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Case Report

Anaphylaxis to Inj. Glycopyrrolate!!- Three Cases Reported.

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ABSTRACT

Anaphylaxis is generally an unanticipated severe allergic reaction, often explosive in onset that cans occurperioperatively, especially during a surgical procedure when multiple drugs are administered during the conduction of anaesthesia[1]. The survey for possible etiological causes, its differentiation from adverse drug reaction and management must be immediate, because anaphylaxis is life threatening and may produce cardiovascular collapse.

INTRODUCTION:

The term "anaphylaxis" was coined by Nobel prize recipients Portier and Richet in 1902[1]. The incidence of anaphylactic reaction during anaesthesia has been reported as 1: 6000 to 1: 20000 anaesthetics. The various agents implicated are muscle relaxants (61.6%), latex (16.6%), antibiotics (8.3%), hypnotics (5.1%), colloids (3.1%), opioids (2.7%) and others (2.6%)(aprotinin, ethylene oxide, local anaesthetic)[2].

Case report: We report cases developing anaphylaxis after premedication for surgical procedures.First case was a 40 yr female patient posted for mastoidectomy for right ear CSOM, second case was a 10 yr old male child for adenoidectomy and third case was a 25 yr old male posted for deviated nasal septum correction. All these patients were ASA I without any comorbidity. There was no past history of drug allergies. Systemic examination revealed normal cardiovascular and respiratory systems. Airway examination and blood investigations were within normal limits. The first and third cases were planned under local anaesthesia with sedation while the second one was planned under general anaesthesia with endotracheal intubation. All these cases occurred over narrow span of time of two days, the first two cases on day one and the third case on day two. These cases were done in different operation theatres by different anaesthesiologists using same company and batch of inj. Glycopyrrolate as the premedication drug. After confirming the NBM status and written informed consent all the patients were taken into the operation theatre. Standard monitoring including PulseOximetry, Electrocardiogram, NIBP were used for all the cases.Patients' vital signs were stable before anaesthesia. First case the 40 yr female,on arrival in the operation theatre, had non-invasive blood pressure of 120/84 mmHg, her heart rate was 58/min and SpO2 100% on room air. Shewas given inj. Glycopyrrolate 0.2mg, inj. Ondansetron 4 mg and inj. Ranitidine 50 mg IV slowly.Immediatelyfollowing premedication she complained of left sided chest pain, restlessness and difficulty in breathing. Cardioscope revealed heart rate of 180/min with ventricular tachycardia and ST segment depression. Also the blood pressure recorded was 86/60mmHg. 100% oxygen was instituited with the help of oxygen mask. Treatment in the form of inj. Lidocaine (xylocard) 50 mg IV was given and intravenous crystalloids were infused.Not only was there symptomatic improvement but stabilisation of heart rate to 100/min and blood pressure to 100/68mmHg. She was observed in ICU for 24 hours and her post event ECG revealed no abnormality.

Second patient a 10 years old male child, 35 kg weight, resident of Sangli,diagnosed to have symptomatic adenoid enlargement was posted for elective adenoidectomy. Patient had history of recurrent upper respiratory tract infection, also diagnosed to have hyper-reactive airway disease and had undergone diagnostic nasal endoscopy with antral puncture 2 months back under general anaesthesia under antibiotic cover with no post-op complications. On arrival in the operation theatre, his non-invasive blood pressure was 110/78 mmHg, heart rate was 90/min and SpO2 100% on room

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air were recorded. IV line was secured with 22G and IV fluid in the form of Ringer's lactate started. For pre-medication inj. Glycopyrrolate 0.004 mg/kg, inj. Pentazocine 0.2 mg/kg and inj. Midazolam 0.02 mg/kg were used. Then, 5 mg/kg inj. Thiopentone was administered and 1.5 mg/kg succinylcholine was used as the muscle relaxant. Trachea was intubated with cuffed endotracheal tube no. 6.0. Cuff inflated. Air entry checked and found bilaterally equal and clear. Anaesthesia maintained on O_2 + N_2O+ Sevoflurane. Vitals being HR-160/min, BP- 100/62mmHg, SpO_2 -99%. Chest was clear. During the time positive‑pressure ventilation was being given, there was a sudden progressive drop in saturation from 99—98—94—88—82—76—70%, associated with HR-120/min, BP-70/50mmHg. Also there was evidence of B/L coarse crepitations all over the chest. Immediately patient was ventilated with 100% O_2, inj. Hydrocortisone 2mg/kg IV injected, vitals improved (SpO_2-90%, HR-108/min and BP-90/60mmHg). Suspecting light plane of anaesthesia long acting muscle relaxant inj. Vecuronium 0.1mg/kg was given. Also, inj. Aminophylline 25 mg was injected slowly. Vitals being SpO_2-94%, HR-100/min, BP-90/60mmHg along with fine crepitations over lung fields (Lt>Rt). In view of the event it was decided to postpone the surgery and reverse the patient from anaesthesia. Accordingly, when respiratory attempts were positive anaesthesia was reversed with inj. Neostigmine 0.06mg/kg and inj. Glycopyrrolate 0.008mg/kg slowly. But it was followed by frothy secretions through the endotracheal tube, coarse crepitations over B/L lung fields as well as worsening of vitals (SpO_2-88-90%, HR-116/min, BP-80/60mmHg). Because there was worsening of respiratory status and the blood pressure was not responding, paediatric physician reference was done, provisional diagnosis of anaphylactic drugreaction was made and resuscitation was started accordingly. Treatment was started with 1mg adrenaline intramuscular (1 in 1000) followed by 1mg IM after five minutes and 0.5mg subcutaneous dose along with inj. Chlorpheniramine maleate 10 mg and inj. Hydrocortisone 2mg/kg IV. Supportive measures in the form of Trendelenberg position and intravenous crystalloids were given. As he was not tolerating the endotracheal tube and in view of pulmonary edema we decided to electively ventilate him, patient was sedated with inj. Midazolam 0.5mg. But after about 15 minutes, the patient's condition started improving and he became hemodynamically stable. The patient was mechanically ventilated till the blood pressure stabilized to 100/60 mmHg and pulse rate to 100/min and decrease in crepitations. There was no evidence of bronchospasm and peak inspiratory pressure remained within normal limits. When the patient was fully awake and there was sufficient leak around the deflated cuff, the trachea was extubated. The patient was kept under observation for 24 hrs in paediatric ICU and discharged home after 48 hrs.

In the third case posted on day 2, the patient had pre-operative HR-70/min, BP-116/76mmHg, SpO_2-100% on room air. He received only inj. Glycopyrrolate 0.2mg IV as premedication. As in the first case, patient immediately complained of left sided chest pain, difficulty in breathing and restlessness.Cardioscope revealed heart rate of 190-210/min with ventricular tachycardia. He was also given inj. Lidocaine (xylocard) 50 mg IV was given and intravenous crystalloids were infused. His chest pain was relieved and heart rate settled to 110/min but breathlessness and restlessness persisted. On examination BP was recorded to be 80/56mmHg and there were B/L basal coarse crepitations. Similar to second case there was worsening of respiratory status and the blood pressure was not responding, hence provisional diagnosis of anaphylactic drug reaction was made and resuscitation was started. Treatment was started with 0.5mg subcutaneous adrenaline (1 in 1000) along with inj. Chlorpheniramine maleate 10 mg and inj. Hydrocortisone 2mg/kg IV. Supportive measures in the form of Trendelenberg position and intravenous crystalloids were given. Vitals stabilised in an hour and patient was shifted to ICU for 24 hrs observation and even his post event ECG revealed no abnormality.

Discussion: Anaphylaxis is an acute life‑threatening systemic reaction that requires quick diagnosis and correct management to save the patient. Although it is a rare intraoperative complication, most drugs used in the perioperative period can lead to anaphylaxis[3]. Adverse drug reactions or side effects are usually expected, are dose dependent, and occur at therapeutic doses. Anaphylactic and anaphylactoid reactions are unexpected and dose independent and can occur at the first exposure to drugs used during anaesthesia[1]. Ninety percent of the anaphylactic reactions occur at the time of induction of anaesthesia.[3]

The risk factor which increases the severity of reaction are preoperative ß blocker administration, history of asthma.[2] The rate of mortality ranges between 3 and 9%.[3]A survey of anaphylaxis during anaesthesia demonstrated that cardiovascular symptoms (73.6%), cutaneous symptoms (69.6%) and bronchospasm (44.2%) were the most common clinical features.[1] Accordingly all our patients developed bronchospasm, cardiovascular symptoms and signs but none had cutaneous symptoms.Management must be immediate, because

anaphylaxis is life threatening and may produce cardiovascular collapse.[1]The management of anaphylaxis consists of withdrawing the offending drug, interrupting the effects of the preformed mediators that were released in response to the antigen, and preventing more mediator release. The guidelines for treatment of anaphylaxis are now well established and epinephrine and fluid resuscitation remain the mainstay of the management.[2]Epinephrine is the drug of choice because its alpha 1 effects help to support the blood pressure while its alpha 2 effects provide bronchial smooth-muscle relaxation. Epinephrine isused at 5- to 10 ug IV bolus (0.2 ug/kg) doses for hypotension and at 0.1- to 0.5-mg IV doses in the presence of cardiovascular collapse and in paediatric age group 0.01mg/kg upto 0.5mg IM . Failure to recognize anaphylaxis and treat it promptly with epinephrine may result in biphasic or protracted anaphylaxis or in a fatal outcome. Airway support with 100% oxygen will increase oxygen delivery and compensate for the increased oxygen consumption. IV crystalloid (2–4 L) replacement will compensate for the peripheral vasodilation that often accompanies anaphylaxis. Histamine 1 blockers (e.g., diphenhydramine 0.5–1 mg/kg), histamine 2 blockers (e.g., ranitidine 150 mg or cimetidine 400-mg IV bolus), bronchodilators (e.g., albuterol and ipratropium bromide nebulizers), and corticosteroids (e.g., hydrocortisone 1–5 mg/kg) should be given.[1] For prevention a careful history regarding adverse drug reactions and allergies should be conducted before any surgical procedures requiring anaesthesia. Identification of atrisk patients will lead to avoidance of a particulardrug and is likely to prevent anaphylaxis. Health-care workers and patients with multiple prior surgical procedures can be sensitized to latex and may develop anaphylaxis when exposed to latex.[1] Females aremore likely than males to have anaphylaxis during anaesthesia, with a 3:1 ratio.[4] Avoidance of drugs that produced anaphylaxis and positive tests during a prior anesthetic has been demonstrated to prevent an episode of anaphylaxis from recurring.[1]

Conclusion: Although anaphylaxis is a rare intraoperative event, most drugs used in the perioperative period can lead to anaphylaxis. Unfortunately, documentation of anaphylaxis is often lacking because the cause and effect relationship is often hard to prove and because the diagnosis is not easy to make with the patient under anaesthesia.[1]

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