Suxamethonium Anaphylaxis

A Case Study

This case study is a detailed reflection of my experience of an anaphylactic reaction to suxamethonium, I was involved with during my training.

It all started on a Saturday night at approximately 2030hrs, when the on-call team for theatre was called to perform a laparoscopy +/- laparotomy for a potential post-operative bleed following a vaginal hysterectomy. I was part of this on-call team and practicing as the anaesthetic nurse. I am a registered nurse, with 4 years theatre experience and I had only just begun my training as an anaesthetic technician at this time. I therefore, did not have the experience to practice as the technician in this acute situation. I was however, watching and learning with great intent, to the qualified technician’s role and skills.

It all appeared to be a routine callout, with no major events anticipated. The patient was a normally fit and healthy middle aged woman, whom had undergone a vaginal hysterectomy under a spinal anaesthetic three days earlier. We prepared all our required equipment for the case, which included the following in regards to the anaesthetic management. A level 3 machine check was completed, laryngoscopes checked and a size 7.5 endotracheal tube was checked and had a syringe attached ready for a rapid sequence induction. Further equipment of a mask, magills, guedal airways, ambu bag, LMA, ET tubes, suction, stylettes, bougie and suxamethonium were all checked for availability before the patient was brought to theatre. A warm bag of Hartmann’s fluid was primed through a giving set, and a rapid infuser was available to use if required. On discussion with the anaesthetic registrar regarding the anaesthetic plan, it was noted that she had difficult intravenous access, due to probable hypovolemia, from the suspected intra-abdominal bleeding, and also from multiple attempts in the emergency department. She had a 20gauge cannula insitu in her right hand, which was patent and able to be used for induction. It was planned to insert a central line after induction.

Rapid sequence inductions are employed to reduce the risk of aspiration for acute patients whom are not classified to be nil by mouth. Delayed gastric emptying occurs as a result of trauma and opioid administration that follows. It is a technique that “contravenes one of the fundamental rules of anaesthesia, namely that muscle relaxants are not given until control of the airway is assured” (Aitkenhead & Smith, 1996, p.524). The risk of aspiration must be considered to be greater than that of losing the airway if rapid sequence induction is used. Adequate pre-assessment of the airway and degree of difficulty for intubation is crucial, and additional staff must be at hand during intubation to assist. Cricoid pressure is used to “compress the oesophagus between the thyroid cartilage and the vertebral column” (Aitkenhead & Smith, 1996, p. 524), as the cricoid cartilage

K Hazlett Trainee Anaesthetic Technician
is a complete ring. Ensuring the correct identification of the cricoid cartilage is crucial, as compression of the thyroid cartilage can distort laryngeal anatomy and make intubation more difficult. Cricoid pressure is not released until the endotracheal tube is confirmed to be in the correct place and the cuff is inflated to create a seal, preventing aspiration from occurring.

Suxamethonium is a depolarizing muscle relaxant used for rapid sequence inductions. It has a rapid onset of within 30 seconds, and a short duration of approximately 10 minutes. These features are beneficial for rapid intubation and securing the airway as quickly as possible. The short duration of action provides us with a 'get out of jail card', if intubation is difficult, as the patient is able to regain their muscle tone and return to spontaneously breathing after only 10 minutes, with the support of bag-mask ventilation until such time. This is in contrast to non-depolarising muscle relaxants which have a duration of approximately 30 minutes, meaning we would have to ventilate for this long amount of time before the patient is able to support their own breathing again. Suxamethonium “is similar to acetylcholine and acts as an acetylcholine agonist on nicotinic receptors” (Galbraith, Bullock & Manias, 2001, p.245). It works by temporarily binding to the receptor sites within the muscle fibers and generates an action potential, causing the muscle to contract. We are able to visualize this contraction as muscle fasciculation’s. Suxamethonium is not destroyed by acetylcholinesterase, unlike acetylcholine, therefore its action is sustained. Repolarisation is prevented and consequent muscle paralysis occurs. Its short duration is due to the enzyme pseudocholinesterase which breaks it down and terminates its action at the receptor site, allowing repolarisation to occur and the muscles to contract again. Adverse effects of suxamethonium include muscle pain, bradycardia, increased intra-ocular pressure and hyperkalemia. It is a known trigger for malignant hyperthermia and “is responsible for over 50% of anaphylactic reactions caused by neuromuscular blocking drugs” (O’Connor & Gwinnutt, n.d, p.3).

On her arrival to the operating theatre, it was clear that she was hypovolemic, due to her pale clammy appearance and tachycardia. She appeared calm despite the unexpected situation she was in. We assisted in her transfer from the trolley to the operating table and secured arm boards for her to rest her arms out on, with gel pads under her elbows to prevent pressure areas. Monitoring of SaO2, NIBP, and a 3lead ECG was commenced, as well as the administration of the Hartmann’s fluid.

Pre-oxygenation began on 100% oxygen at 6 liters, ensuring a good mask seal and clear CO2 waveform on our monitor. After approximately 3 minutes and achieving an end tidal oxygen percentage above 75%, induction began. Pre-oxygenation is important to washout the nitrogen in the lungs, and increases the oxygen concentration, providing us with an increase apneic time, should intubation be difficult. Clear information and reassurance had been given to the patient about the use of cricoid pressure and the possibility of pain on injection.
from the propofol. The anaesthetist injected a bolus of fentanyl (1-5mcg/kg), followed by propofol (1.5-2mg/kg) then suxamethonium (0.5-2.0mg/kg). Cricoid pressure was applied by the technician, to a pressure of 40newtons as consciousness was lost. Laryngoscopy was performed achieving a grade 1 view and the endotracheal tube (ET) was placed.

During the checks of ensuring the correct placement of the ET tube, there was a delay in the carbon dioxide waveform showing on our monitor. The anaesthetist was adamant the tube was in the correct place due to clear visualization of the vocal cords, misting of the tube and symmetrical chest rising on ventilation. He commented that the chest felt “tight” on manual ventilation and asked for a stethoscope. He listened to the chest and announced that the chest sounded “wheezy”. At this point our patient’s vital signs began to change dramatically with a decrease in blood pressure, tachycardia, and a rash was noticed to be developing on her skin. The anaesthetist clearly announced that he thinks we have an anaphylaxis reaction occurring, and specifically directed an instruction at me to call for help and begin drawing up the adrenaline.

An anaphylactic reaction is defined by the Resuscitation Council UK (2008) as a “severe, life threatening generalized or systemic hypersensitivity reaction, characterized by rapidly developing airway and/or circulation problems, and is usually associated with skin and mucosal changes” (p.9). Anaphylactic reactions can be classified into two groups, IgE mediated and Non-IgE mediated, with the difference being if a sensitizing exposure is required or not. Throughout our bodies are mast cells and basophils, which secrete histamine. These cells are commonly referred to as mediators. When they become sensitized to a specific allergen they elicit the generation of an antigen-specific immunogloblin (IgE) antibody. On re-exposure, the antigen combines with the IgE attached to the mast cells, which triggers degranulation and release of histamine” (Marieb, 2001, p.826). The release of histamine induces the inflammatory response and causes a “rapid onset of increased secretion from mucous membranes, increased bronchial smooth muscle tone, decreased vascular smooth muscle tone, and increased capillary permeability” (Krause, 2010). Non-Ig mediated reactions occur without a sensitizing exposure. This is the type of reaction that occurs to suxamethonium, which we encountered in this case study, due to the anaphylactic reaction occurring on the initial exposure.

Signs and symptoms of anaphylaxis are described by the Resuscitation Council UK (2008), through the acronym ABCDE.

A – airway: swelling, stridor
B – breathing: shortness of breath, wheezy, tachypnea, oxygen saturations <92%, cyanosis, respiratory arrest
C – circulation: pale, clammy, tachycardia, hypotension, ECG changes, myocardial depression, bradycardia, myocardial infarction, cardiac arrest

K Hazlett Trainee Anaesthetic Technician
D – disability: confusion, hypoxia, decreased brain perfusion, decreased level of consciousness
E – exposure: skin changes, erythema, urticaria

In regards to this case study, our patient displayed many of these signs and symptoms, which lead our anaesthetist to quickly diagnosis anaphylaxis. Help was sought quickly by pressing the emergency button on our wall, which saw our two other registered nurses and our health care assistant arrive very quickly. I began drawing up the adrenaline, 1 in 10,000 into a 10ml syringe for the anaesthetist to administer 0.1mg per ml, as required. Further instructions were directed to the anaesthetic technician to release cricoid pressure and secure the tube, as confirmation was made that the tube was in the correct place. Focus was then put on attempting another intravenous cannulation. Fortunately, we were able to gain additional venous access with another 20g in the foot and began infusing a stat bag of colloid fluid (starquinn) to increase our patient’s cardiovascular volume and restore her blood pressure. Further help was sought by telephoning a consultant anaesthetist. This task was done by the health care assistant.

Very quickly things turned for the worst as the anaesthetist announced we had lost cardiac output and directed for chest compressions to begin. The surgeon began this task, whilst the anaesthetist administered 1mg of adrenaline intravenously and then continued ventilating the patient. The defibrillator was brought into the room by one of the nurses and I helped set it up quickly and prepare to attach the pads to the patient’s chest following the cycle of CPR.

Adrenaline is a sympathomimetic agent, which directly acts on the adrenergic receptors to produce a ‘fight or flight’ response. It acts on both beta and alpha receptors causing an increase in the force of myocardial contraction, increasing heart rate and cardiac output, constricting blood vessels to increase the blood pressure, dilates bronchial airways and suppresses histamine release. It can be administered via the intravascular or intramuscular route, or via the ET tube. According the guidelines published by the New Zealand Resuscitation Council for the management of anaphylaxis, adrenaline should be administered 0.5mg via the IM route initially, and then revert to 1-2mg via the IV route if cardiac arrest occurs. Adverse effects include headache, sweating and necrosis to peripheral tissues due to vasoconstriction,

Fortunately, cardiac output was regained within the 2 minute cycle of CPR and the defibrillator was not required. We focused on maintaining the patients ABC’s (airway, breathing and circulation), with a phenylephrenine infusion, continued colloid fluid infusion and ventilation with 100% oxygen. An adrenaline infusion was requested to be set up in our intensive care unit to utilize staff, and was brought down as a precaution.

K Hazlett Trainee Anaesthetic Technician
The administration of further propofol, sevoflurane and rocurium were required as the patient began emerging from the anaesthetic. Due to all the excitement, no further doses had been given, and the initial dosages had worn off!

A central line and an arterial femoral line were inserted as soon as the patient was stabilized, to enable close monitoring of our patients vital signs. An indwelling catheter was inserted to enable accurate measurement of fluid balance. Promethazine, an antihistamine; and hydrocortisone, a steroid, were given to stop any further histamine release and prevent delayed reactions from occurring.

The surgery was questioned as to whether it needed to be done, however due to the acute nature it was decided that it was required. The surgery was completed as quickly as possible and fortunately no major bleeding or bowel perforation was found.

Post-operative management included the patient being transferred to the intensive care unit where she was extubated and closely observed overnight. Blood tests were obtained for tryptase levels and she was referred to allergy testing. Tryptase is a major protein component of mast cell secretory granules. During anaphylaxis, mast cell degranulation occurs, which leads to an increase in the blood tryptase concentration. Timing is important for these blood tests, as the levels increase within the first hour, reaching maximum concentration after 6 hours, before returning to normal range after 24 hours. Full documentation was recorded in her clinical notes to prevent this occurring again. Alternatives for using suxamethonium in the future, would be an increase in the dosage of rocuronium, the use of opioids to help blunt reflexes enough to intubate or an awake fibreoptic intubation.

This event highlighted to everyone the importance of always being prepared for emergencies as they can happen at any time when you least expect them. The use of clear communication is of paramount importance. Our anaesthetist remained calm throughout the crisis and gave us clear directed instructions with specific tasks to complete. This ensured that everyone had a task to complete and was efficient in getting these tasks completed, instead of everyone trying to complete the same task and other things getting missed.

The lack of human resources, due to it occurring out of hours, was a critical factor in this crisis situation. This highlighted the importance of thinking outside the square of just the initial team of the anaesthetist, technician and nurses, where we utilized our health care assistant, the surgeon, and the ICU nurses, to assist in performing tasks. The use of an individual team leader, whom can observe and guide the crisis, without being involved in the clinical care, is the ideal situation to ensure tunnel vision is avoided in a crisis situation. This was not possible in this situation, due to it occurring out of hours with very limited staff. The anaesthetist registrar, however, performed this role very well despite being

K Hazlett Trainee Anaesthetic Technician
fully involved in the clinical care. The consultant anaesthetist overtook this role upon her arrival and guided everyone further on the management of the situation once stabilization had occurred. This was helpful as she came in with fresh eyes and ensured further management issues were covered, that we had overlooked due to being caught up in all the drama.

I believe that this crisis was managed well with a successful outcome due to the excellent communication by the anaesthetist and the clear directions which corresponded with the NZRC Guidelines for the Management of Anaphylaxis, (see attached copy), and basic CPR guidelines that every health professional should know. I have learnt a lot from this situation and am more aware of the availability of emergency equipment within our department, and feel more confident in my abilities to assist as required in a crisis situation. Understanding the importance of clear communication is an essential part in coping with a crisis situation, and I will endeavor to maintain this throughout my practice.

K Hazlett Trainee Anaesthetic Technician
References/Bibliography


K Hazlett Trainee Anaesthetic Technician


Scott, M, Dr. (2010). *Crisis Management in Anaesthesia: A Problem Based Approach*. Powerpoint Presentation at Anaesthesia III Block Course on 7th August 2010, Auckland AUT.