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A Rare Case of Angioedema Following Propofol Administration in Remote Area

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Abstract: ***One life threatening manifestation of drug hypersensitivity is angioedema. In some rare cases, propofol may induced angioedema and is proposed to be allergy related. This case report presented a case of angioedema induced by propofol administration in remote area. Limited resources available added another challenge in confirming diagnosis and treating the disease. Nevertheless, identifying and discontinuing the suspected etiology should not be postponed and monitoring intensively should be initiated early to prevent lethal condition.***

Keywords: Angioedema, propofol, hypersensitivity, allergy, bradikinin

1. Introduction

Angioedema is an event of paramount importance, due to the possibility of life-threatening event followed. Mechanisms lead to angioedema formation is mainly divided into two; allergy and non-allergy. Each has a different approach in treatment, yet both need intensive observation and immediate intervention.

Propofol is one of the drug in operative setting proposed to be responsible for such event, even the number of case is extremely low. Many experts claimed propofol to be safe even for patient with history of allergy to peanut. This is a case of angioedema formation in patient with peanut allergy following propofol administration.

2. Case Report/ Case Presentation

A 47-years old female patient, 50kg, Asian ethnicity,developed angioedema following administration of propofol during a scheduled gluteal debridement. The patient was first admitted to the emergency department with diabetic ketoacidosis, diabetic type II, gluteal abscess, acute kidney injury and urinary tract infection. She had a history ofcomorbid diabetic type II under treatment with metformin (500mg/three times daily) and history of allergy to peanut, corn and dry fish. Allergic manifestation including rashes and itchiness in all parts of body. Family history of allergy was denied, and there was no personal or family history of anaphylactic reaction.There was no history of smoking, alcohol consumption, antihypertensive drugs therapyor previous surgical procedure.

Initial physical examinationshows blood pressure of 160/90 mmHg, heart rate 92 bpm, respiratory rate 28 per minute and oxygen saturation 86% on roomair and temperature. Laboratory examination result shows random blood glucose 470 mg/dL, hemoglobin 11.5 g/dl, hematocrit 23%,white blood cells 29,600/µL, platelet count 626,000/mm3, creatinine 20 mg/dL, Na 122 mmol/L, K 5.4 mmol/L, Cl 92 mmol/L. Urinalysis shows ketone4+, protein 2+ and leukocyte 1-5/power field.

After 4 days of hospitalization and treatment in ICU, the patient was transferred to internal medicine ward and then surgical ward for further treatment for the gluteal abscess. Current treatment included NaCl infusion, Ceftriaxone (2gr daily), Metronidazole (500mg three times daily), Ranitidine (50mg twice daily), rapid acting insulin (Novorapid 12-12-12 U daily), long acting insulin (Levemir 18 U daily), and potassium chloride (KSR 600mg daily). Gluteal debridement was scheduled, with no preanesthetic medication. She was admitted to the operating room with blood pressure of120/90, heart rate equal to 88 beats per minute, respiratory rate of 16 per minute, and SpO2 99% in room air and temperature. Total intravenous anesthesia (TIVA) was performed with Propofol (30 mg) and Fentanyl (50 mcg). All medical team used latex gloves during the procedure. The anesthetic-surgical procedure was uneventful with duration of 45 minutes. Since there was no recovery room in remote area hospital, the patient was then transferred back to surgical ward with planning of additional medication of ketorolac (30 mg three times daily) for two days.

Upon returning to the ward, swelling of the upper lipand periorbital edema became noticeable. The tongue and oropharynx were normal, and there were no stridor, urticaria, rash or difficulty in breathing or swallowing. No medication had been given after the patient arrived at the ward. Physical examination revealed no decrease in consciousness, blood pressure of 100/60, heart rate equal to 102 beats per minute, respiratory rate of 16 per minute and SpO2 98% in room air and temperature. Diphenhydramine (10 mg four times daily) and Methylprednisolone (62.5 mg once daily) were administered, while cancelling addition of ketorolac therapy. She was continuously monitored for increase in edema, stridor or change in haemodynamics. The swelling began to decrease after 24 hours and completely subsided by 48 hours. This reaction was first time to be occurred according to the patient. Follow-up examination

revealed no other reaction occurred,, and the patient was educated about the condition to be more aware in the future.

3. Discussion / Conclusion

The case of angioedema (AE) following drugs administration is of importance, due to the possibility of life-threatening events. Airway patency is to be monitored to ensure patient oxygenation, which in 11% of the cases there are risk of hypoxia needed for prompt intervention such as surgical tracheostomy and in turn increases mortality as high as 30-40%, morbidity and length of hospital stay.[1]

AE or Quincke's edema is an acute-onset transient edema involving the skin, subcutaneous tissues, and mucous membranes of the face, oral cavity, airway structures or gastrointestinal tract, the upper and lower extremities. The mechanisms causing this reaction are mainly divided into two; mast cell-mediated and bradykinin-mediated.[2] Different etiologic mechanisms will result in different treatment strategy.

AE from allergic reaction is mediated by antigen-IgE which release inflammatory mediators and histamine from the basophils and mast cells.[3]Effective medication included epinephrine, glucocorticoids, antihistamine and oxygen.[1,4]This patient was administered Diphenhydramine and Methylprednisolone as empiric therapy, with justification that there was possibility of deterioration of the respiratory function.

In contrast, drug-induced AE of non-allergic origin related to overproduction or decreased degradation of bradykinin. The increase of bradykinin will increase microvascular permeability, promotes tissue edema, hence induce arterial hypotension and bronchospasm. Though bradykinin has a half-life of only 15 seconds and rapidly metabolized, the edema and capillary leak may last longer. Factors linked to bradykinin-mediated AE are treatment with ACE inhibitor with the highest incidence of 25-39%, angiotensin 2 receptor antagonists, non-steroidal anti-inflammatory drugs, latex allergy, surgical stress and oropharyngeal instrumentation, including laryngoscopy. The principal therapy is discontinuation of the offending drugs and oxygenation. Bradykinin-related AE is resistant to glucocorticoids and antihistamines, instead administration of Fresh frozen plasma (FFP) has been proposed. FFP contains kinases that accelerate breakdown of bradykinin.

In remote area with limited sources, it is hard to determine the event etiology and pathophysiology of the AE. In this patient, latex allergy can be omitted, because since admission to surgical ward there was no sign or symptoms of allergy despite the nurse using latex gloves for caring of the patient.[5]Since the AE appeared after the patient had undergone TIVA and no other suspected drugs in therapy during hospitalization, propofol is proposed to be the possible drug responsible for the reaction.

Propofol is an alkylphenol derivative (2,6-diisopropylphenol).[6]New propofol formulation, using soybean oil (10%), was found to cause clinically insignificant histamine release compared with other intravenous anesthetic agent. Propofol with refined soy oil is safe for people with soy or peanut allergy because during the refining process the allergenic proteins are removed.[6]Several studies support the safety of propofol given to individuals with soybean and peanut allergy.[7]

However, in some rare cases propofol may induce an aphylactoid reactions, such as bronchospasm and wheals of the skin. As reported in France, propofol is more likely than other anesthetic drugs to cause an allergy reaction, with incidence of 2.0% of perioperative anaphylactic shock is related to propofol.[8]

Hypersensitivity reaction triggered by propofol is proposed to be an IgE-mediated reaction. Administration of propofol in patient with history of allergy to egg, soy and peanut is still a matter of concern.[5]Nevertheless, a study in Denmark showed three out of four patient with propofol hypersensitivity failed to show positive skin test.[9] This suggested non-IgM mediated mechanism underlying propofol hypersensitivity. Propofol may increase bradykinin levels in tissues, typically occurred local to the site of injection such as transient burning sensation during drug administration.[1]Factors contributing to incidence of AE related to propofol include pain, anxiety, significant physical and surgical stress, infection and temperature changes. Propofol associated AE typically develop immediately following injection, with more than 90% ot the reactions occur within 5 minutes of administration. [8]

The present case illustrates an acute AE following first exposure to propofol during hospital stay in a remote area. The reaction occurred involved swelling of the mucous of the upper lip and periorbital area. This patient developed AE approximately 60 minutes following propofol injection, which might resulted from propofol injection. Propofol, in conjunction with surgical stress may contribute to AE formation in this patient.[1]Examination that may aid in diagnosis includes tryptase test, skin prick test and intradermal test.[10]In remote area, oftentimes a final diagnosis cannot be established, and symptomatic therapy to stabilize the patient become a priority. The most important step after the initial management is to recognize the etiology and immediate discontinuation of the suspected drug.

4. Statements

Statement of Ethics
Informed patient consent was obtained for publication of the case details. Work was approved by ethics committee.

Disclosure Statement
The authors have no conflicts of interest to declare.

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