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Fatal Anaphylactic Shock from Wasp Sting Despite Desensitisation - A Case Report

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1. Abstract

Anaphylaxis from Hymenoptera despite conventional venom immunotherapy is rare. We present a case in a previously healthy male with suspected cutaneous mastocytosis, who developed a fatal anaphylaxis due to cerebral hypoperfusion after wasp sting. The report is a reminder that up to 20% of patients allergic to Hymenoptera venom are not protected by conventional venom immunotherapy.

2. Case Presentation

We are reporting a case of a previously fit and healthy male in his forties, with a medical history of suspected mast cell disease due to urticaria pigmentosa (although never confirm with biopsy) and having completed an allergen specific immunotherapy program within the last 12 months. The patient had been stung 5 times over the summer from wasps without any severe reactions. He had never needed to use injectable adrenaline (EpiPen® Auto-Injector) previously.

While collecting berries along a local stream in rural Norway, the patient was stung and an immediate anaphylactic reaction occurred. His wife was present and ran to collect EpiPen® Auto-Injector and alerted local emergency services; after injecting adrenaline (0.3mg) approximately 20 minutes later, CPR was started and ROSC achieved. Emergency medical services (EMS) accompanied by local general practitioner (GP) arrived after 31 minutes and hydrocortisone was administered (Solu-Cortef® 100mg). First set of vital at scene was recorded as: BP 187/160, HR 60, RR 26/min, Temp. 34.3 Celsius, BM 15.7mmol/l and SpO2 96% on 9L O2.

On arrival in hospital 1 hour and 38 minutes after the incident the vitals were: BP 100/60, HR 139, Saturation 77%, RR 24, CRT>5 sec with neurological extension pattern of upper extremities. The airway was secured with rapid sequence intubation with Ketamine® and Suxamethonium® along with ongoing fluid resuscitation and adrenaline-infusion. Also further hydrocortisone was administered (Solu-Cortef® 250mg) along with a 1st generation antihistamine (Dexchlorpheniramine® 10mg).

IgE specific allergy testing taken 14min after arrival to the hospital showed S-IgE of 11KU/L (ref.<120KU/L), S-Tryptase >200ug/L (ref.<12ug/L), S-I1 0.37KU/L (bee) (ref.<0.35KU/L) and S-I3 (wasp) 1.05KU/L (ref.<0.35KU/L).

Initial CT-scan of head on arrival gave indications of diminished gray/white matter differentiation but no other intracranial pathologies were identified. The next 24hrs of care included an ongoing adrenaline-infusion. The patient suffered significant diarrhoea with negative OGD findings. Repeat CT-scan head after 48hrs showed worsening lack of gray/white matter differentiation indicative of severe hypoxic brain injury along with signs of transtentorial herniation. MRI brain on day 4 showed changes consistent with hypoxic ischaemic brain injury. EEG day 5 showed electro cerebral inactivity. Brain death testing done on day 6 showed ceased function and organ donation discussed with family.

3. Discussion

Anaphylaxis is the clinical manifestation of immediate hypersensitivity by triggering the release of mediators from tissue mast cells and circulating basophils. Death may occur suddenly through airway obstruction or irreversible vascular collapse. In 1902 Portier and Richet observed that injecting a previously tolerated sea anemone antigen into a dog produced a fatal reaction as opposed to the anticipated prophylaxis. They called this phenomenon “ana-

phylaxis". They observed two factors likely essential for anaphylaxis: increased sensitivity to a toxin after previous injection of the same toxin and an incubation period of at least 2 weeks to 3 weeks [1]. Richet was recognised as the founder of the new science of allergy and was awarded the Nobel Prize in 1913.

Anaphylaxis occurs in 30/100,000 population/year (mortality 1-2%) and is caused by foods (35%), drugs/ biologicals (25%), insect stings (15%), exercise (5%) or is idiopathic (20%). Onset of anaphylaxis to stings or allergen injections is usually rapid: 70% begin in < 20 minutes and 90% in < 40 minutes [2]. Fatal drug anaphylaxis may be increasing, but rates of fatal anaphylaxis to venom and food are stable [3].

In one series of anaphylactic deaths, 70% died of respiratory complications and 24% of cardiovascular failure [4]. Gastrointestinal manifestations include nausea, vomiting, intense diarrhoea (rarely bloody), and cramping pain in the abdomen. Neurological manifestations of confusion, dizziness, syncope, seizures, and loss of consciousness may occur as a results of cerebral hypoperfusion or as a direct toxic effect of mediator release [5].

Persistent, also referred to as protracted or recurrent, anaphylaxis last 5 hours to 48 hours despite therapy. The estimated rate of persistent anaphylaxis is 23% to 28% [6]. Protracted anaphylaxis and biphasic anaphylaxis cannot be predicted from the severity of the initial event [7].

July and August are the major wasp (Hymenoptera species) months in Norway.

Insect venom allergies are most commonly caused by bee stings and wasp stings. Unlike many other allergies, insect venom allergies can be treated with allergen-specific immunotherapy (desensitisation). The aim of allergen-specific immunotherapy is to prevent anaphylactic reactions or reduce their severity. Research [8] has shown that immunotherapy is very effective in the treatment of insect venom allergies. In the first few years after treatment, about 90% of people do not have anaphylactic reactions to insect stings.

The only other extant reported fatal case of anaphylaxis with mastocytosis is a case report in a 33-year-old female patient with indolent systemic mastocytosis and urticaria pigmentosa, who died of an anaphylactic reaction after a yellow jacket sting [9].

The peak tryptase level (typically within 1-2 hour of anaphylaxis onset) usually correlates with the severity of symptoms, particular with the nadir of mean arterial pressure [6]. Significant increase in tryptase is defined as 20% increase above baseline tryptase + 2ug/L. Larger releases of tryptase can be detected longer and have been reported to be elevated for many hours after severe anaphylaxis. Tryptase is elevated in individuals with excessive number of mast cells, such as in mastocytosis [10]. The measured tryptase level one hour and 50 minutes after incidens was >200 ug/L, after 8 hours it had decreased to 190 ug/L, and 14 hours later its level was 44,3ug/L. This is believed to be one of the highest ever recorded in Norway (personal communication with Haukeland Hospital, Bergen, Norway, reference laboratorium).

Systemic mastocytosis patients who had at least one episode of anaphylaxis seems to have lower baseline tryptase than those who had not, indicating that mast cell burden by itself is not a risk factor for anaphylaxis [11]. Furthermore, the anaphylaxis risk appeared to be bell-shaped with rising tryptase level. Up to a tryptase level of 40 μg/L, the anaphylaxis risk seems to increase, which decreases with tryptase greater than 40 μg/L [12].

One article [13] suggest that due to the high rate of severe reactions and the fatal case underscore the importance of adequate diagnostic sensitivity of IgE in patients with Indolent Systemic Mastocytosis (ISM). The sensitivity of IgE can be ameliorated by lowering the threshold to 0.17 kUA/L, retaining good specificity. They recommend IgE screening in all patients with ISM and discussing immunotherapy when Yellow Jacket wasp (genera Vespula) venom IgE exceeds 0.17 kUA/L [13].

Up to 20% of patients allergic to Hymenoptera venom are not protected by conventional Venom Immunotherapy (VIT) [14].

The proportion of patients with elevated baseline serum tryptase levels necessitates further investigation of a possible association between mastocytosis and potentially treatment failure of conventionally dosed VIT [14].

Despite the above we think that all patients with mastocytosis and anaphylaxis must be instructed about avoiding the responsible elicitors and should carry an emergency kit with adrenaline for self-application. In mastocytosis patients with anaphylaxis due to Hymenoptera stings, venom immunotherapy should be recommended for life [15-17].

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