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**Research Paper**

# Anaesthesia Management of a Patient with Multiple Drug Allergy for Breast Incisional Biopsy

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***Abstract***
*During the preoperative evaluation, patients frequently indicate 'multiple drug allergies', most of which have not been validated. Potential allergic cross-reactivity between drugs and foods is frequently considered as a risk factor for perioperative hypersensitivity. The aim of this review is to facilitate the recognition of risk factors for perioperative anaphylaxis and help the management of patient with 'multiple drug allergies' during the perioperative period. Patient with multiple drug hypersensitivity often pose significant challenge during surgery because of the greater risk of developing anaphylactic shock that could be fatal. The use of novel pharmaceutical agents such as anesthetic drugs and medical devices made from a wide range of chemicals and materials with unknown antigenicity potentially predisposes this subset of patient to hypersensitivity reactions in the perioperative period.*

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## I. Introduction

Surgery and anesthesia not only induce stress response, but also expose a patient to different classes of drugs, such as anxiolytics, sedative agents, analgesics, muscle relaxants, antibiotics and antiemetics. During the preoperative evaluation, patient frequently indicate 'multiple drug allergies'. Such statement may lead to a difficult perioperative management by the anaesthesiologist in charge. The concept of 'multiple drug allergy syndrome' is defined by a propensity to react against different chemically unrelated antibiotic or nonantibiotic drugs [1]. However, there is disagreement about what constitutes a true allergic reaction. Immediate hypersensitivity reactions now encompass IgE-mediated and non-IgE-mediated reactions. Allergenic cross reactivity is a property defined by individual antibodies to other allergens with structural similarity and can be seen in families of drugs or agents used during the perioperative period. The aim of this review is to provide guidance for practitioners by reviewing the clinical presentation pattern of patients with 'multiple drug allergies' and help establish evidence, or refute false beliefs, of cross-reactivity for drugs used during anesthesia to facilitate the perioperative management of these patients.

## II. Case Report

A 32-year-old female patient with a history of multiple drug allergies was diagnosed to have breast lump and was posted for incisional biopsy. History of dust allergy, allergy to drugs with wheezing and urticaria was elicited. Skin allergy test revealed sensitivity to all tested opioids expect fentanyl, muscle relaxants, local anesthethics, analgesics, anticholinergics, atropine, ephedrine, mephentaramine, etomidate, thiopentone sodium, neostigmine and ondansetron. She had less sensitive reaction to propofol. She gave a history of allergy to paracetamol also.

The patient was assessed in the preanesthesia clinic with a detailed history and relevant investigations. Intradermal allergy test results were documented. Patient was sensitive to commonly used drugs during anesthesia as described . The patient was allergic to most of the antibiotics as well.Patient was admitted on the day prior to surgery and was kept fasting for 6 h for solids and 2 h for clear fluids. No premedication was administered on night prior to surgery.

The plan of anesthesia in this patient was an anesthetic gas-based general anesthesia without using muscle relaxants but with use of intravenous fentanyl and propofol if patient was not allergic to it. In the operation theater,

all emergency drugs and equipment were kept ready for resuscitation. Adrenaline was diluted and loaded as 10 μg/mL and 100 μg/mL solutions in syringes and infusion pumps. Patient received hydrocortisone 100 mg and dexamethasone 8 mg intravenously on arrival to the operation theater. Steroids were used to reduce the risk of recurring or protracted anaphylaxis as well as for supplemental analgesic and antiemetic effects. After attaching preinduction monitors such as electrocardiogram, noninvasive blood pressure monitor, and pulse oximeter, fentanyl received 10 μg of fentanyl intravenously initially as a slow bolus. After 5 min, if there was no itching, urticaria, tachycardia, hypotension, or difficulty in breathing, the remaining fentanyl (2 μg/kg) was also given over 5 min.

Patient was informed about inhalation induction and was asked to maximally inhale and exhale through a tight-fitting face mask. Sevoflurane 8% in oxygen was used for induction with a gas flow rate of 6 L/min . When the patient lost consciousness, 50% nitrous oxide was added to anesthetic mixture and as depth of anesthesia became deeper and respiration became shallower, breaths were assisted by gentle bag ventilation synchronizing with patient effort till they became apneic. Heart rate and blood pressures were closely monitored for the development of hypotension or bradycardia. Following cessation of breathing and along with boluses of propofol igel of size 3 is inserted. There was no evidence of bronchospasm and managed by hyperventilating with sevoflurane 8% dial setting.

Anesthesia was maintained with sevoflurane , oxygen and nitrous oxide (1:2) mixture and with boluses of propofol. Patient was mechanically ventilated with a tidal volume of 6–8 ml/kg body weight and at higher respiratory rate (14–16 $min^{-1}$) so as to maintain an end-tidal carbon dioxide ($EtCO_2$) level of 25–30 mmHg. A lower $EtCO_2$ was maintained to reduce spontaneous respiratory efforts during surgery. Any attempt at breathing was managed by hyperventilating with bag and temporarily increasing sevoflurane concentration by 1%–2%. At the end of surgery, all anesthetic gases were cutoff, and patient was bag ventilated till she regained spontaneous breathing and adequate tidal volume . Igel removed and vitals stable.Postoperative analgesia was provided with fentanyl iv. Administration of dexamethasone preoperatively might have had a blunting effect on postoperative pain and emesis .

## III. Discussion

As an anesthesiologist, it is important to know about two entities namely anaphylaxis and anaphylactoid reactions. Anaphylaxis is an immune (IgE)-mediated Type-1 hypersensitivity reaction where there is always a prior exposure to the allergen, whereas anaphylactoid reaction is nonimmune mediated, without any previous exposure to allergen.[2] However, the clinical manifestations of both are similar. On exposure to allergen, there is release of the preformed mediators inside mast cells (histamine, serum tryptase, eosinophillic chemotactic factor, and neutrophillic chemotactic factor), followed by release of mediators synthesized after mast cell activation (leukotrienes, TXA2, cytokines, prostaglandins, and platelet activating factor). Anaphylaxis presents clinically with profound hypotension due to vasodilation and increased capillary permeability; there will be bronchospasm manifesting as raised peak airway pressures, urticarial lesions, and cutaneous flushing along with angioedema, and in more severe cases, laryngeal edema, cardiovascular collapse, and laryngospasm as well.[3]

The most common anesthetic agents implicated in the occurrence of intraoperative anaphylaxis are neuromuscular blocking agents (NMBAs) (70%). Among which higher chance of anaphylaxis is for succinylcholine, vecuronium (anaphylaxis), followed by atracurium (anaphylactoid). The allergy is mainly secondary to the quaternary ammonium compound and hence there is a high incidence of cross-reactivity among NMBAs.[4] The second most common allergen is latex (12.6%), followed by colloids (4.7%) (gelatin > dextran > albumin), intravenous induction agents (3.6%, thiopentone > propofol > etomidate), and antibiotics (2.6%, cephalosporins, carbapenems, and vancomycin). There have been reports of allergy to benzodiazepines and opioids (morphine and meperidine); however, reaction to synthetic opioids like fentanyl is rare. Allergy to local anesthetics (esters > amides) is less, but when it occurs, it is mainly due to the added preservative (benzoates and metabisulfite) in amide group and due to para amino benzoic acid (PABA ) moiety in ester group. Delayed Type-4 hypersensitivity is more common than Type-1 hypersensitivity with local anesthetics.[5] It is important to note that allergy to blood products, atropine, protamine, and bone cement has also been documented. Anaphylactoid reactions are most commonly due to radiocontrast agents.

It is important to recognize anaphylaxis immediately, and management involves stopping the implicated agent immediately followed by administering 100% oxygen, intravenous epinephrine 5–10 mcg boluses, or intramuscular 0.5–1 mg (1:1000 doses). Other supportive measures include adequate intravenous crystalloids up to 2–4 L for adults, antihistamines (chlorpheniramine 10–20 mg), corticosteroids (hydrocortisone 100–500 mg), and bronchodilators (salbutamol, albuterol). Sometimes, continuous infusion of epinephrine will be required in the postoperative period and vasopressin can be used in cases of refractory hypotension.

Proper documentation of anaphylaxis is important, and further evaluation for causative allergen by *in vitro* methods (RAST, serum tryptase, and serum IgE levels) and *in vivo* methods (skin prick/intradermal skin tests/provocation or challenge tests) has to be done.[4] Serum tryptase is used as a measure to differentiate

anaphylaxis and anaphylactoid reactions when levels < 15 ng/mL (anaphylactoid/mild anaphylaxis), and when levels >20 ng/mL, anaphylaxis is confirmed. Ideally, three samples should be taken, first at the onset of reaction, then 1–2 h after reaction, and the third value 6–24 h after the initial reaction. Ratio of tryptase during reaction to baseline tryptase >2 is suggestive of anaphylaxis (TDR/BT >2).

Anaphylaxis to volatile agents has not been reported so far. However, there have been reports of immune-mediated (IgG) hepatic toxicity toward a trifluoroacetyl metabolite of halothane. Prior administration of halothane increases incidence and severity of hepatitis. Enflurane and isoflurane have also been associated with immune-mediated hepatic injury without prior exposure to halothane. Therefore, sevoflurane as it is not metabolized to trifluoroacetyl metabolites and will not cause immune-mediated hepatitis. Moreover, it has a pleasant smell and is associated with a smooth, rapid induction, and recovery following discontinuation of anesthesia. It has been also noted that maintenance of anesthesia with sevoflurane resulted in less patient movement and more favorable hemodynamic responses intraoperatively.

It had been shown that sevoflurane induction can provide good intubating conditions without use of muscle relaxants. Inhalation induction with 6%–7% sevoflurane in 66% nitrous oxide and 28% oxygen by face mask, the time for achieving acceptable tracheal intubating conditions following manual hyperventilation by mask was 4.7 min (3.7–5.7) in adult patients. Addition of nitrous oxide to sevoflurane has the advantage of deepening the plane of anesthesia which will provide more favorable intubating condition in the absence of relaxants. However, the margin of safety may be reduced in an apneic patient if encountered with a difficult airway.. Although emergence delirium and agitation were documented to be more following sevoflurane anesthesia.

## IV. Conclusion

Anaphylaxis is a life-threatening complication that should be anticipated in a patient with multiple drug allergies, such patients have to be evaluated for causative allergens prior to surgery and anesthesia. In these patients, inhalational agent-based general anesthesia can be considered as a safe alternative to regular anesthetic practice involving polypharmacy, with a reduced risk of perioperative adverse events.

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