

Severe Anaphylactic Reaction at induction of Anaesthesia

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Abstract

Anaphylaxis is an IgE mediated severe allergic reaction causing release of vasoactive substances from mast cells and basophils after re-exposure to an antigen. Signs and symptoms include flushing, urticaria, hypotension, tachycardia, bronchospasm, cardio-respiratory arrest etc. It can occur at induction of anaesthesia when multiple drugs are being administered, but prompt diagnosis with correct management is the key to a successful outcome. This case report describes a patient who developed severe bronchospasm with difficulty in inflating the lungs and dropping oxygen saturations, alongwith hypotension, tachycardia and widespread flushing, at induction of anaesthesia for elective breast surgery. She was promptly managed and her hypotension was corrected, but the

bronchospasm was more resistant to treatment. The patient also developed ST segment elevation, which was successfully managed with intravenous glyceryltrinitrate. The bronchospasm responded slowly to salbutamol and aminophylline. The patient underwent surgery and was discharged home on the third postoperative day.

Introduction

Anaphylaxis during general anaesthesia is a rare but dramatic event that can be frightening to deal with because of its rapid onset and severity, and it may lead to death even when appropriately treated.¹⁻³ Anaphylaxis is caused by an IgE mediated reaction with release of vasoactive substances from mast cells and basophils after re-exposure to an antigen to which there has been previous sensitization.⁴ Occasionally anaphylaxis can occur on first exposure because of cross-

reactivity among many commercial products and drugs.⁵ In this article we report a patient who developed severe anaphylactic reaction at induction of anaesthesia, but managed successfully and underwent surgery uneventfully.

Case Report

A 53 years old female with no known co-morbidities was scheduled to undergo wide local excision of right breast lump for carcinoma of the breast. At preoperative assessment she was stable with normal systemic examination. Her laboratory investigations were normal and her electrocardiogram (ECG) and chest radiograph were within normal limits. She had no history of previous exposure to anaesthesia.

General anaesthesia was planned and she was premedicated with tablet midazolam 7.5mg. In the operating room ECG, non-invasive blood pressure (BP), pulse oximeter and capnograph were applied. She had a heart rate of 71 beats per minute, BP of 132/79 mmHg and oxygen saturation (SpO₂) of 98%. An infusion of Ringer's lactate solution was started. Pre-oxygenation anaesthesia was induced with slow intravenous (IV) pethidine 50mg and sodium thiopentone 350mg. After ensuring manual ventilation of the lungs with facemask, 35mg of atracurium was given. Soon afterwards manual ventilation of the lungs became difficult with no visible chest movement and the patient became flushed all over. The pulse was weak and on auscultation the chest was silent. SpO₂ dropped to 70% and capnogram became unrecordable. She developed tachycardia of 120 beats/minute.

A call for help was given and the patient was intubated and manually ventilated with 100% oxygen. Her blood pressure dropped to 74/50 mmHg, but responded quickly to rapid IV infusion of Ringer's lactate solution. Once the BP improved, volatile anaesthetic agent halothane was started to relieve the bronchospasm. SpO₂ improved slowly to 92% and an up-going tracing of capnogram began to appear, but widespread bronchospasm was still present with high airway pressures. Epinephrine was prepared in a strength of 1:10000 while halothane was replaced with isoflurane, as a combination of halothane and epinephrine can lead to serious arrhythmias. Before epinephrine was administered patient developed increasing ST elevation. Epinephrine was not advisable in this situation and glyceryltrinitrate (GTN) was administered in aliquots of 100 micrograms and nebulization with salbutamol 5mg was started. Oxygen saturation improved to 97-98%, and ST elevation resolved after two doses of GTN. Hydrocortisone 100mg and chlorpheniramine 10mg were administered to further stabilize the condition.

An arterial line was placed and blood sample was

collected for Troponin I levels and cardiology consultation was sought. A 12-lead ECG did not reveal any acute ischaemic changes and a portable echocardiogram showed an ejection fraction of 55-60%, with no wall motion abnormality. Arterial blood gases were normal. Since the patient had needle localization performed in radiology department and had traveled from another city to undergo this surgery, the relatives requested to proceed with the surgery. After discussion amongst the anaesthesiologist, surgeon, and cardiologist, it was decided to go ahead with the surgery. As the reaction had followed atracurium administration, no neuromuscular blocking agent was used to avoid the risk of cross-reactivity. Surgery was carried out with midazolam boluses of 1mg and fentanyl boluses of 50 microgram, as required. Patient was ventilated with oxygen 50% in air and isoflurane 1-2%. As some residual bronchospasm was still present, injection aminophylline 250mg was given stat followed by infusion of 0.5mg kg⁻¹ hour⁻¹ and nebulization with salbutamol 5mg was repeated. SpO₂ improved to 100%.

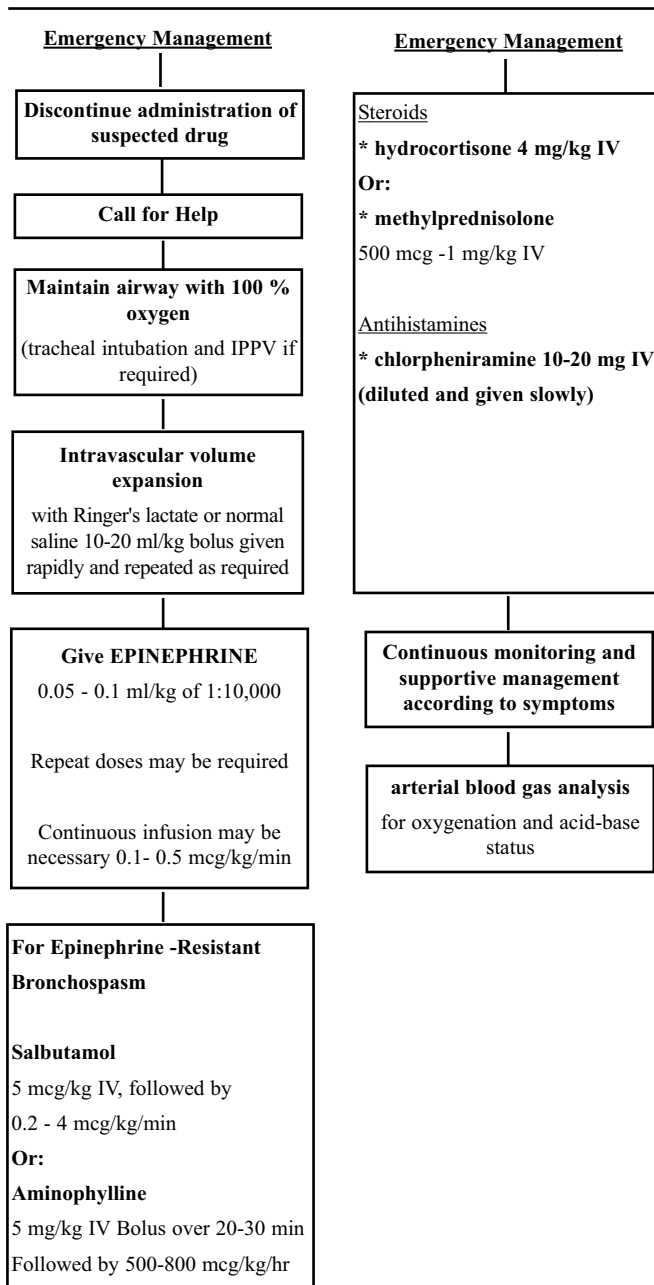
The surgery was uneventful and patient woke up with good cardiorespiratory stability. She was extubated smoothly and shifted to recovery room where she was kept overnight due to unavailability of bed in ICU. Her chest x-ray did not reveal acute changes and her bronchospasm resolved completely within six hours with regular salbutamol nebulization. Her troponin I levels were normal. The patient and her family were given written information and warned about future precautions and she was referred to a cardiologist for work-up of possible underlying coronary artery disease. She was discharged home on the third postoperative day.

Discussion

Anaphylactic reactions can range from mild reactions to severe anaphylactic shock and death.⁶ Signs and symptoms include flushing, urticaria, hypotension, tachycardia, difficulty in inflating the lungs, bronchospasm, cardio-respiratory arrest etc.⁷ The manifestations include any combination of these symptoms.⁴ Neuromuscular blocking agents, latex and antibiotics represent the most frequently involved substances in the perioperative period.^{7,8} The predominant symptom in our patient was severe bronchospasm occurring at induction along with hypotension and tachycardia, within seconds of administering atracurium. It could be argued that this occurred due to aspiration of gastric contents, but marked hypotension and widespread flushing along with bronchospasm goes strongly in favour of anaphylaxis.

The immediate and secondary management of acute anaphylactic reactions is given in Table 1.^{6,7-9,10} ECG, blood

Table 1. Recommended management of acute anaphylaxis during anaesthesia; doses adapted from GOSH / Thames PAC, Drs Howard R and Walker I. November 2004⁹.



IPPV: Intermittent positive pressure ventilation

pressure, SpO₂ and end tidal CO₂ should be monitored throughout.¹ Epinephrine is the most useful drug as it is effective in both bronchospasm and cardiovascular collapse.¹⁰ If the bronchospasm is epinephrine resistant, intravenous salbutamol 5mcg kg⁻¹ followed by 0.2-4 mcg kg⁻¹ min⁻¹ or aminophylline 5mg kg⁻¹ over 20 minutes followed by 500-800 mcg kg⁻¹ hour⁻¹ should be considered.^{5,10} Salbutamol 5 mg could be nebulized and the

dose repeated as required. If cardiovascular collapse is epinephrine-resistant, other vasopressors like phenylephrine, ephedrine or vasopressin might prove to be life-saving.^{1,5} Anaphylactic reactions may take several hours to resolve and the patient must be closely observed and managed symptomatically until stable.⁶ Antihistamines and corticosteroids are usually administered after the acute phase.^{7,9}

The authors followed the conventional recommendations for management of anaphylactic reactions (Table 1), but before epinephrine could be administered, ECG monitor showed marked ST elevation, preventing us from using epinephrine. The most likely cause of ST elevation was the significant hypoxia, alongwith hypotension and tachycardia and possible underlying coronary artery disease. Fortunately, ST elevation reverted with GTN and bronchospasm responded to salbutamol nebulization and aminophylline infusion

Once the patient has stabilized, a decision is required whether to continue with or postpone the surgery. This should be a joint decision of the surgical-anaesthesia team. As our patient was undergoing an elective procedure and had some ongoing bronchospasm, a postponement of surgery was discussed with the surgeon and relatives. Since the patient had traveled from another town and had localization needles in place, the relatives requested for proceeding with surgery. .

Ideally the patient should have been sent to the intensive care unit (ICU) for postoperative recovery, but as no ICU bed was available, she was kept overnight in the recovery room. Every patient with a suspected anaphylactic reaction during anaesthesia should be investigated postoperatively with tryptase levels, skin prick tests, radioimmunoassays etc., to identify the responsible drug^{5,7}, because subsequent re-exposure may be disastrous.⁴ Most of these tests are currently unavailable in Pakistan. The patient should be provided with written instructions and if further anaesthesia is required, the use of all suspected precipitating agents must be avoided.

Conclusion

Anaphylaxis during general anaesthesia is a dramatic event that can lead to serious consequences. Prompt diagnosis and correct management is the key to a successful outcome in severe reactions.

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