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Management of Anaphylaxis Induced by Atracurium: A Case Report

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Abstract

Neuromuscular blockers are the commonest cause of anaphylaxis in perioperative settings. The pathological process involves IgE-mediated hypersensitivity reaction with release of vasoactive inflammatory substances in response to exposure to the causative agent. Here, we present a case of atracurium-induced anaphylaxis in a patient scheduled for open cholecystectomy. The patient was treated with epinephrine. The surgery was done after a gap of 1 month post recovery. This time induction was done with vecuronium and the surgery was completed uneventfully.

Keywords: *anaphylaxis; atracurium; epinephrine; general anaesthesia; IgE hypersensitivity; muscle relaxant.*

Introduction

Anaphylaxis is a multisystem allergic reaction which couldpotentially be fatal. Anaesthesiologists should be able to recognize anaphylaxis, especially in perioperative settings, and treat it accurately. Neuromuscular blocking agents are the commonest cause of anaphylaxis in perioperative settings. The drug of choice for anaphylaxis is epinephrine, to be administered at the earliest. Adjunctive measures include securing airway, fluid resuscitation, antihistamines and steroids.

Case Report

A 45 year old female was admitted for elective open cholecystectomy in view of cholelithiasis in the surgery ward. The patient was a known hypertensive on treatment. Patient had no other comorbidity. There was no previous history of any drug allergy. Her weight was 50 kg and height was155cm. Systemic examination was within normal limits. Surgery was planned for the next morning after getting all the investigations done and was kept nil per oral (NPO) till then.

Next day in morning, patient was taken to operation theatre for open cholecystectomy. Routine monitors were attached and intravenous line with 18G cannula was secured. Preoxygenation was done for 3 minutes and induction was done with inj. propofol 100mg, inj.midazolam 2mg, inj.fentanyl 100mcg and inj. atracurium 25mg. Tracheal intubation was done successfully and patient was put on controlled mode of ventilation with airway pressures being in normal range.

Suddenly, the patient developed redness and skin rash in her right forearm which progressed to arm and neck in few minutes. There was sudden drop in blood pressure (systolic BP - 60 mm Hg) and tachycardia (HR- 144/min). Oxygen saturation dropped to 89%. Electrocardiogram showed sinus tachycardia with no ischemic changes. Rise in airway pressures to 45cm of H₂O was also seen. On auscultation, bronchospasm was detected. The capnograph showed pattern suggestive of bronchospasm. Patient was switched to manual bag ventilation and repeated boluses of inj. phenylephrine were given but no improvement was seen in blood pressure.

Based on clinical findings the diagnosis of anaphylaxis was kept. The patient was immediately administered inj. epinephrine 50 microgram bolus intravenously alongwith inj.hydrocortisone 200mg and inj.ranitidine 50mg. Metered dose inhaler (MDI)6-8 puffs were given via endotracheal tube for alleviating bronchospasm. Blood pressure was stabilized within 20 minutes of starting the treatment. Intraarterial blood pressure monitoring was started. Central line was inserted in right internal jugular vein and nor-adrenaline infusion was started at the rate of 0.05mcg/kg/min. The surgery was postponed and after stabilizing the vitals, patient was shifted to ICU with endotracheal tube in-situ for further management.

The non-adrenaline infusion was continued with target mean arterial pressure (MAP) above 65 mm Hg. Angioedema around lips was also detected later on. On day 2 of ICU, the patient's vitals were stable. The non-adrenaline infusion was gradually titrated. The patient was conscious and extubated fully awake. Oxygen supplementation was done with facemask. Nebulization with adrenaline and β_2 -agonists was done for hoarseness and laryngeal edema. Next day, after full recovery, she was shifted to general ward from ICU. She was further advised to get her skin patch test which later on came reactive for inj. atracurium.

Surgery was performed one month later under general anaesthesia. Induction was done with inj.midazolam, inj.propofol, inj.fentanyl and inj. vecuronium. No reaction was seen with these drugs. Surgery went uneventful and was completed successfully.

Discussion

Anaphylaxis term was coined by Portier and Richet in 1902.^[1] In Greek, anaphylaxis means "opposite protection" or "against protection".^[2] Anaphylaxis refers to the most severe form of hypersensitivity immediate reaction with cardiovascular, respiratory, cutaneous, and gastrointestinal manifestations due to exposure to a causative agent.^[3] The incidence of anaphylaxis reactions during anaesthesia is estimated around 1 in 3,500 to 1 in 13,000.^[4] Muscle relaxants are the most common agents responsible for anaphylaxis in perioperative period. Other agents leading to anaphylaxis include latex, antibiotics, anaesthesia induction agents, colloids and opioids.^[5]

The pathological process involves IgE-mediated hypersensitivity reaction with release of vasoactive inflammatory substances in response to exposure to the causative agent. Upon exposure to allergic agent, IgE induces activation of mast cells and basophils resulting in release of histamine and other inflammatory mediators like leukotrienes, prostaglandins and cytokines.^[6]An anaphylactoid reaction occurs by a different, non-immunogenic mechanism without the involvement of IgE.

Anaphylactic reactions occur within minutesof an exposure to the triggering agent. Some reactions are delayed and can appear even after 72 hours of exposure. Due to release of histamine and other inflammatory mediators, there is increased vascular permeability with extravasation of fluid, thereby depleting the intravascular volume leading to sudden hypotension and cardiovascular collapse. Rapid onset reactions are associated with higher mortality.^[7]

Anaphylaxis is a clinical diagnosis. The clinical manifestations can include dermatologic (pruritus, flushing, urticaria, rashes, angioedema),

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respiratory tract (dyspnea, wheezing, rhinorrhea, upper airway angioedema, bronchospasm), cardiovascular (dysrhythmias, profound hypotension, arrest), gastrointestinal (abdominal cramping, vomiting, diarrhea), and genitourinary (urgency, cramping). However, diagnosis of anaphylaxis in perioperative setting is different from anaphylaxis in ward.^[8] During anaesthesia, patient can't describe any manifestation like urticaria, itching or sense of discomfort unlike in ward setting, where patient is conscious and can describe his complaints. Therefore, vigilant monitoring is needed in perioperative setting. The haemodynamic changes associated with anaphylaxis pose an extraordinary challenge to the anaesthesiologist which is best met by quick thinking and an aggressive approach.

For the management of anaphylaxis, resuscitation must begin with airway, breathing, and circulation. Patients with confirmed or suspected anaphylaxis should be placed on a cardiac monitor with pulse oximetry, and intravenous access should be secured.

- a. Administer 100% oxygen. Angioedema or respiratory distress should prompt early consideration for securing airway
- b. Limit further exposure of any triggering agent.
- c. First line therapy for anaphylaxis is epinephrine. It blocks the release of inflammatory mediators from sensitized basophils and mast cells. Treatment is with epinephrine 0.3 to 0.5 mg (0.3 to 0.5 mL of 1:1,000; pediatric dose, 0.01 mg/kg) by intramuscular injection in lateral thigh. The dose may be repeated every 5 min as needed. Patients with significant cardiovascular shock should receive intravenous inj. epinephrine bolus of 100 mcg over 5 to 10 min followed by an infusion of 1 to 5 mcg/min, with close observation for chest pain or arrhythmias.
- d. Aggressive fluid resuscitation with normal saline 1 to 2 L (pediatric dose, 10 to 20

mL/kg) in patients with hypotension to prevent hypovolemic shock.

- e. Steroids can be given to control persistent or delayed reactions. Severe cases can be treated with methylprednisolone 1-2mg/kg IV. Mild allergic reactions can be treated with oral prednisolone 60 mg (pediatric dose,2 mg/kg). Along with it, antihistamines such as diphenhydramine (H1 blocker) and H2 blocker ranitidine 50 mg IV (pediatric dose, 0.5 mg/kg) can be given.
- f. Consider nebulisation with β_2 -agonists like albuterol and inhaled anticholinergic like ipratropium bromide to relieve bronchospasm. For refractory cases, inj.Magnesium 2 g (25 to 50 mg/kg in children) over 20 to 30 min can be considered.
- g. For anaphylactic reactions which are refractory to epinephrine in patients on β -blocker, consider use of glucagon 1 mg IV over 5 min.
- h. After stabilizing the vitals, patient can be considered for admission to the intensive care unit for observation. On complete recovery, patient should be discharged on an antihistamine and a short course of steroid.

Conclusion

Anaphylaxis can be a severe life threatening condition which is commonly seen with exposure to drugs in perioperative setting. Vigilant monitoring, early diagnosis and prompt intervention in the management of anaphylaxis is life saving.

References

- Portier MM, Richet C. De l'action anaphylactique de certains venims. C R Soc Biol. 1902;54:170-2.
- 2. Levy JH, Yegin A. Anaphylaxis: What is monitored to make a diagnosis? How is therapy monitored? Anesthesiol Clin North Am.2001;19:705-15.

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- Angela W Tang. A practical guide to anaphylaxis. Am Fam Physician. 2004;69(5):1049
- Vervloet D, Magnan A, Birnbaum J, Pradal M.Allergic emergencies seen in surgical suites. Clin Rev Allergy Immunol. 1999;17:459-67.
- 5. Lieberman P. Anaphylactic reactions during surgical and medical procedures. J Allergy Clin Immunol. 2002;110:S64-9.
- Kraft S, Kinet JP. New developments in FcepsilonRI regulation, function and inhibition. Nat Rev Immunol.2007;7:365-78.
- Liberman P, Nicklas RA, Oppenheimer J, et al. The diagnosis and management of anaphylaxis practice parameter: 2010 update. J Allergy Clin Immunol 2010;126(6):1104.
- Ebo DG, Fisher MM, Hagendorens MM, Bridts CH, Stevens WJ. Anaphylaxis during anaesthesia:diagnostic approach. Allergy. 2007;62:471-87.