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A Case of Suspected Anaphylactic Reaction to Propofol Leading to Difficult Intubation

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Abstract: ***This article highlights the significance of recognizing and managing perioperative anaphylaxis, with a particular focus on a case involving suspected anaphylactic reaction to propofol during anaesthesia induction. While such occurrences are relatively rare, they can lead to life-threatening situations, underscoring the importance of prompt and effective response strategies. The discussion delves into the immunological mechanisms behind allergic and non-allergic anaphylaxis, shedding light on the role of propofol as a potential allergen. The incidence, diagnostic challenges, and treatment approaches for propofol-induced anaphylaxis are thoroughly explored. This case emphasizes the need for vigilant preoperative assessment, preparedness, and the pursuit of allergologic investigations to pinpoint causative agents and mitigate future occurrences.***

Keywords: perioperative anaphylaxis, propofol, allergic reactions, anesthesia induction, immunological mechanisms, treatment strategies

1. Introduction

A substantial contributor to anaesthesia related morbidity and mortality is perioperative anaphylaxis. Although the actual prevalence is unclear and most likely underreported, it is thought to be a rare occurrence. We describe a case of suspected anaphylaxis brought on by propofol following induction. Allergic reactions to propofol are uncommon and account for less than 2% of all reactions to general anaesthetics.

2. Case Report

A 45-year-old Female, vegetarian by diet, came with complaints of lower back pain for 1 month radiating to bilateral lower limbs, diagnosed as IVDP L3- L4 and L4-L5. She was planned for Discectomy and fusion under General anaesthesia. All Pre-Operative investigations were within normal limits except for raised blood sugars. Case was accepted under ASA III. Intraoperatively all standard ASA monitors were connected. Patient was preoxygenated with 100% Fio2 for 3 minutes. Patient was premedicated with Inj. MIDAZOLAM 1mg,Inj GLYCOPYRROLATE 0.2mg. Induced with Inj. PROPOFOL 100mg IV and relaxed with Inj. VECURONIUM.First attempt at intubation done with 7.0 mm sized cuffed ET Tube. Intubation attempt unsuccessful due to non-visibility of vocal cords. Cormack Lahane grade IV noted. Patient was oxygenated with 100% FiO2 with mask, vitals remained stable. Second attempt at intubation done with 6.5mm sized cuffed ET Tube, intubation unsuccessful due to non-visibility of cords. Laryngeal edema was noted along with swelling of lips.

Patient was again taken on mask ventilation. Chest rise was inadequate. Mask repositioned, tight seal maintained. Head tilt, Chin lift, jaw thrust ensured.

Chest rise still inadequate. Drop in Oxygen saturation was noted from 98% to 88%. A drop in mean blood pressure was noted from 70mmHg to 56mmHg. Not able to ventilate, Not able to intubate situation noted. Laryngeal edema due to anaphylaxis was suspected. Inj HYDROCORTISONE 100mg IV, Inj DEXAMETHASONE 8mg IV ,Inj EPHIDRINE 6mg IV, Inj ADRENALINE 0.5mg IM stat given. Mask ventilation was continued, slight chest rise noted. SpO2 increase to 95% with 100% O2. Third attempt at laryngoscopy was done, vocal cords were visible, and patient was intubated with 6.0mm sized Cuffed ET Tube. Adrenaline soaked ribbon gauze was placed as a throat pack to relieve the edema. Patient was connected to mechanical ventilator and vitals were noted to be stable. Intra operative vitals were stable and was uneventful. Fibreoptic bronchoscopy was done prior to extubation to check for any laryngeal edema. Mild edema noted. Ultrasound of Vocal cords done to assess for any edema. Mild edema noted. Prior to extubation, Inj HYDROCORTISONE 100mg IV stat given. ET Tube cuff was deflated, cuff leak was observed. Extubation was planned, with all equipment ready for emergency reintubation. Inj. MYOPYROLLATE IV given, and was extubated after patient was spontaneously breathing and was fully conscious. Vitals were stable. Patient shifted to ICU for monitoring. Patient was advised for post operative investigations such as ESR , CRP, IgE antibody levels, tryptase enzyme. ESR and CRP was found to be elevated post procedure. IgE antibody was 12kIU/L. Arterial blood gas analysis showed pH 7.404, pCO_2 42.5 mmHg, pO_2 119.6 mmHg, HCO_3- 26 mEq/L, and SaO_2 98.3%

3. Discussion

Anaphylaxis may be allergic or nonallergic. Specific immunologic mechanisms—IgE or non-IgE mediated (IgG and immune complex complement related)—are involved in allergy reactions. Nonallergic anaphylaxis, on the other hand, takes place without a particular immunological reaction. Several alternative methods, including the direct activation of mast cells and the activation of complement systems without the generation of anaphylatoxins, may be involved.

Propofol has sedative and hypnotic effects via activating the GABA (gamma amino butyric acid) receptor chloride ion

molecule. Using soybean oil (10%) and egg lecithin (1.2%) as emulsifiers, propofol is an alkylphenol derivative (2,6 di-isopropyl phenol). Lecithin is a pure phosphatide that is present in egg yolks, but it is not the allergic trigger because people who are allergic to eggs usually develop sensitization and respond to the proteins in the egg white.The refined soybean oil used to make propofol removes the allergenic proteins from the finished product. In many cases, reexposure to the phenol or isopropyl group, rather than the lipid vehicle, is what causes an allergic reaction to propofol.

Propofol was suspected by the study to be the cause of this anaphylactic incident. The medical literature has documented a small number of instances of anaphylaxis occurring after propofol. Although reports of IgE-mediated reactions (often urticaria, but occasionally even anaphylactic reactions) have been made, non-IgE mediated reactions are more frequent. The isopropyl groups, which may function as epitopes and are present in a variety of drugs and cosmetics, are typically to blame for allergic reactions after initial contact. The phenol molecule is typically the source of allergic responses after re-exposure. The raised levels of IgE indicate that anaphylaxis is the cause in the above case. The incidence of anaphylaxis of propofol is higher than the incidence of the other drugs used in the above case. The incidence of anaphylactic reactions propofol has been reported as 1:60,000. With a mortality rate ranging from 3.5% to 4.7%, these responses are uncommon but can nonetheless be fatal even when properly treated.

As soon as a severe anaphylactic reaction is identified, appropriate action should be taken, including halting the anaphylactogen immediately and administering fluid replacement. Epinephrine, dopamine, and hydrocortisone should all be given at the same time. Important techniques include keeping the airway open, giving 100% oxygen, tracheal intubation and mechanical ventilation. The use of hydrocortisone 200 mg IV, adrenaline 0.5mg (0.5 ml of 1:10,000 solutions), and other vasopressors may be considered if the blood pressure does not improve.

4. Conclusion

In conclusion, careful examination of a history of allergy may help to prevent anaphylaxis brought on by propofol. Other intravenous anaesthetic drugs are to be taken into consideration in patients with a history of hypersensitivity.Propofol anaphylaxis calls for the immediate administration of steroids, antihistamines, and airway protection. Epinephrine and rapid expansion of intravascular volume should be administered even if hemodynamic changes are visible or bronchial spasms occur. Above all, these medications need to be easily accessible. Before extubating the trachea, the airway must be assessed since severe oropharyngeal edoema is possible, as it was in this instance.

The anesthesiologist must be ready to not only handle the immediate situation with anaphylaxis in the operating room environment but also to start all the necessary procedures to diagnose the cause of unexpected anaphylaxis. Since there are no preventative therapeutic strategies, the effective anticipation, prevention, and treatment of these reactions are largely dependent on the knowledge and awareness of the attending clinicians as well as subsequent allergologic investigations to identify the offending agent and prevent recurrence.

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