade II: Presents signs of moderate multiorgan involvement.
Grade III: Involves severe cardiovascular and respiratory compromise, characterized by severe hypotension, cardiac

arrhythmia (tachycardia or paradoxical bradycardia), and severe bronchospasm.

Grade IV: Constitutes total circulatory collapse or cardiac arrest.

Pathophysiology of anaphylactic shock and hemodynamic refractoriness.

The massive release of mediators leads to a decrease in systemic vascular resistance, increased capillary permeability with fluid extravasation into the interstitial space, and, as a result, a combination of vasodilation and relative hypovolemia. In some patients, there may also be myocardial depression and impaired ventricular function, which contribute to the state of shock [1,11].

This pathophysiology explains why a patient may present with severe hypotension, even in the absence of significant bleeding. Because effective circulating volume can drop rapidly, the administration of crystalloids is an essential component of treatment, along with epinephrine. In this context, initial hemodynamic refractoriness should be interpreted as a manifestation of a multifactorial pathophysiological disorder; when there is severe vasodilation, fluid extravasation, and possible myocardial depression, an isolated increase in vascular resistance may not be sufficient to restore adequate perfusion.

Vasopressor Drugs: Epinephrine vs. Norepinephrine

The first-line pharmacological treatment for anaphylaxis remains epinephrine, due to its effects on β- and α-adrenergic receptors. β1 activation improves cardiac output and contractility, while α1 activation causes vasoconstriction and increases systemic vascular resistance, and β2 activation produces bronchodilation and may help limit the excessive release of mediators by mast cells and basophils. This combination of effects allows for simultaneous action on various mechanisms that cause physiological deterioration [1,6].

As for norepinephrine, it has significant β1 activity and predominant α-adrenergic activity, but its β2 activity is much lower than that of epinephrine. Therefore, although it is an effective vasopressor for increasing vascular resistance, it does not fully replicate the pharmacological profile of epinephrine in cases of anaphylaxis. This could explain why, despite the administration of norepinephrine, blood pressure may remain abnormal when severe anaphylaxis is the predominant condition; however, this does not mean that norepinephrine is an "ineffective" drug for anaphylaxis, as it can be used as an adjunctive agent in cases of persistent hypotension. The key point is that it cannot replace epinephrine as the specific initial therapy for anaphylaxis [6,14].

In this case, a hemodynamic response was suspected following anesthetic induction; therefore, norepinephrine was initially administered. Upon observing no response to this drug and suspecting anaphylactic shock based on the patient's clinical findings, epinephrine was administered, resulting in a favorable response.

The NAP6 study showed that, although most patients were treated with epinephrine, there was room to optimize its use and supportive care, particularly in the management of cardiac arrest, where there is potential to improve the recognition and treatment of this emergency, even in healthcare systems with established protocols [3].

Management of Refractory Anaphylaxis

The strategy should include appropriate administration of epinephrine, fluid management, oxygen therapy, and airway management and its complications. If hypotension persists after initial treatment, refractory anaphylaxis should be considered, and at the same time, the possibility of alternative diagnoses should be reevaluated; in severe cases, continuous epinephrine infusion may be required. If vasoplegia persists, other vasopressors may be used as adjuncts. Angiotensin II is an option of interest in situations involving refractory vasodilator shock (limited evidence), while vasopressin is a non-catecholaminergic alternative [15].

In the case presented, two vasopressors—norepinephrine and epinephrine—were administered; the former was gradually withdrawn shortly after initiation, leaving low-dose epinephrine infusion as the mainstay of treatment, with gradual dose reductions and eventual discontinuation, so the patient did not develop refractory anaphylaxis.

Serum tryptase and Confirmation of mast cell activation

One of the most important biomarker tests for confirming the presence of mast cell activation is tryptase; therefore, whenever possible, its measurement should be included in the investigation of a perioperative anaphylaxis episode; however, interpretation requires samples that have been collected at the appropriate time (including the time of reaction onset and the exact time of collection) and compared with a baseline concentration (which may provide additional information in patients with persistently elevated concentrations). The consensus criterion used to assess a significant increase is: Acute tryptase >1.2 × baseline tryptase + 2 ng/mL. Tryptase should be viewed as an additional tool, not as a standalone test capable of confirming or ruling out anaphylaxis [16].

Documenting acute and baseline tryptase levels in this case would have provided an additional objective component to support the hypothesis of mast cell activation. However, even a non-elevated tryptase level does not, by itself, rule out the diagnosis when the clinical presentation and timeline are highly consistent with it.

Allergy testing after the event

Etiological investigation becomes a crucial component of treatment once the patient has been stabilized. The EAACI recommends a structured evaluation because, during anesthesia, multiple substances are administered and there may be many different causes of bronchospasm and hypotension; therefore, the investigation should begin with a detailed reconstruction of the anesthesia: medications used, doses, route of administration, time of administration, and the onset of the first clinical signs [4].

Depending on the suspected agent, skin tests, specific IgE testing, basophil activation tests, and, in selected situations, supervised challenge tests may be used (these should be performed exclusively in experienced centers and under strict control due to the potential risk of a severe reaction). Keep in mind that validated tests are not available for all agents, and a negative test result does not necessarily rule out a previous reaction. Therefore, the medical history and test results must be integrated to establish the diagnosis. Pharmacological challenge tests, when indicated, should [4,17].

In this patient, the investigation should focus primarily on methylene blue and ceftriaxone, although it should not be limited to these alone, given that antibiotics, neuromuscular blockers, antiseptics, and other drugs were administered at the same time; this means that all relevant agents must be reviewed chronologically.

Implications for future anesthesia

One of the primary goals of research on perioperative anaphylaxis is to ensure that patients can undergo future procedures as safely as possible. Current recommendations support the administration of anesthesia again if there is a clinical indication, provided that an appropriate diagnosis has been made and a plan has been developed to substitute and avoid the agents involved [6].

This information must be clearly documented in the medical record and communicated to the patient and family members, including the suspected agent, the nature of the reaction, and its severity. This ensures that, during future procedures, the surgical team—especially the anesthesiologist—can review this information before administering any medication.

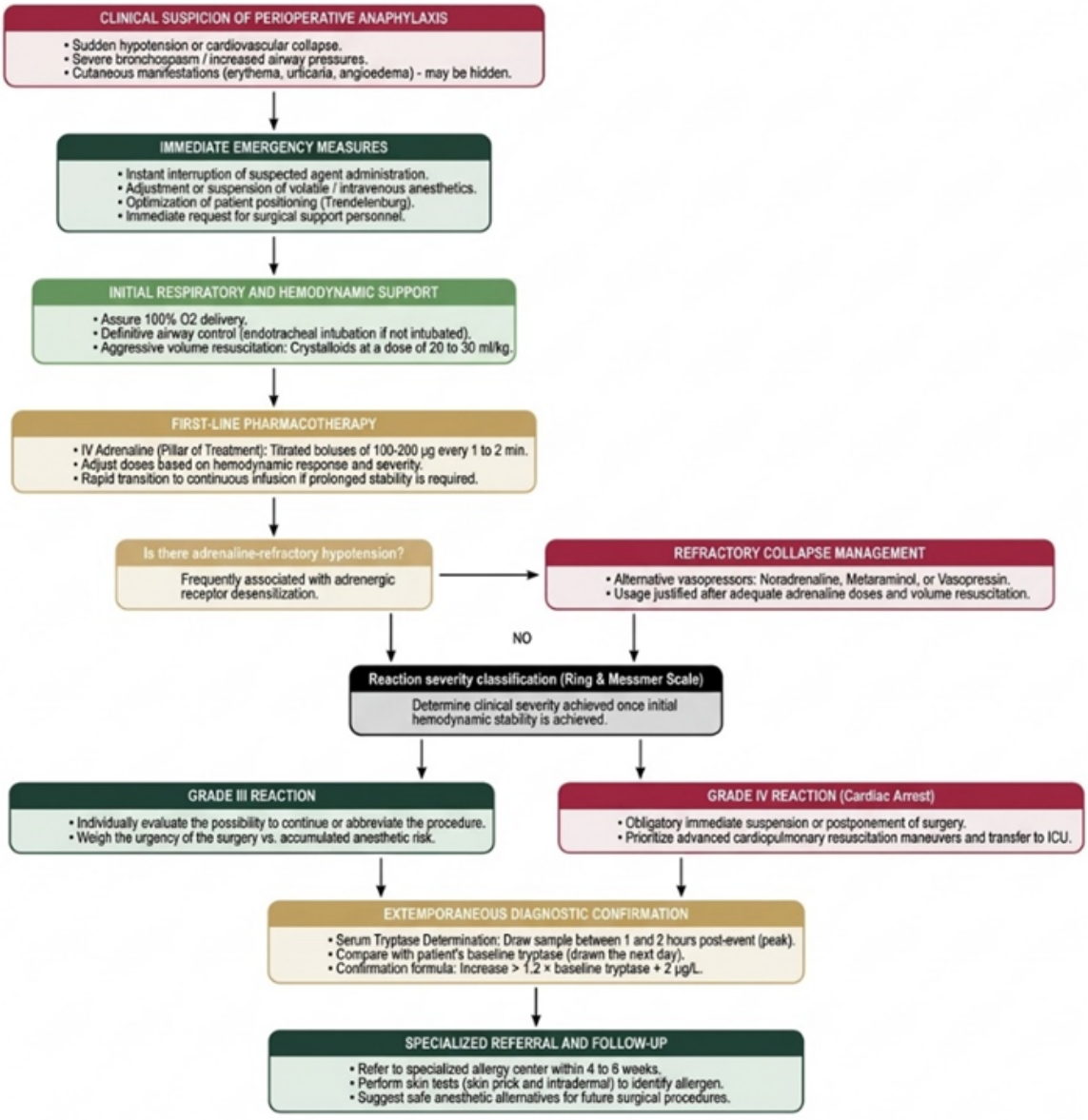


Figure 2: Algorithm for the therapeutic approach and emergency protocol for perioperative anaphylaxis: Immediate management, hemodynamic support, and rescue pharmacotherapy. Source: Adapted from Admass et al [18].

Conclusions

Anaphylaxis is a serious emergency that can occur during an anesthetic-surgical procedure, making it difficult to identify in patients under general anesthesia. The greatest strength of this case is the close temporal relationship between the administration of methylene blue and cardiovascular deterioration in a patient with a recent history of a severe anaphylactic episode, as well as the response to epinephrine and the clinical course, which support the suspicion of severe perioperative anaphylaxis.

Since patients are exposed to various agents during general anesthesia, all of these must be reviewed chronologically; however, causality cannot be established solely based on a temporal association. Similarly, even if tryptase levels are measured correctly, this does not rule out the presence of anaphylaxis. Likewise, the presence or absence of cutaneous manifestations during anesthesia has limited diagnostic value and should not be the sole criterion. Therefore, identifying this event is a significant challenge in which time is of the essence to prevent a fatal outcome.

If faced with a future case of anaphylaxis, consideration should be given to collecting laboratory samples for both specialized tests—such as tryptase—and blood gas analysis to document the patient's condition beyond the clinical response. Given that the main challenge in managing perioperative anaphylaxis does not lie solely in optimizing in vitro biomarkers or refining in vivo tests, interdisciplinary channels for continuous communication should be established between the departments of Anesthesiology, Allergy, and Surgery. This collaboration is critical to ensuring the accurate identification of the causative agent and the safe selection of pharmacological alternatives for future surgical procedures. Although the acute event was resolved according to standard protocol and consultation with Allergy was scheduled for follow-up, the absence of such an evaluation following the first event in our case is undeniable. We hope that the data provided will serve as a guide for future clinical trials and analysis of results.

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