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CASE REPORT

Post-General Anesthesia Angioedema After General Anesthesia

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ABSTRACT

Background: General anesthesia includes the administration of avariety medicines to patient in fairly short period. Numerous negative consequences can occur because of reaction from the drugs given, one example is anaphylaxis that may lead to death if not been recognized and handled quickly & promptly.

Case Illustration: A sixty-seven years old male patient 152 cm 65 kg with multiple cholecystitis and obstructive jaundice, who was scheduled to undergo laparoscopic cholecystectomy. Patient had no records of allergy to medication and food. Family has no records to hypersensitive reactions or angioedema. At initial evaluation, blood pressure was 190/90 mmHg and hemoglobin 10,4 g/dL while the rest parameter was within normal limits. We given propofol and rocuronium to induce relaxation and facilitate intubation in surgery. Approximately 90 minutes after rocuronium injection, patient evolved angioedema across the face area, tongue, and floor of the mouth. Patient also complained of not being able to talk. Soon after that, we give 10 mg of dexamethasone and 100 mg hydrocortisone to the patient. Within 60 minutes after hydrocortisone administration the patient shows clinical improvement that marked by swelling began to decrease and the patient was able to speak again. Then we educate patient about angioedema and its alert.

Conclusion: Anaphylaxis is a type of hypersensitivity response that take place to a particular allergen and mostly self-limited, but 11% of hypersensitive reaction require airway intervention. Most of anesthetic medication such as thiopental sodium, propofol, muscle relaxants, and etomidate may induce anaphylactic reaction during anesthesia induction. Discontinuation of underlying agent and airway control may prevent mortality and morbidity.

Keywords: Angioedema; Anesthesia; Hypersensitive

INTRODUCTION

General anesthesia includes various of medicine for patients in a fairly short period of time. Numerous negative consequences can occur because of reactions to the drugs given, one example is anaphylaxis reaction which is a severe reaction and life-threatening. Anaphylaxis is a uncommon side effect, but may lead to death if not been recognized and handled quickly and promptly. Patient characteristics of instant hypersensitive reaction not simplest consist of hemodynamic changes but additionally can be manifest with urticaria, redness, as well as angioedema, because of its inspection of a skin reaction is critical for recognize and prompt treatment. Furthermore, figuring out the causative agent is the most essential process to prevent later recurrence[1,2].

Here we documented a case of hypersensitivity reaction followed with angioedema after propofol and rocuronium was given to induce relaxation and facilitate intubation in surgery in a 67 y.o. male patient with multiple cholecystitis and obstructive jaundice, who was scheduled to undergo laparoscopic cholecystectomy, we provide potential underlying predisposing mechanism and available therapeutic approaches with a overview of the applicable literature.

CASE ILLUSTRATION

A 67 y.o. male patient, 152 cm, 65 kg with multiple cholecystitis and obstructive jaundice, was scheduled to undergo laparoscopic cholecystectomy (Figure 1). He has an underlying disease, which is hypertension with a history of taking amlodipine 5mg 1 time a day. He had no records of allergy to medication and food, and there has been no own family records of hypersensitive reactions or angioedema. At the initial evaluation of the patient in the ER, abnormal results were found in the form of blood pressure 190/90 mmHg and hemoglobin 10.4 g/dL, the rest parameter were within normal limits.

After arriving inside the operation room, the ECG, non-invasive blood pressure manometer, heart rate, end tidal CO, and pulse oximeter were monitored. The initial vital signs showed a BP, respiratory rate, pulse rate, and oxygen saturation of 116/81 mmHg, 25 per min, 70 beats per min and 100% respectively. preoxygenation with 10 lpm, 5mg of

dexamethasone, 5mg of midazolam, and for analgesia 100mcg of fentanyl were injected as preanesthetic medication. Intravenous propofol 100mg and IV rocuronium 50mg was given to induce relaxation and facilitate intubation in surgery. Endotracheal intubation was done 1 minute after rocuronium injection.

During the procedural of general anesthesia, 30 minutes after sign-in, the operation must be canceled because the surgeon was confirmed infected by COVID-19. This whole procedure was carried out according to the hospital policy. Patient was planned to be woken up. After finding the patient's breath is positif and had a sufficient tidal volume, reversal drug was injected using 1.5mg of neostigmine and patient was extubated and transferred to ward. After 1 hour later the patient reported experiencing swelling in the facial area and tongue (figure 2), lower and upper lip was abnormal. There was difficulty on swallowing and respiration. There was no associated urticaria or rash. The patient also complained of not being able to talk cause of tongue swelling. The patient was compos mentis but on auscultation we found additional breath sounds which is snoring and wheezing in both lung fields. He had a blood pressure 150/90 mmHg, pulse rate of 110 bpm, respiratory rate 30/min, and SpO_2 95%. A diagnosis of drug induced angioedema was made and the patient was given dexamethasone 10mg IV but within 30 minutes did not show any improvement. After that the patient was given hydrocortisone 100 mg IV and within 1 hour the patient experienced improvement which was marked by swelling of the face and tongue area began to decrease and the patient was able to speak again. He was continuosly monitored and the swelling completely subside by 48 hours. He was educated about angioedema and its alert.

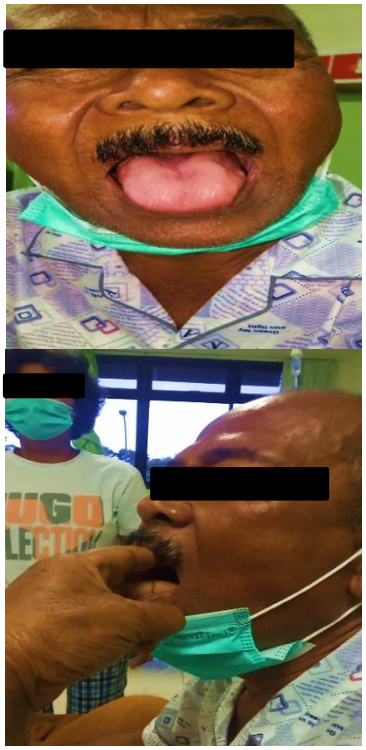

Figure 1. patient's condition before surgery

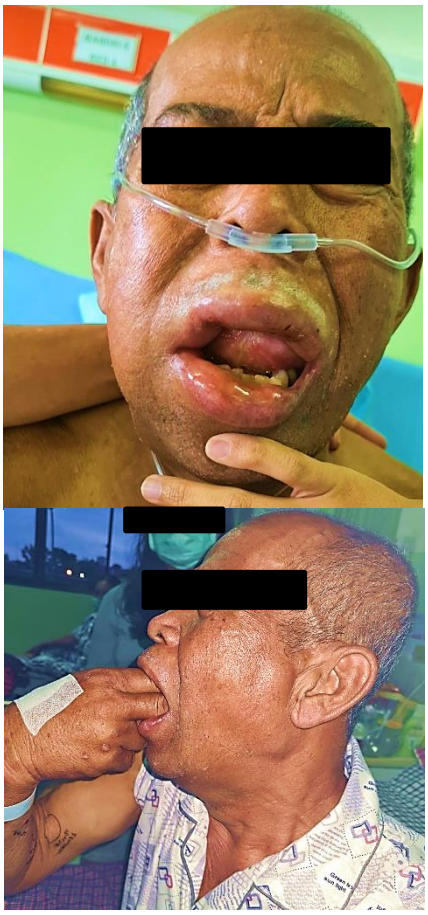

Figure 2. patient with facial angioedema; watch for swelling on facial, upper-lower lips, and tongue.

DISCUSSION

Angioedema (Quincke's edema) is an acute-onset temporary edema related to the skin, subcutan tissue, and mucous membrane of the airway structures, oral cavity, face, GI tract, or the lower and upper extremities[1,3–6]. Angioedema rarely complicates the perioperative or postoperative period. Anaphylaxis prevalence during a general anesthesia ranges from 1:4,500 - 1:20,000. Mortality rates of anaphylaxis are 3-6 % and the rates of extreme neurological complication is 2%. Most anesthetic medication that given (thiopental sodium, propofol, muscle relaxants, and etomidate) can produce anaphylactic reactions during anesthesia induction. More than 90% of intravenous drug-induced allergy responses occur within minutes after injection. Anaphylactic reactions to propofol are rare, Hepner et al reported an incidence of propofol anaphylactic reaction was 1:60.000. Meanwhile a recent survey found that the most common causative agent of anaphylaxis in perioperative period was rocuronium (58.2%), with the reported incidence of anaphylaxis approximately 1:3500 – 1:445.000[2,7,8].

Anaphylaxis is a type of hypersensitivity response that take place in response to a particular allergen. While a particular allergen is delivered to the body, IgE antibodies produce by plasma cells can bind tightly to Fc receptors at the mast cells and basophils surface throughout physiological immunologic reactions. The sensitized and degranulation of mast cells and basophils is caused by cross-linking causing various mediator released, including histamine set off numerous

physiological reaction[2,4].

Anaphylaxis additionally can be caused through secondary reactions in different cells, such as neutrophils, eosinophils, monocytes, platelets, and T lymphocytes. As well as the impacts of these substances may cause airway tightness, edema, and erythema[2,4].

Histamine binds to specific H_1, H_2, and H_3 receptors on target cells when it is released by mast cells. The location and effects of these histamine receptors differ. Histamine binding to H_1 receptors causes constriction of the smooth muscle, elevated vascular osmolality, and goblet cells mucus secretion in most allergic reactions, whereas histamine binding to H_2 receptors causes secretion of the exocrine gland. Prostaglandins and leukotrienes act more slowly than histamine, but their effects stronger and continue longer than those of histamine. Leukotriene is a potent bronchoconstrictor that also increases mucus production and vascular permeability. Furthermore prostaglandin D_2 is also a bronchoconstrictor[2,4].

The clinical manifestation that involves during hypersensitive reaction characterized by: collapse of cardiovascular systems (Tachycardia, bradycardia, or hypotension), respiratory system (hypoxia, bronchospasm, and pulmonary edema), and skin manifestation (edema or systemic lupus erythematosus). In most of cases, it stays self-limited and could simplest require monitoring and supportive theraphy. While 11% hypersensitive reaction require airway intervention to save the patient life. In such cases, morbidity and mortality can be high (30-40%)[1,2,4].

The episode of anaphylaxis reaction in this patient was recognized with the manifestation of edema on the facial area and airway resistance with no skin manifestation. Due to the fact of increased vascular permeability and vasodilatation that leads to extravasation of plasma is assumed become the contributing factors causing angioedema and facial area is the most commonly site[2,4]. The inciting occasion of inflammatory cascade main to angioedema is sometime idiopathic. But angioedema has been related to sure medicinal drug, physical trauma, allergens, and acquired or hereditary autoimmune abnormalities. thinking about that it's far not possible to make a particular analysis of the causative agent of anaphylaxis in our patient. The

causative agent of anaphylaxis in this patient has been unknown since there was no further examination such as intradermal skin test or skin prick test that are the gold standard methods to identify a potential anaphylactogen. The test commonly recommended in three weeks and no more than after three monts after anaphylactic episode. In this case, we only can presumed that the causative agent might be rocuronium, since this drug having the most common caused of anaphylactic.

Propofol is a sedative-hypnotic anesthetic drug that given intravenously and works in a similar mechanism to thiopental and may be used to preserve anesthesia with a continuous infusion. Propofol, is much more likely to trigger an allergic reaction compared to other anesthetic medicines, propofol attributed at least 2% of perioperative anaphylactic shock. Patients with medication allergies, asthma, and allergic rhinitis were the most likely to have anaphylactic reactions to propofol[2,8].

Cremophor EL, the first formulation of propofol, was related to a higher rate of vascular spasms and allergic responses. In comparison to previous intravenous anesthetic drugs, a novel propofol components using soybean oil significantly generate clinically negligible of histamine release[1,7].

The essential management of angioedema is discontinuation of the underlying agent and airway control. The swelling typically resolves spontaneously for 2-3 days. Right identification of angioedema that requiring airway intervention could lessen mortality and morbidity, and recognition of its self-restricting route can prevent needless surgical airway intervention. Chiu et al stated that, 21% of type 2 and 33% of type 3 angioedema patients requiring intubation (Table 1)[3]. In this case, our patient had characteristics of type 2 angioedema (tongue, face, and floor of the mouth) in order that maintain his airway with tracheal intubation is non-obligatory. In treating angioedema (all etiologies) intramuscular epinephrine administration, antihistaminergic, or steroids drugs should be given to prevent clinical deterioration. In our case, the patient was given dexamethasone 10mg and hydrocortisone 100mg within 90 minutes the patient showed clinical improvement. The exact mechanism of the anaphylactic occurance after the patient was given dexamethasone 5 mg is

still unknown[4,8].

Table 1. Angioedema Classification[3]

Pattern	Clinical description	Management
Type 1	Swelling is limited to face and oral cavity and excludes the floor of the mouth	May be observed on the ward
Type 2	Swelling extends to the floor of the mouth, tongue, soft palate, and/or uvula	Close observation in the intensive care unit. If severe, proceed to fiberoptic intubation.
Type 3	Swelling extends to supraglottic and glottic structures	Close observation in the intensive care unit. If severe, proceed to fiberoptic intubation.

Offering the right treatment within the occasion of anaphylaxis is vital however pretreatment to prevent exposure to its inducers is lots greater important it is almost not possible to carry out a skin test on each medicine administered in the cour se of anesthesia for each patient but, in patients with a capability threat of anaphylaxis, along with patients who've experienced an allergic reaction to a medicine, it is important to perform a skin test to selectively discover the inducing substance. When responsible substance are recognized, the management of the causative substance and cross reactioninduced materials need tobe averted[2,6,7].

CONSLUSION

In summary, there have to be a excessive index of suspicion for angioedema in a patient with tongue swelling within the postoperative length it's miles critical to understand pattern of swelling and to be acquainted with the differential diagnosis of angioedema to take care of patients with this critical complication Expedient evaluation of the airway and suitable intervention may prevent mortality and morbidity related to the late recognition of airway compromise.

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