

treatment and increases the chances of recovery. But deciding the appropriate choice of antibiotic is difficult as the results of susceptibility testing vary when different methods are used. Though our patient responded to treatment with vancomycin but there are reports showing failure of this drug in some cases.

From medline search and literature review, the present patient with *Elizabethkingia meningoseptica* diarrhoea is unique as this seems to be the first case being reported from India with manifestation of diarrhoeal due to this microorganism.

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Case Report

Atracurium Anaphylaxis: A Case Report and Review of Literature.

Vineet Kaur, J. K. Bansal, Sarabjit Singh, Gurinder Singh, Dinesh Garg

Departments of Surgery and Anaesthesia

Government Multispeciality Hospital, Sector-16, Chandigarh, India

Abstract: Anaphylaxis is a severe, life-threatening, generalized or systemic hypersensitivity reaction characterized by rapidly developing life threatening airway, breathing and/or circulation problems usually associated with skin and mucosal changes. Anaesthesia is a unique situation for several reasons as many different drugs are used in rapid succession. Adrenaline is the most important drug for the treatment of an anaphylactic reaction. Mortality of anaphylaxis increases if the administration of Adrenaline is delayed or if Adrenaline is used inappropriately. Prompt diagnosis of anaphylaxis and early administration of Adrenaline is important to save the patient. This report describes a life threatening anaphylactic reaction due to injection of Atracurium in a 40 years old female who was scheduled for surgery for laparoscopic cholecystectomy. Anaphylactic and anaphylactoid reactions during anaesthesia are rare, but potentially life-threatening allergic events. The worst manifestations are cardiovascular collapse, bronchospasm and laryngeal oedema. All the drugs and adjuvants we inject in anesthetic practice may be responsible for anaphylactic reactions. But some of them are more allergenic. That is the case with muscle relaxants (NMRs) which induce 50 to 60% of anaphylactic reactions during anaesthesia.

INTRODUCTION

Anaphylaxis is a severe, life-threatening, generalized or systemic hypersensitivity reaction characterized by rapidly developing life threatening airway, breathing and/or circulation problems usually associated with skin and mucosal changes. The incidence of anaphylaxis is increasing and there has been a dramatic growth in the rate of related hospital admissions in the last two decades. Anaphylaxis can occur following exposure to a very broad range of triggers. It has a range of possible presentations and the lack of any consistent clinical manifestations continues to cause diagnostic difficulty. In the study of Jacobsen, none of the 42 anaesthesiologists tested on an anaesthesia simulator, made the correct diagnosis during the first 10 min of anaphylaxis¹. Adrenaline is the most important drug for the treatment of an anaphylactic reaction. Mortality of anaphylaxis increases if the administration of Adrenaline is delayed or if Adrenaline is used inappropriately². The discovery of anaphylaxis goes back to the beginning of the 20th century. Richet and Portier studied the toxic dose of extracts of the sea anemone. In some dogs the first dose of toxin did quick and fatal systemic reaction with respiratory distress and diarrhea. They called this reaction "anaphylaxis", derived from the Greek words a (na), meaning "not or contrary to" and phylaxis, meaning "protection". Thus repeated exposure to a toxin caused harm instead of prophylaxis or

immunization. For this discovery Richet received the Nobel Prize in Physiology and Medicine in 1913^{3,4,5}.

Anaphylaxis can be caused by allergic and non allergic mechanisms. Allergic anaphylaxis is caused by an immediate (type I) hypersensitivity reaction following exposure to an allergen to which the patient has become sensitized. The allergen stimulates IgE-mediated degranulation of mast cells, releasing large quantities of histamine into the circulation which causes intense smooth muscle contraction, increased vascular permeability and vasodilatation. The clinical presentation is the same regardless of whether the reaction has an allergic or nonallergic mechanism.

Anaesthesia is a unique situation for several reasons. Many different drugs are used in rapid succession, not only anaesthetics, but also antibiotics, fluids, nonsteroidal anti-inflammatory drugs and other compounds (e.g. disinfectants, latex, ...). Most of the drugs are given intravenously and in bolus, bypassing the body's primary immune filters and presenting high concentrations of antigen directly to the mast cells and basophils. So it is difficult to say which drug caused the suspected anaphylactic reaction or that the reaction was the result from the additive side effects of several drugs injected simultaneously^{6,7}.

Anaphylaxis remains the most serious adverse reaction due to NMRs, more severe than pharmacological histamine release. It is unpredictable,

Correspondence: Dr. Sarabjit Singh, Surgical Specialist, House No. 3338, Sector 35-D, Chandigarh-160022 India
e-mail : sarabvineet@yahoo.co.in

may be lethal or responsible for severe anoxic sequelae. The incidence of NMRs anaphylaxis has been estimated in France, in 1996, as 1 in 6,500 anaesthetics in which a NMR was used. Allergy to NMRs has been known since 1967, following a publication of Jerums concerning an IgE-dependant anaphylaxis in response to Suxamethonium. During the last 30 years, it has been proven that all the need to be sensitised to QA ion. In three quarters of patients, this sensitisation might have occurred during a previous anaesthetic for which a NMR was used. But 25% of patients who suffered from anaphylaxis to NMRs had never been anaesthetized beforehand. It has been suggested that these patients became sensitized by repeated contact with QA ions contained in cosmetics, antiseptics, detergents^{8,9}.

CASE REPORT

A 40 years old female was scheduled for laparoscopic cholecystectomy. Patient was thoroughly investigated; detailed history was taken and recorded in indoor file. There was no history of previous drug reaction and patient was not asthmatic. Pre-anaesthetic examination was done one day before surgery and no adverse condition was found. For surgery, General anaesthesia was induced with inj Butorphanol 0.5 mg, inj Propofol 100 mg and Suxamethonium 75 mg along with oxygen, nitrous oxide and isoflurane. Following introduction her vitals were stable. On return of spontaneous ventilation, she was administered 25 mg of inj atracurium. Immediately following inj atracurium, she developed severe bronchospasm and became difficult to ventilate. As patient was still on manual ventilation, the rebreathing bag was found stony hard. Endotracheal tube obstruction was ruled out, endobronchial intubation ruled out, circuits were rechecked and found in order, patient was maintained on 100% oxygen through endotracheal tube. Nitrous oxide and Isoflurane were discontinued. Inj deriphyline 20mg and inj hydrocortisone 200 mg were given immediately and ventilation continued. Despite all this, bronchospasm was there and ventilation was difficult so inj terbutaline 0.5mg was given subcutaneously. Till this time patient had tachycardia but blood pressure was maintained, suddenly patients had profound hypotension & peripheral pulses were not felt but on ECG, sinus tachycardia persisted with pulse rate 150/min, oxygen saturation started falling sharply to 75% and patient became cold and clammy. Unconscious and blood pressure became unrecordable. Urgent call to fellow anaesthetists on duty & physician was sent. As this dramatic event had happened immediately following inj atracurium, anaphylaxis to atracurium was diagnosed and inj adrenaline 1ml of 1:10000 I.V. was given to treat the patient. Foot end was raised, fluids rushed on 2 I/V lines & ventilation continued on 100% oxygen. Inj chlorpheniramine 20 mg I/v was given and nebulisation with inj salbutamol was done through endotracheal tube. With no response and patient still in shock, Inj adrenaline 1.0 ml 1:1000 was repeated intramuscularly. Patient responded & bronchospasm cleared in 3 min. saturation was not recordable but blood pressure improved to 70/50 mm Hg. Nor adrenaline infusion was started with 2 mg in 500 ml 5% Dextrose, with initial 2 ml given fast then as BP improved to 84/60, maintained at 0.5 ml/min to maintain blood pressure between 90-100 mm Hg systolic. Surgery was postponed. As patient started having spontaneous respiratory efforts after 25 min of inj atracurium, she was reversed with combination inj of neostigmine 2.5 mg and glycopyrrolate 0.4 mg. patient's respiratory efforts improved but she was kept intubated and assisted spontaneous respiration maintained. After 1hr of inj atracurium, she had respiratory rate of 16-18/min, tidal volume was adequate, reflexes were there, she was conscious & responding to verbal commands so was extubated. She had hoarse voice but no difficulty in breathing & was kept on ventimask. It was noticed that in the meantime, she had urinary incontinence. She was catheterised to monitor urine output which was in the normal limits in post anaphylaxis period. Patient was shifted and monitored in intensive care unit where she recovered but needed vasopressor (nor adrenaline infusion) for 36 hours. She was maintained on inj chlorpheniramine 20mg and inj hydrocortisone 100 mg tds for 3 days. Patient was discharged in satisfactory condition after 3 days with instructions to visit allergy clinic at tertiary care

hospital and all details of the complication and treatment noted on discharge card.

DISCUSSION

There is a range of signs and symptoms. None of which are entirely specific for anaphylactic reaction, however, certain combinations of signs make the diagnosis more likely. Most anaphylactic reactions develop suddenly and evolve rapidly following exposure to a trigger (allergen). There is a rapid progression of symptoms resulting in life-threatening compromise of airway, breathing and/or circulation. Mechanism of anaphylaxis with NMRs is immune mediated causing degranulation of the mast cells and the basophils which release several mediators. The effects of these mediators are decreased myocardial contractility, increased heart rate, coronary and pulmonary vasoconstriction, peripheral vasodilatation, increased hepatic venous resistance with pooling of blood in the splanchnic system, increased permeability (with up to 40% less of intravascular fluid), smooth muscle contraction in the bronchi and the gastro-intestinal tract, increased mucus production, stimulation of sensory nerve endings and attraction of other inflammatory cells. Drugs like atracurium and propofol, release histamine from the mast cell of the lung. Only atracurium releases histamine from the mast cell of the heart. In addition vecuronium inhibits N-methyltransferase, the enzyme that breaks down histamine^{10,11}. The incidence of an anaphylactic reactions to NMRs is 1 in 6500, which is by far the highest of all the anaesthetic. Whittington et al.⁷ described the incidence of the clinical features of anaphylaxis during anaesthesia in more than 500 patients. Cardiovascular collapse was the most common feature in 88% and the worst sign in 78% of the cases. The incidence of cardiac arrest was about 10%. Cutaneous symptoms were recognized in 70% of the cases. This also means that they were absent in 30%, possibly because the patients were anaesthetized and under drapes. Bronchospasm was almost inevitable in patients with pre-existing asthma and was the worst feature in 20% of the cases. Sometimes cardiovascular collapse or bronchospasm were the only signs of anaphylaxis, which made the diagnosis difficult. The most common initial feature during anaesthesia was absence of pulse, difficulty to ventilate the lungs and flushing (in 26%, 24% and 18% of the cases respectively⁷).

The goals of the management of anaphylaxis are-interrupting contact with the responsible drug, modulating the effects of the released mediators and preventing more mediator production and release. Endotracheal intubation should be performed immediately if the airway appears to be at risk (e.g. stridor, oedema of the face or upper airway). To compensate for the intravascular fluid loss, volume expansion is provided with colloids or crystalloids. Elevation of the legs will instantaneously increase the circulating volume with more than half a litre. The cornerstone of successful therapy is Adrenaline. Adrenaline counteracts some of the effects of mediator release stimulation of the $\alpha 1$ -adrenergic receptors constricts the capacitance and resistance blood vessels, stimulation of the $\beta 1$ -adrenergic receptors increases myocardial contractility and stimulation of the $\beta 2$ -adrenergic receptors dilates the smooth muscles of the bronchi. The dose depends on the severity of the symptoms. For less severe reactions, adrenaline can be given intramuscularly in the lateral thigh in a dose of 10 μ g/kg. if adrenaline is given intravenously, it is important to dilute and titrate adrenaline to avoid possible side-effects, like arrhythmias,

hypertension, myocardial ischaemia and infraction. If the patient is hypotensive, boluses of 5 to 10 µg of adrenaline are given every 1 to 2 min. In the case of cardiovascular collapse, boluses of 100 µg are administered every minute together with closed chest cardiac compressions. A higher dose of adrenaline is needed during anaesthesia in comparison to the non-anaesthesia setting, because both general and regional anaesthesia impair the sympathetic response². After the initial therapy, some other drugs, although less important, can be given, like histamine 1 receptor antagonists (promethazine IM). Histamine antagonists compete with histamine at the receptor sites. The usefulness of corticosteroids in treating acute reactions is controversial too. Corticosteroids may require 12-24 h to work; these inhibit phospholipase A2, thus decreasing the mediators formed out of arachidonic acid. Other therapies are inhaled bronchodilators for persistent bronchospasm and catecholamines in infusion for persistent hypotension. After the reaction the patient can be extubated if there is no residual airway oedema. Facial or sclera oedema and absence of an air leak after deflation of the cuff of the endotracheal tube suggest residual airway oedema⁴.

CONCLUSION

Anaphylaxis during anaesthesia presents a diagnostic dilemma. A high index of suspicion should be kept as early diagnosis and treatment is vital for survival of the patient.

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Case Report

Radiological Imaging in Trigeminal Nerve Schwannoma: A Case Report and Review of Literature.

Shibani Mehra, U.C. Garga, Suresh

Department of Radiology, Dr. Ram Manohar Lohia Hospital & PGIMER, New Delhi.

Abstract: Trigeminal nerve schwannomas are rare benign slow growing tumors that constitute 1-2% of all cerebellopontine angle masses and 0.7-0.28 % of all intra cranial tumors. Depending on the site of origin, namely, from the trigeminal root, the gasserian ganglion or the cisternal portion of the nerve, these tumors may present as posterior fossa, middle cranial fossa or dumb bell shaped masses. Extracranial extension to other compartments in the head and neck also occur. Imaging has a major role to play in diagnosis of these slow growing tumors. Radiological Imaging with both CT and MRI not only provides the accurate diagnosis of these tumors based on their site of origin and the extent of the nerve involvement; it also differentiates these tumors from other posterior or middle cranial fossa masses. Imaging has the potential to detect malignant transformation in these otherwise benign tumors. MR Imaging is decidedly superior to CT imaging in the precise diagnosis of Trigeminal schwannomas, but the skull base foramina, through which the V cranial nerve branches exit, are best assessed by computed tomography.

INTRODUCTION

Schwannomas are tumors arising from the Schwann cells in the axon myelin sheaths. Trigeminal Schwannomas are rare intracranial tumors that account for a mere 0.8 - 8% of intracranial Schwannomas¹. These benign tumors are known to have an extracranial component and can grow out into the infratemporal compartment or into the pterygopalatine fossa, through the foramina in the skull vault². They may occur sporadically or in association with Neurofibromatosis type 2. Malignant transformation of previous benign masses is known³. The advances in radiological imaging and the advent of Magnetic Resonance Imaging in particular, have enabled noninvasive imaging of the cranial nerves and Trigeminal Schwannomas can be diagnosed using these modalities before these slow growing tumors become large enough to cause symptoms⁴. Two cases of Trigeminal schwannoma are being discussed and presented with their radiological imaging findings.

CASE REPORT

Case-I

We discuss and present the imaging findings in a 57 year old female who presented with right sided proptosis and diplopia and ipsilateral facial pain. The patient was referred for imaging. MR imaging was performed on a 1.5 Tesla Siemens Somatom Balance scanner in the axial, sagittal and coronal planes using phased head coil for the brain and subsequently oblique sagittal and coronal imaging of the orbit was also performed. T1W images in all three planes were obtained after an intravenous Gadolinium injection. MR images demonstrated a large, well marginated mass in the middle cranial fossa that was isointense to gray matter on T1W images and hyperintense to it on T2W and FLAIR images. The mass was centred in the middle cranial fossa at the Meckel's cave with involvement of right cavernous sinus and encasement of the right internal carotid artery [Fig 1a,b]. Anteriorly the mass was seen to extend into the right orbit, displacing the globe and the optic nerve and causing proptosis, while posteriorly, it was