Vol 5 No 1 April 2025
Solo Journal of Anesthesi, Pain and Critical Care

E-ISSN : 2797-0035
P- ISSN : 2776-1770

CASE REPORT

Intra-Anesthetic Anaphylactic Shock Due to Rocuronium: Diagnosis and Treatment

Fildza Sasri Peddyandhari[*], Anthonio Barswot Lengkong[✉*]

Article Info :
Submitted :
01-09-2023
Accepted :
27-04-2025
Published :
30-04-2025
https://doi.org/10.20961/sojaV5i1.78500

Authors' affiliations:
[*]Department of Anesthesiology and Intensive Care
Faculty of Medicine, Universitas Indonesia – Cipto Mangunkusumo Hospital Jakarta
[✉]Correspondence:
barswot.anthonio@gmail.com

ABSTRACT

Background: Intraoperative anaphylactic events are rare, but are important to pay attention to as they are associated with morbidity and mortality rates. Intraoperative anaphylactic events can be caused by anesthetic drugs including muscle paralysis, rocuronium, succinylcholine and attracurium are the most common causes of intraoperative anaphylaxis.

Case Illustration: An 11-year-old girl diagnosed with 3rd degree microtia with severe conductive hearing loss in the right ear. It is planned to undergo stage 1 auriculoplasty measures accompanied by rib harvesting. After induction of anesthesia with the administration of rocuronium as a muscle relaxant. Patients are suspected of anaphylaxis due to a decrease in blood pressure, increased pulse and low EtCO2 levels. Anaphylactic management is done quickly so that the patient experiences good resolution.

Conclusion: The cause of anaphylaxis during intraoperative occurrence is caused by muscle relaxant, one of which is rocuronium where most likely the cause of anaphylaxis that occurs in patients due to rocuronium administration. In addition to observing the patient's blood pressure and pulse, a decrease in EtCO2 can be a sign of anaphylactic activity. The initial goals of anaphylactic treatment are improving cardiovascular homeostasis, intravenous epinephrine administration and intravascular volume replenishment. Proper recognition and management of intraoperative anaphylactic events will provide a good prognosis.

Keywords: Anaphylaxis; Diagnosis; Rocuronium; Management.

INTRODUCTION

Intraoperative anaphylactic events are life-threatening hypersensitivity conditions mediated by immunoglobulin E (IgE). Rare, but important to note as it relates to morbidity and mortality rates.[1,2] Intraoperative anaphylactic events can be caused by anesthetic drugs including muscle relaxant. The use of rocuronium, succinylcholine and attracurium is the most common cause of intraoperative anaphylaxis.[3] Recognition and diagnosis of perioperative anaphylaxis is a challenge. During the process, many factors complicate the assessment. Drugs are generally given sequentially. Draping or covering with a sterile cloth can prevent early signs of detection, such as urticaria or angioedema. Reactions caused by mast cell mediator release can also be ambiguous where the cause of hypotension or increased resistance to airflow is due to other causes.[1,4] It is important that we are able to recognize the symptoms and signs of intraoperative anaphylaxis well, to provide prompt and appropriate treatment.

CASE ILUSTRATION

One girl, aged 11, with complaints of a right ear appeared small and did not grow perfectly. After physical examination and support, the patient was diagnosed with 3rd degree microtia accompanied by severe conductive type hearing loss in the right ear. Furthermore, it is planned to undergo elective surgery i.e. stage 1 auriculoplasty accompanied by rib harvesting. At the pre-anesthesia visit, no other complaints were found. The presence of symptoms of infection or disorders of the upper airway is denied. History of asthma and allergies or other comorbid diseases is denied. On physical examination, only abnormalities were found in the right ear, where the auricle appeared small, there was no ear hole and structures that could not be evaluated. Other physical examinations within normal limits. The results of laboratory tests or other supports are within normal limits. Patients are assigned with ASA (American Society of Anesthesiologist) I physical status, without airway complication. Furthermore, the patient is decided to perform anesthesia with general anesthesia and after the procedure will be treated again in the treatment room. Patients and families are educated about the anesthesia procedure that will be performed and satisfied before the procedure.

The patient arrived at the operating room without complaint with a stable condition for anesthesia. Pre-induction evaluation was carried out on the patient and obtained body weight 41 kg, height 141 cm with blood pressure 112/60 mmHg, pulse 90 times per minute, respiratory rate 18 times per minute, oxygen saturation 99% room air. Intravenously induction with lidocaine 20 mg and fentanyl 150 mcg, followed by titration of propofol 80 mg, after ventilation is successfully mastered, rocuronium 40 mg is given. Patients were intubated with non-kinking ETT number 6.5, a depth of 17 cm by comparing until the breath sound sounded the same in both lungs. After intubation, haemodynamic obtained blood pressure 108/62 mmHg, pulse 86 times per minute, respiratory rate 12 times per minute, oxygen saturation 100% with pressure control ventilator mode 14/12/5/40%. Capnograph waves appear normal with EtCO2 32 – 35 mmHg, auscultation of vesicular breath sounds and no ronkhi or wheezing. We were given cefazolin antibiotics at a dose of 2 grams intravenously, previously skin tests had been carried out and no allergic reactions were obtained.

After approximately 35 minutes, the induction was successful and continued with incision action by the operator, starting from rib harvesting. When the rib harvesting procedure lasted about 13 minutes, there was an increase in the patient's pulse up to 104 times per minute, an increase in blood pressure of 142/113 mmHg, EtCO2 33 mmHg and a tidal volume of 350-380 mL. The condition continues until 2 minutes later, the patient gets tachycardia and EtCO2 is illegible with the below not expanding. The pulse reaches 133 times per minute, the oxygen saturation is still at 100% but the volume tidal reads at only 30 mL.

On examination of the breathing tube and connection with the machine, no kinking or leakage was found. Oxygen saturation began to drop to 85% with the condition of tachycardia, the action was decided to be temporarily stopped. Ventilation was followed by manual bagging, bagging was heavy and auscultation inspection found inadequate ventilation. The patient's condition is increasingly desaturated and cyanosis appears, in haemodynamic the patient's blood pressure drops to 88/47 mmHg. Removal of ETT was carried out, no blockage was found in ETT. Manual bagging with a lid is continued, it is still

felt quite heavy, there is no visible lung development, bronchospasm is suspected. Furthermore, ephedrine 10 mg intravenously is given, ventilation assistance is given with a fraction of 100% in the condition of the patient is still 30-40% desaturated with a tidal volume of 90-100 ml and Ppeak of 30-35 mmHg. Anesthesia is deepened by administering propofol 50 mg, fentanyl 100 mcg and rocuronium 20 mg, then intubation is done. After re-intubation, there is still no chest wall development, minimal left apex lung sound and wheezing is obtained in the lower right lung rib harvesting area. In haemodynamic, patients still get hypotension and tachycardia, blood pressure 82/48 mmHg, pulse 119 times per minute, suspected anaphylactic reaction. Furthermore, management was carried out including the administration of dexamethasone 5 mg and epinephrine 0.02 mg intravenously, and epinephrine 0.04 mg through ETT. Loading of 300 ml crystalloid liquid and 200 ml colloidal fluid is carried out. Then given aminophylline loading dose 100 mg intravenously in 10 minutes, followed by a maintenance dose of 40 mg per hour intravenously, and drip epinephrine 0.02 mcg/kg/minute.

After that, ventilation improved, the chest began to look expanded with a tidal volume of 200-250 ml, but the peak was still 30 mmHg. Ketamine drip 30 mg/hour is given and gradually saturation begins to read. In approximately 7 minutes, blood pressure became 109/68, pulse 133 times per minute, oxygen saturation 98%, without apparent cyanosis. In patients, thoracic ultrasound evaluation was carried out and no pneumothorax was found. Furthermore, the action was resumed, but only the rib harvesting area was closed, the auricularly action was decided to be postponed. The patient is given a combination inhalation of pulmicort and combivent. Epinephrine drip and ketamine are stopped slowly until the action is completed, but aminophylline drip is continued. After the procedure is completed, patient monitoring is carried out in the PICU room without extubation.

DISCUSSION

Background and Definition

Intraoperative anaphylaxis is a fairly rare occurrence but is important to pay special attention to because it is associated with morbidity and mortality rates. Anaphylaxis itself is defined as a

severe and potentially life-threatening systemic hypersensitivity reaction.

Anaphylaxis that occurs during the perioperative period is a clinical syndrome involving many organ systems and is usually caused by drugs or substances used for anesthesia or surgery. Anaphylaxis is a rare and potentially fatal complication with a broad spectrum of clinical presentation. The ability to recognize and treat them in a perioperative setting is essential for any anesthesiologist.[2,5]

Incidence and Causative Agent

Based on the data, there are 1 in 10,000 – 20,000 cases of perioperative anaphylactic. These incidence rates may vary from country to country, where there are differences in local practices and drug preferences, completeness of allergy evaluations and genetic or environmental factors.[2] The causes of perioperative anaphylaxis are generally associated with agents administered intravenously. The UK study found the main causative agents of anaphylaxis were muscle relaxant (25%) and antibiotics (48%), while in France muscle paralysis (61%) was the most common drug, followed by antibiotics (18%). β-lactam antibiotics are a class of antibiotics identified as causing anaphylaxis.[1] Rocuronium and succinylcholine are known to be the most common causes of muscle paralysis. Rocuronium occurs in 1 in 2,499 cases, while for succinylcholine it occurs in 1 in 2,080 cases and attracurium occurs in 1 in 22,451 cases.[3,6]

The incidence of perioperative anaphylaxis causes 9-19% of surgical complications globally and 5-7% of anesthesia-related deaths. Secondary brain injury due to anoxia is the most important morbidity and occurs in approximately 2% of patients with anaphylaxis.[7]

Differential Diagnosis and Diagnosis

The onset and severity of allergic reactions vary with different causes and differ in each patient. When there is exposure to an allergen by injection of an allergen, such as allergen vaccines, insect stings and some medications, the onset of the reaction generally occurs rapidly and the severity of the allergic reaction gets worse if the onset gets faster. The onset of anaphylaxis to an allergen sting or injection usually proceeds quickly: 70% begin within <20 minutes and 90% within <40 minutes. There are four known patterns of anaphylactic reactions, namely, immediate, biphasic, protracted and

delayed. Biphasic anaphylaxis has a direct phase with a period of improvement and response to initial therapy, but usually recurs symptoms 2 - 6 hours from the initial reaction. Protracted anaphylaxis causes prolonged manifestations (usually respiratory distress or hypotensive shock) for 5 – 32 hours and is often resistant to treatment. While for delayed anaphylaxis, reactions without a direct phase generally do not occur, there are not many reports of delayed anaphylactic reaskance.[8]

The diagnosis of anaphylaxis is a clinical diagnosis. Lab tests are not useful in diagnosing anaphylaxis at the time the condition occurs because it takes time to process and is prone to negative or false positive results. The introduction and diagnosis of perioperative anaphylactic is a challenge for us, because it will be difficult for us to recognize when the patient is covered with sterile cloth. So knowledge is needed to know the symptoms and signs that can be caused. Clinically anaphylaxis can be divided into 4 groups (Ring and Messmer classification), based on the severity of anaphylactic. First, skin symptoms include erythema, urticaria, and angioedema. Second, it can be measured but not life-threatening symptoms, namely skin symptoms, hypotension, and tachycardia. Third, life-threatening symptoms such as tachycardia or bradycardia, arrhythmia and bronchospasm. Four, respiratory arrest and cardiac arrest. The sooner it is recognized, the better the treatment decision making and the better the patient's prognosis.[1,5] Anaphylaxis should be suspected in case of unexplained vasopressor refractory hypotension, or unexplained ventilation difficulties and bronchospasm.[9]

Nonallergic differential diagnosis of peri-operative anaphylaxis associated with anesthesia or other surgical management. If symptoms occur from only one organ and serum triptase is not elevated, it may not be anaphylaxis. When hypotension occurs, as a single symptom, several possible causes are pharmacological effects of other drugs, massive bleeding or other types of shock. It can also be seen in induction of patients taking tricyclic antidepressants or antihypertensive drugs. Symptoms of the upper airway can be caused by swelling of the airway after traumatic intubation or angioedema of patients with ACE inhibitors or hereditary angioedema. Symptoms of the lower airway may be induced by aspiration of

gastric contents, a history of previous airway hyperreactivity, undiagnosed or uncontrolled asthma. Isolated skin symptoms can result from the release of non-specific histamine in response to some medications, most commonly opioids. This reaction is not severe and can be reduced or prevented by administering antihistamines. Other differential diagnoses of anaphylaxis include asthma exacerbations, tension pneumothorax, myocardial ischemia, pulmonary embolism, C1 esterase deficiency, mastocytosis, and clonal mast cell disorders.[10]

In addition to observing the patient's blood pressure and pulse, EtCO2 observation can be a sign of anaphylactic activity. A decrease in EtCO2 is one indication of an acute hypersensitivity reaction in intra-anesthesia, but must still be considered with the clinical symptoms of hypersensitivity that occur.[11,12] In these patients, rapid hemodynamic changes are obtained, ranging from tachycardia, a decrease in EtCO2 and hypotension simultaneously. After manual bagging is done, bagging feels heavy and inadequate ventilation so that it can be categorized in anaphylactic category 3, where bronchospasm has occurred in patients.

The causes of anaphylactic events during intraoperative events are caused by drugs and materials such as latex. The group of drugs that include drugs that cause anaphylaxis include muscle paralyzers, antibiotics, hypnotic drugs, opioids, local anesthetic drugs and colloids. Based on observational data from cohort studies from 2006 to 2012, muscle paralysis is known to most often cause anaphylactic events in about 50-70% of cases, rocuronium and succinylcholine are the most common causes when compared to attracurium. The class of muscle paralyzing drugs can induce the release of triptase and histamine from mast cells, which will exert a direct vasodilating effect on blood vessels and induce changes in capillary permeability, urticaria, erythema, angioedema, hypotension, and bronchospasm.[3,13,14]

Antibiotics are also a major cause of anaphylaxis in surgical patients. Anaphylaxis triggered by antibiotics is generally caused by penicillin and cephalosporins (70%) that share a β-lactam ring. Therefore, an oral provocation test in patients with a suggestive clinical history and a negative skin test result of β-lactam is recommended. Anaphylaxis to hypnotics such as thiopental or propofol is rarely

reported. Anaphylaxis to opioids is also very rare. Anaphylaxis to local anesthesia is extremely rare and its frequency decreases due to a decrease in the use of ester groups of local anesthesia. Most allergic reactions are caused by the general metabolic product of the local anesthetic ester, the para-amino benzoic acid.[2] In patients in this case, muscle relaxant in the form of rocuronium is given. This operation is the first operation for patients. Previous history of allergies is denied, before the procedure, a hypersensitivity test for antibiotics with negative results is performed. This indicates that most likely the cause of anaphylaxis that occurs in patients is due to the administration of rocuronium.

Pathophysiology

Anaphylactic reactions arise from activation of mast cells and basophils through mechanisms that generally involve crosslinking of immunoglobulin (Ig)E and aggregation of high-affinity receptors for IgE, FcεRI. Upon activation, mast cells and/or basophils rapidly release preformed mediators from secretory that include histamine, tryptase, carboxypeptidase A, and proteoglycans. Further fosfolipase activation pathway A2 (PLA2), followed by cyclooxygenase and lipoxygenase, produces arachidonic acid metabolites, including prostaglandins, leukotrienes, and platelet activation factor (PAF). Inflammatory cytokines, tumor necrosis factor-α (TNF-α) act as preformed mediators, and also as late-phase mediators with other cytokines and chemokines. Many of these mediators contribute to the pathophysiology of anaphylaxis. Histamine stimulates vasodilation, and increases vascular permeability, heart rate, heart contraction, and glandular secretion. Prostaglandin D2 acts as a bronchoconstrictor, pulmonary and coronary vasoconstrictor, and peripheral vasodilator. Leukotrienes cause bronchoconstriction, increase vascular permeability, and improve airway remodelling. Furthermore, PAF which is a strong bronchoconstrictor and increases vascular permeability. While TNF-α activates neutrophils, recruits other effector cells, and increases chemokine synthesis. It is these overlapping and synergistic physiological effects that contribute to the overall pathophysiology of anaphylaxis variations, such as urticaria, angioedema, bronchospasm, other respiratory symptoms, hypotension,

syncope, cardiovascular or other gastrointestinal symptoms.[1,15,16]

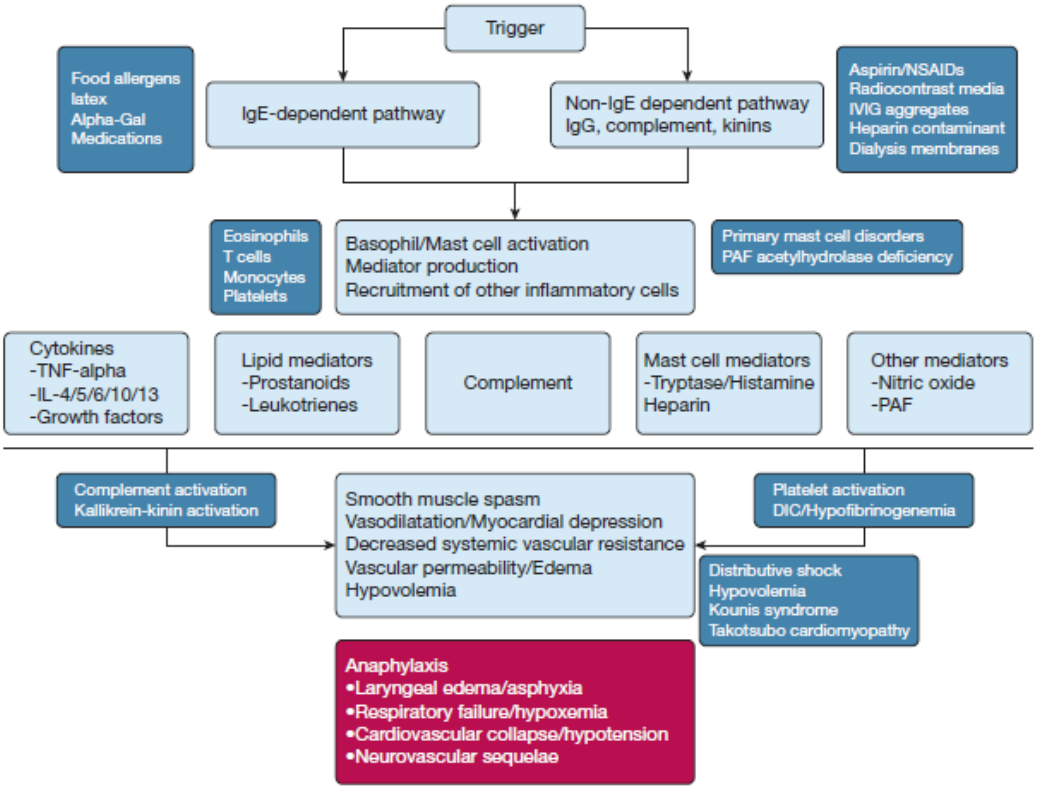

Figure 1. Mechanism of Anaphylactic Pathway

Management

Early recognition and early management of anaphylactic events is particularly important for a patient's prognosis. Treatment of perioperative anaphylactic patients that must be done is:

a. Stop administering the drug.
b. Also stop the anesthetic drug that is being run during the induction period.
c. Maintain airway with 100% oxygen administration.
d. Request help, especially for grade 3 and 4 anaphylactic reactions.
e. Consider giving epinephrine in case of anaphylactic reactions of grades 3 and 4. It can be administered intramuscularly at a dose of 0.5 mg to 1 mg (0.5 to 1 mL 1:1,000) and can be repeated every 10 minutes according to arterial pressure and pulse rate until improvement occurs. Alternate doses, 50 to 100 µg

intravenously (0.5 to 1 mL 1:10,000) for 1 minute have been recommended for hypotension with further dose titration as needed. The dose of pediatric epinephrine depends on the age of the child, children with age >12 years 500 µg IM (0.5 mL), 6-12 years 250 µg IM (0.25 mL), >6 months-6 years 120 µg IM (0.12 mL), <6 months 50 µg IM (0.05 mL).

f. Start a quick intravenous infusion with colloidal or crystalloid. Adult patients may require 2 to 4 L of crystalloids.
g. Position the supine patient with the Trendelenburg position.
h. Shorten the operating procedure.

The initial goal of anaphylactic treatment is to improve cardiovascular homeostasis, intravenous epinephrine administration and intravascular volume filling are important parts of treatment. If bronchospasm occurs, beta 2 inhalation can be given agonists, which can be given bolus and then drip continue if there is persistent bronchospasm. In addition, corticosteroids can also be given (100 to 500 mg hydrocortisone slowly IV).[3,4,14]

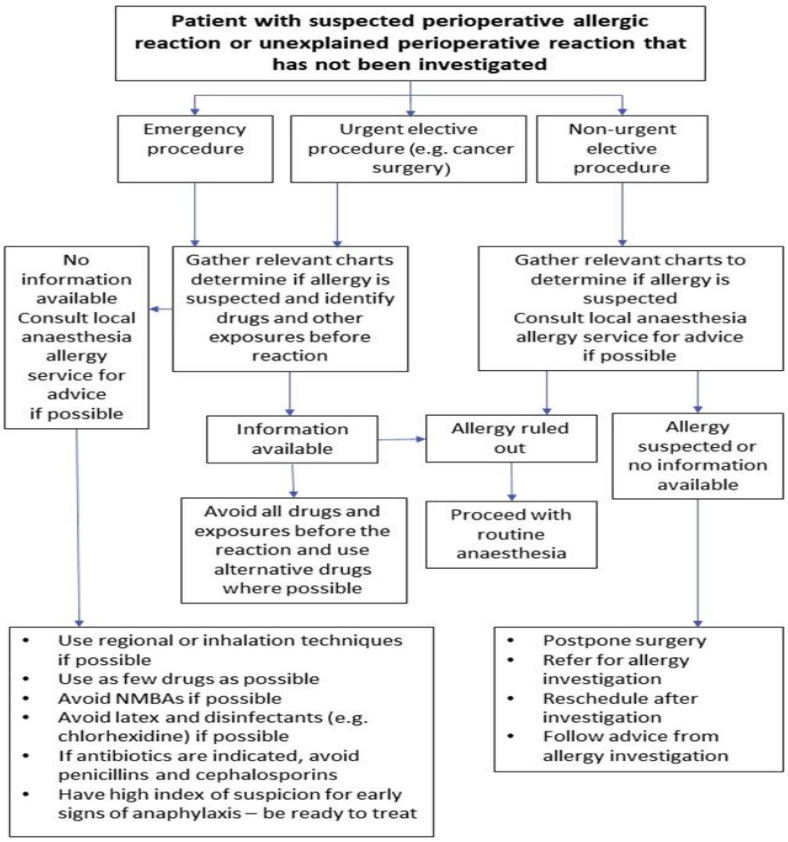

Figure 2. Management of patients with suspected perioperative allergic reactions[10]

In this case, bronchospasm is initially suspected due to a blockage in the endotracheal tube, so that after the ventilation bagging does not enter and like there is resistance, ETT is removed and tried to be done manually bagging. After ETT is removed and evaluated, there are no folds or mucus/glue in it. When manual bagging is done, there are difficulties where bagging feels heavy and difficult, so manipulation is carried out by providing positive pressure ventilation with 100% oxygenation.

Furthermore, in patients it is decided to re-intubate, with additional administration of anesthetic drugs in the form of fentanyl and rocuronium. Intubation was successful, but it was still felt heavy when bagging and ventilation were judged inadequate. After re-evaluation is carried out, the patient is suspected of anaphylaxis, so epinephrine is immediately given intravenously and intratracheal tubes and added aminophylline administration by bolus and drip. After treatment is performed, bronchospasm begins resolution, the patient's haemodynamic improve and surgery can continue. The operation proceeds with only closing the parts that have been incised. The auriculoplasty procedure for this patient is an elective procedure so that a delay in the procedure is carried out.

The next important step is to conduct an evaluation after perioperative anaphylaxis. This evaluation serves to determine:[1]

a. Determine whether anaphylaxis has occurred, or determine alternative differential diagnoses.
b. Identify the underlying mechanisms and drugs that cause anaphylaxis.
c. Identify safe alternatives to causative drugs.
d. Provide explanations to patients and detailed medical reports, including suggestions for subsequent anesthesia.

Measurement of histamine and tryptase release to determine the occurrence of anaphylaxis. Histamine is a mediator produced by basophils and mast cells, measured in plasma (N<10 nmol L−1). Histamine concentrations should be measured within 30 minutes after the onset of anaphylaxis, and up to 1-2 hours after. Triptase is a neutral serine protease contained mainly within mast cells. Measurements should be taken within 2 or 6 hours after the onset of anaphylaxis.

In vivo skin tests in the form of skin prick tests are the gold standard for detecting allergies mediated by IgE, by

exposing skin mast cells to suspected allergens. Evaluation for 4-6 weeks after administration is highly recommended in preparation for the continuation of surgery. Good preparation, will improve the output in the patient.

The selection of the right drugs will affect the mortality and morbidity of patients who perform surgery.[2,5] Furthermore, in these patients, if anesthesia will be carried out again, it is recommended to ensure that every drug used is tested for allergies first. In these patients should be re-evaluated after 4-6 weeks.

CONCLUSION

Perioperative anaphylactic events can cause mortality if not treated quickly and appropriately. Rocuronium is one of the causes of perioperative anaphylactic events. The ability to recognize intraoperative anaphylactic symptoms and signs must be improved because as an anesthesiologist it will be difficult to assess the patient's condition when it has been covered with a sterile cloth. It is a challenge how to recognize that anaphylactic event quickly and precisely through adequate monitoring during the induction process, even until surgery or action begins. Not only recognition of symptoms and signs is needed, but also the ability to quickly and precisely manage. Good management will produce good outcomes for patients. Based on recommendations, anaphylactic actions or procedures are not differentiated based on cause, whatever the cause, the action taken remains the same.

REFERENCES

1. Dewachter P, Savic L. Perioperative anaphylaxis: pathophysiology, clinical presentation and management. *BJA Educ*. 2019;19(10):313-320. doi:10.1016/j.bjae.2019.06.002
2. Dewachter P, Mouton C, Emala C. Anaphylaxis and Anesthesia: Controversies and New Insights. *Anesthesiology*. 2009;111:1141-1150. doi:10.1097/ALN.0b013e3181bbd443
3. Reddy JI, Hannam JA. Anaphylaxis Is More Common with Rocuronium and Succinylcholine than with Atracurium. *Perioper Med*.
4. Anaphylaxis during general anesthesia, the intraoperative period, and the postoperative period. *J Allergy Clin Immunol*. 1998;101(6):S512-S516.

doi:10.1016/S0091-6749(18)30584-0

5. Simons FER, Ardusso LRF, Bilò MB, et al. World Allergy Organization Guidelines for the Assessment and Management of Anaphylaxis. *World Allergy Organ J*. 2011;4(2):13-37. doi:10.1097/WOX.0b013e318211496c

6. Takahashi K, Tanaka S, Watanabe M, Yamakage M. Rocuronium-induced anaphylaxis: a case report. *JA Clin Rep*. 2019;5(1):81. doi:10.1186/s40981-019-0303-5

7. Admass BA, Hassen AE, Agegnehu AF, et al. Management of perioperative anaphylaxis: Systematic review. *Int J Surg Open*. 2023;52:100595. doi:10.1016/j.ijso.2023.100595

8. Golden DBK. Patterns of Anaphylaxis: Acute and Late Phase Features of Allergic Reactions. In: Bock G, Goode J, eds. *Novartis Foundation Symposia*. John Wiley & Sons, Ltd; 2008:101-115. doi:10.1002/0470861193.ch9

9. Yim DR. An update on PERIOPERATIVE ANAPHYLAXIS. Published online 2016.

10. Garvey LH, Dewachter P, Hepner DL, et al. Management of suspected immediate perioperative allergic reactions: an international overview and consensus recommendations. *Br J Anaesth*. 2019;123(1):e50-e64. doi:10.1016/j.bja.2019.04.044

11. Gouel-Chéron A, De Chaisemartin L, Jönsson F, et al. Low end-tidal CO2 as a real-time severity marker of intra-anaesthetic acute hypersensitivity reactions. *Br J Anaesth*. 2017;119(5):908-917. doi:10.1093/bja/aex260

12. Erlich C, Lamer A, Moussa MD, Martin J, Rogeau S, Tavernier B. End-tidal Carbon Dioxide for Diagnosing Anaphylaxis in Patients with Severe Postinduction Hypotension. *Anesthesiology*. 2022;136(3):472-481. doi:10.1097/ALN.0000000000004123

13. Butterworth JF, Mackey DC, Wasnick JD, Morgan GE, Mikhail MS. *Morgan & Mikhail's Clinical*

Anesthesiology. Sixth edition. McGraw-Hill Education; 2018.

14. Kim SM, Oh S hoon, Ryu SA. Treatment of rocuronium-induced anaphylaxis using sugammadex - A case report -. *APM*. 2020;16(1):56-59. doi:10.17085/apm.20074

15. Peavy RD, Metcalfe DD. Understanding the mechanisms of anaphylaxis. *Curr Opin Allergy Clin Immunol*. 2008;8(4):310-315. doi:10.1097/ACI.0b013e3283036a90

16. LoVerde D, Iweala OI, Eginli A, Krishnaswamy G. Anaphylaxis. *Chest*. 2018;153(2):528-543. doi:10.1016/j.chest.2017.07.033

17. Kolawole H, Marshall SD, Crilly H, Kerridge R, Roessler P. Australian and New Zealand Anaesthetic Allergy Group/ Australian and New Zealand College of Anaesthetists Perioperative Anaphylaxis Management Guidelines. *Anaesth Intensive Care*. 2017;45(2):151-158. doi:10.1177/0310057X1704500204