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Case Report

Anesthetic management of allergic hypersensitivity during a surgical procedure: A case report and literature review

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Abstract

Introduction: Perioperative anaphylaxis is a rare but serious, life-threatening condition that presents challenges in diagnosis and management. The incidence of severe perioperative reactions is estimated to be approximately 1:7,000–10,000.

Case report: A 75-year-old patient underwent a surgical procedure due to adhesions of previous surgeries. She had a serious anaphylactic reaction in the intraoperative period, which was quickly addressed with fluid and adrenaline administration.

Discussion: Proper management involves both immediate stabilization of the patient and identification of the causing agent. This identification is essential to avoid the recurrence of the event in other surgeries. A previous history of perioperative hypersensitivity reaction is the main risk factor for anaphylaxis.

Introduction

The perioperative period represents a pharmacologically unique situation, during which patients are exposed to multiple drugs and chemicals, including anesthetics, analgesics, antibiotics, antiseptics, blood products, heparin, polypeptides, and plasma expanders, which have the potential to produce a variety of both predictable and unpredictable adverse reactions. Adverse drug reactions have been recognized as one of the most common causes of morbidity and mortality in the practice of anesthesiology [1].

During the perioperative period, patients are exposed to multiple agents that can induce hypersensitivity reactions, with an estimated incidence of 1:10,000 anesthetic procedures. The mortality of anaphylactic reactions is around 3.5 and 4.8% [2].

Although almost every medication and substance used during anesthesia and surgery were implied as causes (including hypnotics, opioids, local anesthetics, colloids and dyes), the most common cause of intraoperative anaphylaxis is neuromuscular blocking agents (NMBAs), antibiotics, disinfectants and latex. In pediatric patients, latex was the substance most frequently related to allergic reactions [1].

Although the clinical presentation and early management are similar, perioperative hypersensitivity reactions have two mechanisms: immunological mechanisms (allergic reactions) and non-immunological mechanisms [3].

Mediated by IgE, immune reactions account for 60% of all reactions and their severity may increase in subsequent surgeries. These reactions go through a sensitization phase, with activation of TH2 and B lymphocytes, and the production of specific IgE antibodies that bind to high-affinity receptors on mast cells and basophils. In a second contact with the sensitizing agent and its binding to specific IgE antibodies, mediators such as histamine, tryptase, PG2, leukotrienes, thromboxane A2, platelet-activating factor, chemokines and cytokines (such as tumor necrosis factor) are released, leading to the development of the reaction. This leads to the recruitment and activation of additional inflammatory cells producing an amplification of the response over several hours. In some cases, a reaction may occur at the first contact, which may be due to cross-reaction to other substances to which the patient is already sensitized [2-4].

The term "allergy" alludes to a hypersensitivity reaction initiated by immunological mechanisms, therefore, hypersensitivities may be allergic and non-allergic [2].

It should be noted that in some cases a reaction may occur on first contact, which may be due to cross-reactivity with other substances to which the patient is sensitized. On rare occasions, immune reactions may not be IgE-mediated, as reported for dextrans, which create IgG immune complexes and activate the complement system; in these cases, the reactions are less severe [3].

Hypersensitivity reactions are classified

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according to severity into grade 1 hypersensitivity reactions, when only mucocutaneous signs are present (urticaria, erythema with or without angioedema), grade 2 hypersensitivity reactions, when they include mucocutaneous signs, accompanied by moderate hypotension, bradycardia or bronchospasm; and grade 3 hypersensitivity reactions or anaphylaxis, when manifested as severe hypotension, tachycardia or bradycardia, shock and/or bronchospasm in addition to mucocutaneous signs [5]. Although most systemic anaphylaxis reactions are immediate, delayed reactions may occur [4].

We describe the perioperative care of a patient who underwent exploratory laparotomy at our institution, who presented with a severe anaphylactic reaction during the procedure.

Case report

A 75-year-old female patient diagnosed with post-hysterectomy adhesions was admitted for surgical treatment, primarily by laparoscopy.

A pre-anesthetic evaluation was performed and the patient was classified as ASA II. The patient did not report previous allergies. All laboratory and imaging tests were normal. An echocardiogram was performed which revealed an ejection fraction of 60% and a grade 1 diastolic dysfunction.

Anesthetic management consisted of continuous epidural anesthesia associated with balanced general anesthesia. The patient was monitored according to ASA standard including ECG, NIBP, peripheral arterial oxygen saturation, capnography, gas analysis and temperature. Initial vital signs were HR= 90bpm, BP= 130x90mmHg. An 18G peripheral venous cannula was introduced. After premedication with midazolam 3mg, epidural anesthesia was performed in the T10/T11 space using the loss of resistance technique and ropivacaine 0.2% 12ml associated with morphine 2mg was administered. Then, an epidural catheter was inserted for postoperative analgesia.

Anesthetic induction was achieved uneventfully with propofol 2.5mg/kg and remifentanil 0.3µg/kg/min. Neuromuscular blockade was obtained with rocuronium and when the sequence of four (TOF) reached zero, the trachea was intubated with a simple number 7.5 tracheal tube. TOF was monitored throughout the surgery. Anesthesia was maintained with 0.1µg/kg/min of remifentanil and sevoflurane.

The patient remained hemodynamically stable during laparoscopy, which lasted approximately 45 minutes.

Due to technical difficulties, the surgery was converted to a laparotomy. Suddenly, the patient presented sudden tachycardia, decreased end-tidal CO2 and hypotension refractory to ephedrine and metaraminol (NIBP=58x30mmHg). Epinephrine boluses were administered, resulting in a NIBP=70x40mmHg. in the face of it all,, a central venous access was obtained in the right internal jugular vein and an arterial access in the left radial artery. Epinephrine infusion was started at 0.1µg/kg/min and noradrenaline at 0.15µg/kg/min, with stabilization of blood pressure. 1000ml of crystalloids and 500mg of hydrocortisone were administered. Point of care echocardiography performed showed normocontractile RV, hyperdynamic LV, absence of hypokinesias.

The total surgical time was approximately 2 hours. Exploratory laparotomy revealed only bands and no considerable bleeding was observed. In the rest of the case, the patient remained tachycardic, with systolic blood pressure values ranging from 85-90 mmHg, receiving inotropic and vasopressor support with adrenaline and noradrenaline.

At the end of the surgery, the surgical drapes were removed, showing a skin rash on the trunks and lower limbs. In view of the situation, the main diagnostic hypothesis was anaphylactic reaction, the most likely agent of which is latex.

Postoperatively, the patient was transferred to the ICU, dependent on vasopressors and mechanical ventilation.

Cystoscopic hydrodistension under general anesthesia

During the cystoscopy procedure under general anesthesia, saline was instilled into the bladder at a height of 80-100 cm away from the pubic symphysis. The bladder was distended under gravity for 8-10 min, and the volume of bladder infusion was less than 700 ml. The maximum bladder perfusion capacity was recorded. Then, the bladder walls were observed for glomerulations or Hunner's ulcers, and photographs were taken. Biopsies were carried out on suspicious lesions, and routine pathological examinations were performed. The three-chamber catheter was indwelled, and the bladder was continuously irrigated or directly drained. The catheter was removed on the first day after the examination. Positive criteria for IC/BPS were diffuse submucosal punctate hemorrhage, with a range of more than three quadrants; each quadrant exceeded 10. The maximum volume of the water-dilated bladder was more than 200 ml in all patients.

Discussion

Anaphylactic reactions during anesthesia can be fatal and are usually caused by drugs or substances used for anesthesia or surgery. Perioperative anaphylaxis (PA) is a rare but serious and life-threatening condition that presents challenges in diagnosis and management. The incidence of severe perioperative reactions is estimated to be approximately 1:7,000–10,000. Proper management involves both immediate stabilization of the patient and identification of the causative agent. This identification is essential to avoid recurrence of the event in surgeries [6].

A previous history of perioperative hypersensitivity reaction is the greatest risk factor for anaphylaxis. Other risk factors include advanced age, female gender, type of procedure, comorbidities such as chronic lung disease, coagulopathy, malignancy, fluid and electrolyte disorders, ASA III and IV patients, obesity, and coronary artery disease [6].

The diagnosis of anaphylaxis is often difficult in the perioperative period, at the time of its presentation; therefore, clinical suspicion is critical, as cardiovascular collapse may be the only manifestation. Tryptase levels should be obtained 1-2 h after the event to help complete the diagnosis [6].

The clinical manifestation of anaphylaxis in anesthesia is similar to that of other forms of anaphylaxis, although there are specific aspects. Given that the patient is usually unconscious, covered by sterile fields, and cannot express what is happening, the prodromal symptoms (pruritus, dyspnea or discomfort) may not be recognized. Moreover, anesthetics can cause alterations that may mimic an early anaphylactic reaction [7].

Respiratory symptoms, such as bronchospasm, are less frequent and are seen in only about half of all patients, particularly those with a previous diagnosis of asthma. The first sign may be an increase in resistance to ventilation or a decrease in oxygen saturation [3,4].

Some mild cases may spontaneously recover, meaning the reaction was gone unnoticed. Subsequently, re-exposure can lead to a severe, potentially fatal reaction [3].

Reactions can occur at any time during anesthesia, although about 90% occur during the induction phase, after intravenous administration of the causing agent (especially antibiotics, NMBAs, and hypnotic drugs).

Sometimes reactions can occur with a longer latency period, depending on of contact pathways (cutaneous, mucosal) [3].

Latex reactions tend to occur late, usually after significant mucosal exposure. Risk factors for latex allergy include (I) atopy, (II) history of immediate hypersensitivity after exposure to latex, (III) patients with spina bifida, (IV) multiple surgeries especially in childhood, (V) occupational exposure to latex, and (VI) allergy to fruits (avocado, banana, kiwi, etc.). The goal of primary prevention is to reduce latex sensitization [7].

As a differential diagnosis of anaphylaxis, any condition that predisposes to hypotension and shock must be considered as the initial presentation, such as myocardial ischemia, cardiac arrhythmias, pulmonary embolism, hemorrhage, sepsis and hypovolemia. Hypotension on induction of anesthesia may be observed, particularly in patients taking antihypertensives or tricyclic antidepressants [6].

Treatment of hypersensitivity reactions should be instituted as early as possible as this influences the patient's prognosis, especially in severe reactions [3]. The first step towards an anaphylactic reaction treatment is the withdrawal of the drug most likely to be the reaction's cause. Early epinephrine administration is mandatory to avoid airway compromise and cardiovascular collapse. Epinephrine leads to the interruption of the pre-formed mediator effects and the prevention of new mediators' release. Epinephrine intravenous doses ranging from 5 to 10mcg (0.2mcg/kg) are used in mild to moderate hypotension treatment. Intravenous doses from 0.1 to 0.5mg are used when there is cardiovascular collapse [8]. Anaphylactic reactions involve changes in vascular permeability, which implies that up to 35%-50% of the intravascular volume can migrate into the interstitial space within 10 minutes. Epinephrine is the drug of choice for the treatment of anaphylaxis, and the delay in its administration negatively influences the prognosis of severe reactions. There is no contraindication to the use of epinephrine during a reaction, although the dosage must be adjusted based on severity to avoid serious adverse effects, especially in patients with heart disease [3]. In cases where the patient is taking β-blockers or has heart disease, other vasoactive agents may be used. Norepinephrine, ephedrine, methoxamine, phenylephrine, and dopamine can be given as an intravenous bolus or continuous infusion [3]. Glucagon can also be used as a rescue medication in patients using β-blockers who may not respond to epinephrine [3]. Another medication that may be used in specific situations is methylene blue. This drug may be useful because of its ability to interfere with the action of nitric oxide in the smooth muscle of vascular walls. It must be given in combination with epinephrine. Corticosteroids are not indicated in the acute phase of the reaction, but can be used to prevent later symptoms [3].

The table below is a summary of the pharmacological management of allergic hypersensitivity reactions, according to severity.

Results

IFollowing stabilization, the patients must be observed in intensive care therapy within the first 24 hours, because the anaphylaxis may persist or present a biphasic response.

Before the patient is discharged, the anesthesiologist must provide the patient with a clinical report containing all relevant information about the reaction, including its severity, any treatment administered and possible causative agents [3].

This report demonstrated a severe late anaphylaxis in the intraoperative period. Although the patient had no previous history of allergies, she underwent other previous surgical procedures and some bladder catheterizations, which are known to be risk factors for latex allergy. Bedside echocardiography is an extremely important tool, as it helps in the differential diagnosis of other possible complications such as myocardial ischemia or pulmonary thromboembolism. Visualization of typical skin changes after removal of the surgical drapes confirmed the diagnosis of anaphylaxis. Skin tests should ideally be obtained 4 weeks to 6 months after the event so that the cause can be identified. In conclusion, the anesthesiologist has an important role to play in both the prevention and treatment of hypersensitivity reactions. Prior to surgery, the anesthesiologist should assess the medical history to identify a previous history of allergy (especially drug and latex allergy), reactions during surgical procedures, and the presence of comorbidities and risk factors for developing a hypersensitivity reaction. In the face of anaphylaxis, adequate management involves both immediate stabilization of the patient and early treatment for better patient prognosis.

Ethics approval and consent to participate

Not applicable.

Consent for publication

Written informed consent was obtained from the patient for publication of this case report and accompanying images.

Availability of data and materials

The data that support the findings of this report are available from the corresponding author on reasonable request.

Competing interests

The authors declare that they have no competing interests.

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Author contributions

All the authors reviewed the case and reviewed the literature. All authors read and approved the final manuscript.

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