Anaphylactic Reaction in a Patient Booked For Elective Subtotal Thyroidectomy under General Anaesthesia A Case Report

By

Adaobi O. Amucheazi

Department Of Anaesthesia, University of Nigeria Teaching Hospital Ituku-Ozalla, Enugu

SUMMARY

Case Definition: This article reports anaphylactic reaction in a patient undergoing thyroidectomy using general anaesthetic technique

Reason for Reporting: A decision was made to report this incidence in order to highlight the importance of the use of clinical skills for patient observation and assessment following the administration of anaesthetic agents and the need for correct diagnosis of an intraoperative event and the prompt treatment of such.

Brief Case Report: The case is reported of acute cardiovascular collapse and cutaneous erythematous rashes immediately after administration of sodium thiopentone and suxamethonium to a 23-year-old lady undergoing thyroidectomy under general anaesthesia. Re-intervention two weeks later with diazepam and suxamethonium led to same collapse leading to the conclusion that suxamethonium was the likely provocative agent.

Conclusion Attention is called to the lack of facilities in our environment for skin testing. However, we must continue to employ the basic clinical skills in order to ensure the optimal outcome for our patients.

Address for Correspondence:
Dr. A.O. Amucheazi
Department of Anaesthesia
University of Nigeria Teaching Hospital
Enugu.
Accepted for Publication: May 30, 2008

Keywords: General anaesthesia, anaphylactic reaction, observation of patient, timely intervention

INTRODUCTION

Anaphylactic reactions are rare but severe in anaesthesia [1]. Clinical presentation may range from hypotension to tachycardia and bronchospasm. Substances, which have been incriminated, include neuromuscular blocking agents such as suxamethonium, induction agents for example sodium thiopentone and infusion such as hexastarch. There may or may not be a history of personal or family atopy, asthma or allergic reaction to substances.

I report the case of a patient who received general anaesthesia using a combination of agents on two separate occasions and consequently developed anaphylactic reaction, which manifested as cutaneous erythematous rash and hypotension.

This case illustrates the need to monitor and observe patients carefully following exposure to anaesthetic agents.

CASE REPORT

A 23-year-old euthyroid lady weighing 70kg was scheduled for cosmetic thyroid surgery. There was no prior exposure to anaesthetic agents. There was no known personal or family history of atopy or asthma or drug allergies. There were no other co-morbidities.

General examination revealed a calm young woman. The pulse was 82 beats per minute, regular and of full volume. Blood pressure was 120/80mmHg. The organs were essentially normal.

Thyroid function tests, serum electrolytes, urea, creatinine and haemoglobin estimation were within the normal limits. She was assessed Mallampati 1 and categorized as ASA

1. Premedication was provided with 10mg of intramuscular diazepam. In the theatre intravenous access was gained with an 18G cannula and connected to one litre of 5% dextrose saline. Monitors attached to the patient gave baseline values and included pulse oximeter (pulse 88beats per minute, saturation of 100%), non-invasive blood pressure (120/80mmHg), and electrocardiogram (normal rhythm). Intravenous atropine 0.6mg was given.

Anaesthesia was induced with thiopentone 375mg. Conditions for intubation were obtained with suxamethonium 70mg. The correct position of the tube was confirmed and the patient was manually ventilated using 2volume percent halothane in oxygen at a flow rate of 8l/minute through a Bain's circuit. Erythematous rashes were then noticed over the shoulders and spread to the arms, chest and lower limbs. The blood pressure dropped to 90/50mmHg and pulse rose to 110-120 beats per minute. The lung fields were clear. A diagnosis of anaphylactic reaction was entertained.

Halothane was turned off and manual ventilation continued with 100% oxygen. The patient was successfully resuscitated using isotonic saline, 1 millimetre of 1:10000 aqueous solution of adrenaline, 200mg of hydrocortisone and 10mg of chlorpheniramine intravenously. With return of adequate spontaneous ventilation, she was extubated awake and returned to the ward. The surgery was deferred for two weeks.

On the night before surgery, 10mg of oral chlorpheniramine and 10mg prednisolone were administered. In theatre, 200 milligram's of hydrocortisone and 10mg of chlorpheniramine were administered intravenously before induction of anaesthesia. Intravenous adrenaline was kept handy in a pre-filled syringe. Anaesthesia was induced with 10mg of diazepam and deepened with 1.5-2 volume percent of halothane in 100% oxygen. Suxamethonium was given to facilitate intubation.

Again, erythematous rashes were noted in the same pattern with a drop in blood pressure and a rise in pulse rate. The patient was resuscitated using oxygen, 100mg of hydrocortisone, isotonic saline and adrenaline. After due consultation, the surgery was continued. All histamine-releasing drugs were avoided. Tramadol, 80mg four hourly and intermittent doses of 20-30mg of ketamine were used to provide analgesia. Anaesthesia was maintained using 2-volume percent halothane in oxygen and pancuronium provided muscle relaxation.

At the end of the surgery, the patient was extubated and moved to the recovery room (post anaesthesia recovery score was 10) for an

extended period. She was moved to the high dependency unit of the ward when she became fully awake (score 15). Postoperative orders included assessment for signs of anaphylaxis and level of consciousness.

Postoperative condition remained satisfactory and she was discharged home nine days later.

DISCUSSION

Clinical anaphylaxis consists of a symptom complex accompanying an acute reaction on exposure to an injected foreign body or an antigen. The incidence during anaesthesia is between 1:5000 and 1:25000 anaesthetics[2].

The agents that have been frequently implicated in anaesthetic practice are suxamethonium[3], rocuronium and sodium thiopentone[2].

During anaphylaxis, sensitization will occur following exposure to an allergenic substance resulting in the production of immunoglobulin E (IgE) which binds to the surface of mast cells and basophils resulting in degranulation and release of proteases such as histamine, interleukin, tryptase, prostaglandins and platelet activating factor. Histamine is responsible for the early signs and symptoms.

These mediators produce vasodilatation, smooth muscle contraction and increased capillary permeability leading to hypovolemia, bronchospasm and erythematous rashes.

The period between exposure and development of symptoms is usually a few minutes when the agent is given parenterally. The reaction occurred in 2-3 minutes in the patient reported. The cardiovascular, respiratory, cutaneous and gastrointestinal systems are affected. This is evidenced by tachycardia, hypotension, erythematous flush, urticaria and bronchospasm. Rhinitis and laryngeal obstruction may also occur. Gastro-intestinal symptoms such as nausea and cramps may not be evident under anaesthesia unlike vomiting and diarrhoea. The patient had hypotension, tachycardia and rashes.

Management plan should be established quickly. Airway management is paramount and involves maintaining a patent airway and oxygen administration. Fortunately, the patient was already intubated. Intravenous fluids should be given to correct the hypovolaemia. The patient was given isotonic saline. Adrenaline, 1-3mls of 1: 10000 aqueous solution, should be administered to reverse the vasodilatation especially for severe reactions[2]. Bronchospasm, if present, is treated with nebulised salbutamol or aminophylline 5-6mg/kg intravenously over 30 minutes. Antihistamines and steroids were used because they counteract the effect of histamine and prevent further mast cell degranulation respectively. However, treatment of the symptoms alone is not sufficient.

Prevention is important in susceptible individuals. Steroids given an hour before anaesthetic agents with or without antihistamines are necessary. Agents known to provoke anaphylaxis should be avoided. Furthermore, the agent must be unravelled. Sodium thiopentone was initially thought to be the culprit. The induction agent was changed to diazepam because midazolam was not available; propfol was expensive for the patient who also rejected inhalational induction. The repeat episode showed that sodium thiopentone was the unlikely provocative agent.

An informed guess (as was the case here) is not a reliable way of determining the cause of supposed allergic reaction[1]. Such may put the patient and others at unnecessary risk. Systematic screening and constitution of allergo-anaesthetic centres to provide expert advice to anaesthetists and allergists are needed in our environment. An investigative protocol should be established and whenever possible, the patient should be referred to a specialist centre. The investigative protocol should be based on the history, skin testing and RIA testing[4]. Skin testing is a reliable tool[1] for such investigations and serves as a guide to future use of neuromuscular blockers. Cross reactivity has been found amongst neuromuscular blockers. Therefore, every available one should be tested[5]. This would provide documented advice for future administration of anaesthetic agents[6].

CONCLUSION

It is needful that facilities for investigation of drug reactions be established in our environment. However, proper clinical assessment and timely intervention would prevent mortality in our patients.

ACKNOWLEDGMENT

I wish to thank Prof B.U.O. Umeh of the Department of Anaesthesia, Nnamdi Azikiwe Teaching Hospital Nnewi for her academic contributions.

REFERENCE

1. Kroiggard M., Garvey L.H., Menne T., Husum B. Allergic reactions in anaesthesia: are suspected cases confirmed by subsequent testing. Br J Anaesth 2005; 95(4): 968-71 Epub 2005 Aug 12

2. Milarec D., Husedzinovic I. Anaphylactic reaction as a side effect of administration of general anaesthetic and neuromuscular blocking agents. Lijec Vjesn 2006; 128 (9-10): 317-21

3 Klein M.T., Moyes D.G. Anaphylaxis due to suxamethonium? - Case report S African Med Journal 1985; 68(8): 601-2

4 Fisher M.M., Baldo B.A. The incidence and clinical features of anaphylactic reactions during anaesthesia in Australia. Ann Fr Anesth Reanim 1993; 12(2): 97-104

5 Dybendal T., Guttornsen A.B., Elsayed S., Askeland B., Harboc T., Florvaag E. Screening for mast cell tryptase and serum IgE antibodies in 18 patients with anaphylactic shock during general anaesthesia Acta Anaesthesiol Scand 2003: 47(10): 1211 8

6 Mertes P.M., Laxennaire M.C. Adverse reactions to neuromuscular blocking agents. Curr Allergy Asthma Rep 2004: 4(1): 7-16