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Anaphylaxis in clinical practice

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Abstract

Anaphylaxis is a life threatening condition every physician is frightened of. We always suspect our patients to develop anaphylaxis to our treatment. In the past 5 years 13 episodes of anaphylaxis were taken with different types of drugs, insect bites, food allergens, vaccinations, local or regional anesthetics agents and antibiotics. The timely identifying and available of resourceful persons, drugs and equipment are necessary to save lives. Counting the fingers to complete the task as thumb for the airway, index for the life line, middle for the injection epinephrine, ring for the Injection dexamethasone and little finger for the ranitidine will save cent percent of lives during anaphylaxis crisis. Education on anaphylaxis to general community is very important, especially for the patients and their families [1]. Prompt medical assistance in patients with anaphylactic symptoms determines their therapeutic response.

Keywords: Allergy, epinephrine (adrenaline), anaphylaxis, occupational health hazards, β -lactam antibiotics, mast cell degranulation, immunotherapy

Introduction

Back Ground: Anaphylaxis is a serious allergic reaction that is rapid in onset and may cause death (2). Charles Richet awarded the Nobel Prize in Physiology or Medicine for his work on anaphylaxis in 1913 [2]. The term comes from the Ancient Greek: *ἀνά ana* ", against the Ancient Greek: *φύλαξις phylaxis* which means protection. The prevalence of allergic diseases is raising worldwide, due to more exposure to environmental allergens and pollutants. The onset may be sudden and without warning. Severity differs in each episode even with an identical stimulus in the same patient. It typically causes a number of symptoms including an itching, rash, low blood pressure and throat swelling. Worldwide, 0.05–2% of the population suffered anaphylaxis at some point in life [3]. Anaphylaxis typically presents with many different symptoms over minutes or hours [4] with an average onset of 5 to 30 minutes if exposure is intravenous and 2 hours for foods [2]. Common triggers include venom from insect bites or stings, foods [5], and medication [7, 8]. Neuromuscular blocking agents, latex and antibiotics are the frequent causes [6]. The cause remains unknown in 32–50% of cases, referred to as "Idiopathic anaphylaxis [7, 8] Six vaccines (MMR, Hepatitis B, Tetanus, Meningococcal Varicella, Influenza) are the cause for anaphylaxis, and HPV may cause anaphylaxis as well [8]. Medication like β -lactam antibiotics (such as penicillin) aspirin and NSAIDs [4] chemotherapy, vaccines, protamine and herbal preparations. [3, 9] (Vancomycin, morphine, x-ray contrast among others) cause anaphylaxis by directly triggering mast cell degranulation [7]. The frequency of a reaction to an agent partly depends on the frequency of its use and partly on its intrinsic properties [13]. Anaphylaxis to penicillin or cephalosporins occurs only after it binds to proteins inside the body with some agents binding more easily than others [2]. Anaphylaxis to penicillin occurs in the frequency of 2,000 to 10,000 courses, with death occurring in one in every 50,000 courses of therapy [2] If someone has a reaction to penicillins, his or her risk of a reaction to cephalosporins is greater [2] People with atopic diseases are prone to anaphylaxis from food, latex, and radio contrast. The longer the time since the last exposure to the agent in question is the lower the risk [2].

Anaphylaxis is a life threatening condition and timely therapy saves the life. In the past 2 decades we observed different drugs causing anaphylaxis reaction and these are illustrated as multiple case studies. These cases were seen in 5 years of span in our clinical practice in a teaching hospital.

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Episode 1: A chartered accountant came to my home to call on me. He was greeted and served snacks and tea. While eating the snacks suddenly he noticed numbness and dry mouth. He informed me as some sesame seeds^[10] are in the snacks for which he is allergic. He had angio neurotic oedema and difficulty in breathing and hypotension. Immediately he was resuscitated with epinephrine, dexamethasone, rantidine and Chlorphenaminmaleat parentally. He was revived and able to sit after one hour. He gave the family history of sesame seed or sesame oil allergy in all his eight family members. Oleosins the allergen, present insesame oils and seeds. In all sesame allergic patients studied, the igE is bound to oleosins^[10].

Episode 2: When I was in Army I saw a subedar who developed hypotension and angioneuratic oedema and course crepitations in chest following ingestion of a tablet of Aspirin. He was resuscitated with epinephrine and rantidine parentally and he recovered. *Aspirin-exacerbated respiratory disease (AERD)* Patients with AERD also have increased respiratory tract expression of the cysteinyl leukotriene 1 receptor and heightened responsiveness to inhaled leukotriene E4. Administration of aspirin leads to inhibition of cyclooxygenase 1 (COX-1) with resultant decrease in prostaglandin E2, and inhibits 5-lipoxygenase, but lack of this effect, arachidonic acid molecules are metabolized in the 5-lipoxygenase pathway, causing increased production of cysteinyl leukotrienes^[11].

Episode 3: A 40 year old nurse, name S, by profession and working in pre & post-operative ward for the last 2 decades. Antibiotic sensitivity test and administration of the antibiotic to the prescribed patients.

Four years ago, she gave the Intradermal antibiotic sensitivity test for Cefoperazone Sodium Injection on right forearm and marked the site. The nurse after few minutes developed nausea and vomiting. She thought it is due to some other reason. After 15 minutes the patient who was tested for the antibiotic lifted the nurse and brought to the examination room. On examination she was conscious and coherent. PR 104/mt and regular. Bp was 98mm of Hg systolic. Rash was seen and foreign body sensation in throat and palpitation felt. On skin: flushing, itching, urticaria, angioedema and difficulty in swallowing seen, nausea and vomiting and bloating complained. cough, wheezing, dyspnea, chest tightness was present. She was resuscitated and parentally DNS started, Inj. Adrenaline 0.5ml^[12], I.M, Chlorpheniramine 2ml^[13], I.V, Inj. Dexamethasone 2ml^[7]. I.V, airway maintained and Inj. Rantidine I.M 2ml^[13] given. Admitted at General hospital, ECG and other vital investigation done. The palpitation and rash subsided after 6 to 8 hours. It is a mystery to the doctors as how she developed allergic reaction without any contact with the drug except possible inhalation while mixing distilled water in to the drug^[14].

The second day without informing to any person she wore operation dress, face mask and cap and she gave the antibiotic to the patient which was recognized later by the doctors and warned her not to venture in such activities.

Two years ago she was working in a orthopedics hospital and she gave the same drug to a post-operative case and she developed anaphylaxis with itching and breathlessness and then resuscitated.

15th January 2015 She was attending to a case of Diabetic foot with the general surgeon and when the patient was advised antibiotic Inj. Piperacillin and Tazobactam, she assisted the surgeon in mixing the distilled water and she had skin wheal, flushing, itching, and difficulty in swallowing seen. Nausea and vomiting, chest tightness were present and immediately she was treated for the anaphylaxis with adrenalin and she recovered in 20 to 30 minutes. But she complained of difficulty in swallowing for one day. Three times with different antibiotics she had anaphylactic reaction^[14].

Episode 4: A 30 year old lady with an abscess reported to a clinic. She was given sulpha meth axazole with trimethaprim combination^[15]. After 2 hours she came again with erythema around the mouth, agioneuroti coede maand itching. She was given epinephrine, dexamethasone, rantidine and chlorpheniramine and she recovered.

Episode 5: Injection Imferon was given to an anemic lady of 35 years parentally. Immediately she developed angioneuroticoedema and strider. She was given epinephrine, dexamethasone and airway and parental fluids. She was revived. Such anaphylaxis seen even while giving the injection iron sucrose, iron gluconate (sodium ferric gluconate), iron dextran, ferric carboxymaltose, and iron isomaltoside 1000^[16]. At our study 450 pregnant anemic cases with iron sucrose we saw 14 cases of mild and one case of fatal reaction after one hour of stopping the drug.

Episode 6: An house wife aged 35 years came with a cut wound with a butchers knife while cutting the mutton. She was given anti tetanus serum (ATS) parentally. Immediately she developed angioneurotic oedema and rashes. She was treated with epinephrine and revived. This is due to ATS related to IgE in horse serum albumin. Equ c 1, Equ c 2, Equ c 3 are potential allergens can cause upper and lower respiratory symptoms in sensitized patients^[17, 18]. Even tetanus toxoid can cause anaphylaxis due to mercury component. If passive immunization is required, Tetanus Immune Globulin (TIG) (Human) should be used^[19].

Episode 7: A 45 year old lady, known diabetic with septic ulcer on right leg was advised insulin. 6 units of bovine insulin were given. Immediately she had rashes, angio-neurotic oedema, erythmatic rshes and hypotension,^[11] shewas treated with epinephrine and revived. However, life-threatening allergic reactions to human insulin and insulin analogs (Aspart, Lispro, and Glargine) have been documented and can be confirmed by appropriate intracutaneous and/or in vitro testing^[20]. The mechanisms of immunogenic reactions to recombinant human insulin are not entirely clear but may relate to structural changes of insulin, including insulin aggregation (fibrillation).

Episode 8: While doing laparoscopic sterilization after a test dose of lidocain, 5ml drug was infiltrated as local anesthesia. Immediately she had thrown fits^[21, 22, 23], She was treated with medizolam and other drugs and revived after 2 hours and cancelled the surgery.

Episode 8a: In a case of laparoscopic sterilization after doing sensitivity dose testing and while injecting 5ml lidocain1% locally she developed angioneurotic oedema and

tetany. She was given epinephrine, decadron and Injection Calcium gluconat 10% and revived the patient.

Episode 8b: In a case of emergency cesarean section after 24 hours she developed hypotension and with fluids and blood transfusion and epinephrine she was revived. This appears as a case of delayed hypersensitivity or biphasic reaction to the spinal anesthesia drug [24].

Episode 9: Injection streptomycin was prescribed to a case of Koch's disease. After the injection she developed anaphylaxis reaction and she was treated with epinephrine injection and supporting treatment she was revived.

Episode 10: A lady of 50 years had a red ant bite [4] on the foot and she developed angioneurotic oedema. Epinephrine and corticosteroids revived her.

Episode 11: A staff nurse, a known hypertensive on β blockers developed rashes, wheel and puffiness of face after she wore latex gloves and was identified as sensitive to latex. She could not be revived with routine anti anaphylactic measures. Later she was given glucagon and anaphylaxis responded [25]. In a normal person, a systemic dose of epinephrine does not have much effect on mean blood pressure because it has both α adrenergic effects (producing vasoconstriction) and β adrenergic effects (producing vasodilation). If a patient on a nonselective β blocker receives a systemic dose of epinephrine, the β -blocker prevents the vasodilation and α vasoconstriction is unopposed. The hypertensive reaction can cause over 200 mm Hg [26]. Cardio selective β blockers, such as acebutolol, atenolol, betaxolol, bisoprolol, esmolol, and metoprolol are not expected to cause hypertensive reactions following a systemic dose of epinephrine. This is because cardioselective β blockers have little effect on the beta-adrenergic receptors in the arterioles. In addition to vasopressin, glucagon may be indicated for the treatment of hypotension and bronchospasm for patients taking β -blockers who are unresponsive to epinephrine. Glucagon is an effective antidote because it directly activates adenylcyclase, precluding receptors.

Episode 12: A karate practitioner aged 16 years had acute abdomen and a practitioner gave baralgin injection to him, after one hour patient had angio neurotic oedema and pulmonary oedema and hypotension. He was given epinephrine and admitted in a gastroenterology hospital and treated but he died. The postmortem reported as septicemia death. This case argued in consumer forum for 5 lakhs. Timely action, information to attendant, drug therapy, transport to higher center were satisfied the arguments and case was dismissed.

Episode 13: A 31 year old young man had diarrhea for 10 days and he was on tablet ciprofloxacin which was changed as intravenous injection. Immediately angioneurotic oedema seen and the drug stopped and epinephrine and corticosteroids, ranitidine, chlorpheniramine maleolate given and revived him. Later he recovered from diarrhea. This is an anaphylactoid reaction as Oral drug ciprofloxacin is never caused anaphylaxis to him.

Discussion: There are three main classifications of anaphylaxis. 1. Anaphylactic shock 2. Biphasic anaphylaxis is the recurrence of symptoms within 1–72 hours with no further exposure to the allergen [7] 3. Pseudoanaphylaxis or anaphylactoid reactions are a type of anaphylaxis that does not involve an allergic reaction but is due to direct mast cell degranulation (28) "true" anaphylaxis is caused by degranulation of mast cells or basophils mediated by immunoglobulin E (IgE), and pseudo-anaphylaxis occurs without IgE mediation (4)

During anaphylaxis, a number of inflammatory mediators are released from basophils and mast cells. Histamine plays an important pivotal role in acute allergic inflammation. It is a complex network of events that involve redundant mediators and signals, including carboxypeptidase, trypsin, platelet-activating factor, leukotrienes, cytokines and prostaglandins. However, there may be sufficient redundancy and amplification are present in systemic responses such that reactions do not respond to a single mediator antagonist (Simons 2004b; Winbery 2002).

Signs and Symptoms of Anaphylaxis

Anaphylaxis typically presents with many different symptoms over minutes or hours [4, 5] with an average onset of 5 to 30 minutes if exposure is intravenous and 2 hours for foods. [6] The most common areas affected include: skin (80–90%), respiratory (70%), gastrointestinal (30–45%), heart and vasculature (10–45%), and central nervous system (10–15%) [5] With usually two or more being involved [3].

On skin itching, flushing, angioedema. Rashes are common. Swelling of the tongue or throat occurs in about 20% of cases. [7] Other features may include a running nose and swelling of the conjunctiva [8]. The skin may also be blue tinged because of cyanosis.

Shortness of breath, wheeze and stridor may be present. Tachycardia, hypotension usually occurs. There may be myocardial infarction, arrhythmias and cardiac arrest due to coronary arterial spasm. The differential diagnosis are asthma, syncope, and panic attacks [3].

Iron sucrose: A total of 317 cases of hypersensitivity were identified from the MAH database, which occurred in 13,824,369 patient years (cut-off date 31 December 2011). The majority of patients were female. In 8 cases of the total 51 life-threatening cases (15.7%; 8/51 cases, one graded as Grade IV patient with known allergy). Known predisposition to an allergy or asthma may result in a more severe reaction [16].

Epinephrine is the treatment of choice for anaphylaxis [12] Epinephrine administration enhances coronary blood flow by increased duration of diastole compared with systole and a vasodilator effect caused by increased myocardial contractility. They usually offset the vasoconstrictor effects of epinephrine on the coronary arteries [12]. Aqueous epinephrine, 0.01 mg/kg (maximum dose, 0.5mg) administered intramuscularly every 5 to 15 minutes as needed is the recommended dosage for controlling symptoms and maintaining blood pressure [27]. The 5-minute interval between injections can be liberalized to permit more frequent injections necessary. Vasopressors, Histamine 1 and Antihistamines (H1- and H2-antagonists) [3] are slower in onset of action than epinephrine, do not effect on blood pressure, and Antihistamine is considered a second-line of treatment for anaphylaxis. Corticosteroids may help in

controlling the biphasic reactions. Post anaphylaxis should carry self-injectable epinephrine for use if anaphylaxis develops^[1].

Prevention: Avoidance of the trigger of anaphylaxis is recommended. Desensitization may be attempted. Immunotherapy with Hymenoptera venoms is effective at desensitizing 80–90% of adults and 98% of children against allergies to insect bite.

Conclusion: The main treatment for anaphylaxis is epinephrine. Corticosteroids and antihistamines, whose binding receptors are either H1—such as diphenhydramine—or H2—such as cimetidine or ranitidine—are only second line therapy in the management of anaphylaxis.

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