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Case Report

Anaphylaxis should be recognized early and managed appropriately

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ABSTRACT

Anaphylaxis is a severe, systemic hypersensitivity reaction characterized by rapid in onset with potentially life-threatening. The prevalence of anaphylaxis has been estimated at 1.6% to 5.1% and increasing nowadays. Drug-Induce-Anaphylaxis is one the most cause of fatal anaphylaxis. A 26-year-old male with anaphylaxis shock due to drug-induced with total IgE as a diagnostic support test. Anaphylaxis is a multisystem allergic emergency. Early recognition and prompt administration of epinephrine remain the cornerstone of management due to its rapid progression.

Keywords: Anaphylaxis shock, IgE total, Epinephrine

INTRODUCTION

Anaphylaxis is a severe systemic hypersensitivity reaction characterized by being rapid in onset with potentially life-threatening for airway, breathing, or circulatory problems and is usually, although not always, associated with skin and mucosal changes.[1] The lifetime prevalence of anaphylaxis has been estimated at 1.6% to 5.1% and increasing nowdays.[2] The leading cause of fatal anaphylaxis is drug (29%-58.5%) such as Nonsteroidal Anti-Inflammatory Drugs (NSAIDs) for 11.5% of cases.[3] Drug-induce anaphylaxis (DIA) usually managed in ED because its rapid progression. Only 8% of patients received epinephrine whereas it is the bedrock of anaphylaxis management. Although anaphylaxis is acute progressive allergic reaction, deaths are rare, with 0.33-3 fatalities per 1.000.000 population per annum. Death occurs in 1% of hospitalization and 0.1% of ED attendance.

Anaphylaxis must be diagnosed and managed quickly, but confirmatory testing is still inefficient. Here we present a case report of a 28-year-old male with anaphylaxis shock due to drug-induced with total IgE as a confirmatory test.

CASE REPORT

A 28-year-old male came to the clinic with headache and myalgia and was given Ibuprofen and vitamin B. He took the medicines and returned home. Around three hours later, his eyes began to itch and swell. The symptoms quickly worsened, he suddenly felt pressure in his chest. He came to the hospital. When the patient arrived, he was still conscious, looked weak, was short of breath, there was no audible wheezing, and had cold hands and feet. This was the first time he had a complaint like this. He never had asthma or allergic rhinitis. Chest pain was denied. There was no history of food or medication allergies. There is no allergy history in the family.

Physical examination showed that he looked seriously ill, compos mentis, with GCS E4V5M6. Blood pressure of 85/60 mmHg, heart rate of 118 bpm, respiratory rate of 28 times/min, temperature 36.7°C, and oxygen saturation of 96% at room air. There was bilateral periorbital edema.

Cardiac examination within normal limits. There were supraclavicular and substernal retractions, vesicular breath sounds, wheezing on both sides of the lungs, and no crackles were found. Abdomen and extremities were within normal limits.

An ECG revealed sinus tachycardia with 115 bpm. A complete blood count showed leukocytes 12,310/µl, hemoglobin 14.9, hematocrit 41.6%, and platelets 264,000. Blood sugar 194 mg/dl. Total IgE 315.04 KIU/l. Roentgen's thorax showed no abnormalities. He was managed with epinephrine 0.3 mg IM, given 4 l/min oxygen using a nasal cannula, nebulized with salbutamol and ipratropium bromide. He got twice injection of epinephrine (his blood pressure (BP) improved to 98/65 mmHg). Intravenous crystalloid NaCl 0.9% 500 cc was given followed by a rate of 20 drops/min. His BP increased to 110/75 mmHg after fluid loading. He also received an injection of methylprednisolone 125 mg, followed by 62.5 mg BID, and loratadine QD. He was discharged after two days of admission.

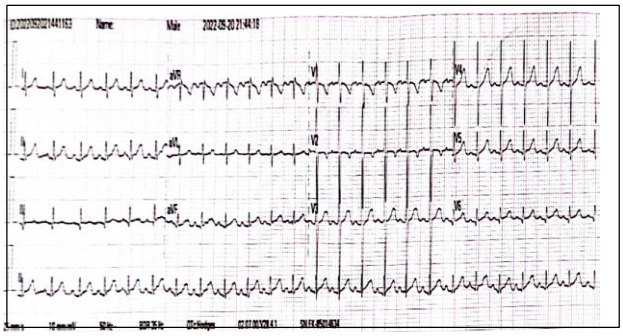

Figure 1: Electrocardiogram result.

DISCUSSION

There are three phases of anaphylaxis process. Sensitization is an asymptomatic primary immune response in which an individual is exposed to an allergen and produces IgE antibodies secreted by B cells. The activation phase occurs when the IgE antibody binds and cross-links to the IgE receptor (FceRI) on the surface of mast cells or basophil cells, causing degranulation and the rapid release of inflammatory mediators. A clinical outcome that occurred during the activation phase is referred to the effector phase.[2-4]

Mast cells and basophils contain preformed granules composed of histamine, proteases (tryptase, chymase), proteoglycans, (heparin, chondroitin sulfate), and TNF-α. High concentration of histamine release can result at hypotension and tachycardia. Some mediators, like cysteinyl leukotrienes and prostaglandin D2, cause bronchoconstriction and increased microvascular permeability. Serum platelet-activating-factor (PAF) levels can correlate with anaphylaxis severity. Tryptase and chymase can activate complement and coagulation pathways, which result in the production of anaphylatoxins (C3a and C5a) and the kallikrein-kinin system, which regulate blood pressure and its permeability. All the component mediators are likely additive or synergistic in the target tissues.[2,4]

Anaphylaxis is present if one of the following three diagnostic criteria is met: (1) acute onset (minutes to hours) with involvement of the skin, mucosal tissue, or both with either respiratory involvement or reduced blood pressure and or associated symptoms of end-organ dysfunction; or (2) two or more of the following that occur rapidly after exposure to a likely allergen including- (i) involvement of skin-mucosal tissue, (ii) respiratory involvement, (iii) reduce blood pressure or associated symptoms, or (iv) gastrointestinal symptoms; or (3) reduced blood pressure as a result of exposure to a known allergen trigger (systolic blood pressure of less than 90 mmHg or greater than 30% decreased from the baseline).[2]

The underlying reasons why some people only had modest type I hypersensitivity while others suffer extreme manifestations remain unknown. It may be influenced by intrinsic (patient-related) and extrinsic factors.[3] Older age, mast cell disease, and a history of using beta-blocker or angiotensin-converting enzyme inhibitors are all risk factors for severe anaphylaxis. Atopy is a risk factor for anaphylaxis triggered by food, exercise, and latex.[2]

IgE was found at the lowest concentrations in the circulation (50-200 ng/ml total). It is crucial to know that increased IgE levels can also be found in the allergic patient and certain other diseases, such as multiple myeloma, heavy chain disease, liver disease, and rheumatoid arthritis. The sensitivity of total IgE was 88.9%, which showed that it is a useful index for screening allergic disease or anaphylaxis at a low cost compared to sIgE.[5] Some patients with life-threatening anaphylaxis may have low or undetectable circulating sIgE or total IgE because it may correlated to non-IgE anaphylaxis.[2,5,6] Serum IgE is also influenced by age. Progressive increase observed up to age 15-year-old, then declines from the 2nd through the 8th decades of life.[7] Total serum IgE may be influenced by medication (omalizumab, rituximab, ligelizumab, dupilumab, and corticosteroids), air pollution, smoking, and vitamin D level.[8]

Based on his complaint, he was weak, feeling tightness on his chest, shortness of breath, and cold in both hands and feet around 3 hours after taking medicine (rapid onset). His BP was low, tachycardia, and there are bilateral swollen palpebrae, with wheezing in both sides of the lungs and cold extremities. This condition meets the criteria of anaphylaxis shock which are acute on onset (less than 6 hours) with involvement of skin, mucosal tissue, and respiratory, and there is reduced blood pressure. Total serum IgE was high. Leucocytosis and elevated blood glucose levels may occur because of stress and increase catecholamine concentration.[6] The first-line therapy for anaphylaxis is epinephrine IM (dosage of 0.01 mg/kg to a maximum of 0.5 mg in adults and 0.3 mg in children) into

the anterolateral thigh as soon as possible.[3] This patient was administered 0.3 mg IM epinephrine. First his BP remained at 85/70 mmHg then increased to 105/85 mmHg after the second injection. Repeat epinephrine every 5-15 min, if the condition does not improve.[2,5] The patient should lay down, do not allow to stand or walk. If unconscious or pregnant, place in recovery position. If breathing is difficult allow the patient to sit with legs outstretched.[9] When using epinephrine auto injector (EAI), positioning is not the priority, the important thing is administered epinephrine as soon as possible.[10] Those who require more than one dosage of epinephrine are at a higher risk of developing biphasic response.[2] There is no biphasic reaction in this case. This patient is also used oxygen through nasal cannula, nebulized with SABA and Ipratropium Bromide to reduce bronchoconstriction, and loading with NaCl 0.9% 500 cc to improve the intravascular volume continue with 20 drop/min. He also had methylprednisolone 125 mg injection, followed by 62.5 mg BID, and loratadine 10 mg QD to reduce the late reaction.

Epinephrine is a nonselective adrenergic agonist. It works rapidly to increase peripheral vascular resistance through vasoconstriction, increasing cardiac output, reverse bronchoconstriction, mucosal edema, and stabilizing mast cells and basophils. Antihistamine is considered a second-line treatment. It used to prevent mast cell degranulation or to target additional mediators of anaphylaxis. Glucocorticoid is frequently used as adjunctive therapy for anaphylaxis. It contributes to inhibit production of new inflammatory mediators.

CONCLUSION

Anaphylaxis is a multisystem allergic emergency. Early recognition and prompt administration of epinephrine remain the cornerstone of management due to its rapid progression.

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