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Anaphylaxis: A Case Report from Azal hospital, Sana'a, Yemen

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Abstract

Anaphylaxis is a severe life-threatening allergic reaction. This was a type I hypersensitivity reaction. Antibody reactions induce the release of inflammatory mediators. In this article, we report the case of a 55 years old female patient who presented with generalized urticaria, pruritus, skin edema, fever, abdominal pain, and dyspnea. Investigations showed only neutrophilia. The patient was immediately administered intravenous (IV) chlorpheniramine 10 mg and intravenous hydrocortisone 100 mg with a slight response. The patient then received intravenous methylprednisolone 500 mg with dramatic improvement. This drug was administered once daily for three consecutive days.

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1. INTRODUCTION

Anaphylaxis is a clinical emergency that requires immediate intervention[1]. Food, medication, and stinging insects are the main causes of anaphylaxis. Idiopathic anaphylaxis accounts for 10–60% of anaphylactic cases [2]. Typically, anaphylaxis is an immunoglobulin (Ig) E-mediated reaction that leads to the degranulation of mast cells. IgG also contributes to anaphylaxis. Food allergies were the most common cause of anaphylaxis, followed by drugs. Symptoms of anaphylaxis involve the skin and mucous membranes, followed by the respiratory and gastrointestinal tract. Adrenaline is the drug of choice for anaphylaxis [3]. Anaphylaxis is a systemic type I hypersensitivity that can be fatal. It involves mast cell-rich organs such as the skin, lungs, and gastrointestinal tract. Histamine and other inflammatory mediators are typically released from mast cells and basophils and promote vascular permeability and smooth muscle contraction [4]. Intramuscular adrenaline is the treatment of choice for anaphylaxis. Intravenous adrenaline with fluid resuscitation should be administered to patients with shock. Early intubation may be necessary for airway obstruction refractory to adrenaline [5]. Drug-induced anaphylaxis is a common cause of anaphylaxis, and includes antibiotics, radiocontrast, and non-steroidal anti-inflammatory drugs [6]. Anaphylaxis usually begins within 2–120 min after exposure to the trigger antigen. The causes of death include asphyxiation from laryngeal or oropharyngeal swelling, collapse from hypotensive shock, cardiac arrest, or acute severe bronchoconstriction, which causes respiratory failure [7]. Anaphylaxis is considered a medical emergency [8]. Useful second-line interventions include removing the trigger whenever possible, correct positioning of the patient, high-flow oxygen, intravenous fluids, inhaled short-acting bronchodilators, and nebulized adrenaline [9]. Cofactors including exercise, non-steroidal anti-inflammatory drugs, alcohol, and sleep deprivation may play a role in approximately 30% of anaphylactic reactions in adults [10]. Moreover, food-dependent exercise-induced anaphylaxis (FDEIA) is an additional cofactor in which symptoms occur only in combination with food intake [11, 12]. Occupational anaphylaxis (OcAn) arising out of triggers and conditions

attributable to a particular work environment, for example hymenoptera stings and natural rubber latex are the commonest triggers of OcAn [13]. From a pathophysiological point of view, anaphylaxis can be an IgE-mediated immune response involving a massive release of biologically active mediators from mast cells and basophils (IgE-dependent). Mastocyt/basophil can also be degranulated via a direct IgE-free pathway, that is, non-allergic or non-IgE-dependent, and it is called anaphylactoid anaphylaxis [14]. Case presentation A 55 years old female presented to the internal medicine clinic of our hospital with itching, skin swelling, redness, and shortness of breath. Nutritional and medical history revealed shrimp intake and ceftriaxone administration. General examination revealed widespread urticaria, generalized edema, and decreased air entry into both lungs. vital signs were as follows: blood pressure, 110/70 mmHg; pulse rate, 69 beats/min; temperature, 38 C and oxygen saturation, 88%. Arterial blood gases (ABG) showed pH 7.5 and bicarbonate 24 mEq/L. Interestingly IgE level was normal at 25 IU/ml. Immediately, the patient received IV chlorpheniramine (10 mg) and IV hydrocortisone (100 mg) with slight improvement, and was admitted to the medical ward. She is a known case of type 2 diabetes mellitus and was on sitagliptin/metformin 50/500 mg 1*2 and empagliflozin 25 mg 1*1. On admission, her random blood sugar was 539 mg/dL, serum albumin 30 mg/dL, and neutrophils 83%, but with normal creatinine and liver function test results. In the medical ward, the patient received the following medication plan by the IV route: chlorpheniramine 10 mg 1*2, hydrocortisone 100 mg 1*2, pantoprazole 40 mg 1*2, ondansetron 4 mg 1*2, levofloxacin 500 mg 1*2, normal saline 500 ml 1*2, and soluble insulin 10:10:8 units, with nocturnal long-acting insulin: glargine 10 units. However, the patient's allergic state deteriorated after 24 hours. Hydrocortisone was then replaced with dexamethasone (80 mg), but there was no improvement. Finally, a decision was made to administer pulse therapy of 500 mg IV methylprednisolone (MP) daily for 3-5 days. After 48 h, the patient's clinical state dramatically improved, and MP was continued for 4 days. Itching, skin swelling, redness, and shortness of breath improved and almost disappeared (Figures (1, 2)).

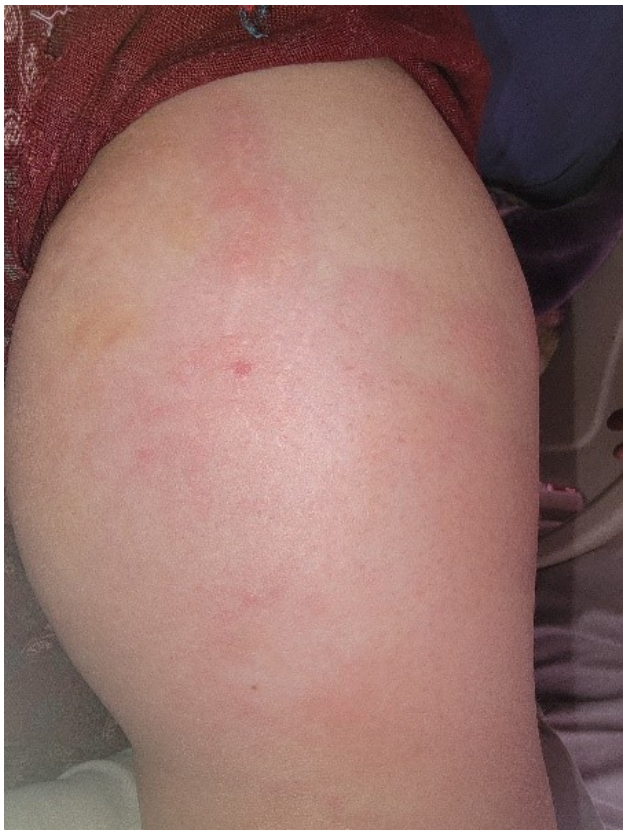

Figure 1. The cubital fossa on admission

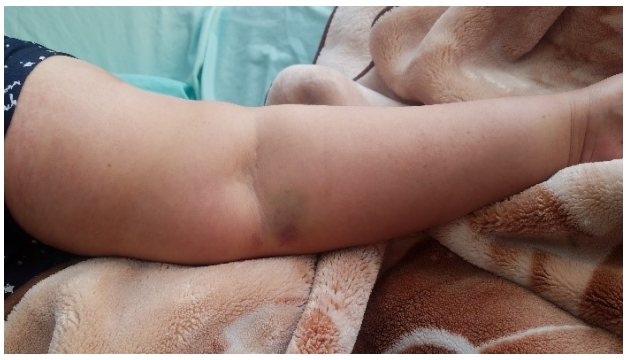

Figure 2. After 48 hours of methylprednisolone

2. DISCUSSION

Anaphylaxis is a medical emergency that necessitates admission to a medical ward. We started the traditional treatment for anaphylaxis: IV chlorpheniramine 10 mg and IV hydrocortisone 100 mg. However, adrenaline was avoided because of the fear of heart complications, and echocardiogram showed grade II impaired diastolic relaxation. Moreover, the patient was not hypotensive and respiratory manifestations were not prominent; both are indications for adrenaline. Levofloxacin was included in

the medication plan as an antimicrobial cover because of neutrophilia (83%) and elevated C-reactive protein level (CRP): 31 mg/L). Beta-lactam antibiotics were avoided due to a history of allergy. Blood sugar levels were controlled using soluble insulin. Methylprednisolone pulse therapy is used for severely resistant cases of autoimmune diseases, and in our case, it showed quick and enormous improvement. A few articles have reported that methylprednisolone itself may cause anaphylaxis [15]. Therefore, care should be taken when using these devices.

3. CONCLUSION

Anaphylaxis is a medical emergency that should be managed immediately to achieve good prognosis. Chlorpheniramine, hydrocortisone, and adrenaline were cardinal treatments in this case. Pulse therapy with IV methylprednisolone 500 mg daily for 4 days is a good choice for resistant cases.

HUMAN ETHICS

Consent was obtained from our patient in this study.

CONFLICT OF INTEREST STATEMENT

The authors declare no conflict of interest.

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