



Case Report

Sustained ventricular tachycardia caused by anaphylactic reaction☆☆☆

Abstract

We report a 30-year-old man who developed severe anaphylactic reactions with sustained ventricular tachycardia after eating seafood. This case emphasizes the need for cardiac monitoring in patients with anaphylaxis to identify malignant ventricular arrhythmias early.

Anaphylaxis is the most severe form of allergic reactions and a life-threatening condition. Anaphylaxis involving cardiovascular system may cause hypotension, shock, cardiac arrhythmias, myocardial infarction, and cardiac arrest [1]. Such clinical diseases are not rare. In the United States, anaphylaxis can cause about 1500 deaths every year [2]. Hence, we should pay more attention to anaphylaxis, especially in the emergency department. Anaphylaxis is a systemic response to a specific allergen, including drugs, foods, insect stings, and physical factors. The anaphylactic reactions are usually induced by the mediators released from tissue mast cells and blood basophils. Numerous chemical mediators are released, including histamine, heparin, renin, platelet-activating factor, bradykinin, tumor necrosis factor, and several types of interleukins [3]. Mast cells are distributed extensively in human organs and tissues, and also in the human heart. The human cardiac mast cells are widely examined, especially within myocardial fibers, around blood vessels, and in the arterial intima [4]. The human heart can manifest to the mediators similar to the other organs and tissues. We report a 30-year-old man who developed severe anaphylactic reactions with sustained ventricular tachycardia (VT) after eating seafood.

A 30-year-old man with no underlying cardiovascular diseases presented to the emergency department of our hospital after eating seafood for 3 hours. He had an allergic history of seafood. After he ate the food, he experienced nausea, vomiting, and palpitation. During the clinical examination, auscultation revealed severe tachycardia, almost 180 beats/min. The body skin was flushed and full of urticaria. Blood pressure was 90/60 mm Hg, and the sound of the lung was clear. Electrocardiogram (Fig.) showed sustained VT. The patient received intravenous injection of 20 mg diphenhydramine and 10 mg dexamethasone, respectively. Then intravenous loading dose of amiodarone was given immediately with a maintenance dose of 1 mg/mL, whereby the clinical condition of the patient improved. One hour later, electrocardiogram monitoring showed that VT was converted to sinus rhythm with a heart rate of 95 beats/min and no abnormal ST-T changing. Electrolyte was normal, and creatinine was 99.7 $\mu\text{mol/L}$ (46–92

$\mu\text{mol/L}$); CK-MB and cTnl were also normal. The echocardiogram showed no abnormality.

In our case, a 30-year-old healthy man with allergic history had manifestation of severe anaphylactic reactions, especially sustained VT. If clinical diagnosis and treatment were delayed, it may result in a fatal outcome. The human heart as a reactive organ of anaphylaxis may have many manifestations such as tachycardia, hypotension, bradycardia, shock, T-wave inversion and ST depression in multiple leads, and cardiac arrest [5]. There are many chemical mediators that may cause these situations. The activated mast cell in the human heart can release significant quantities of renin [6]. Renin released from mast cell may activate the cardiac renin-angiotensin system, which may be responsible for cardiac arrhythmias. In addition, the cardiac mast cell can release more quantities of chymase than other tissues. It is shown that chymase has a potent angiotensin-converting enzyme activity and can convert angiotensin I into angiotensin II [7]. Histamine is one of the key point mediators that can induce cardiac arrhythmias. The arrhythmogenic actions of histamine were fully described by Wolff and Levi [8]. The authors reviewed the medical literature and concluded that arrhythmias in anaphylaxis were caused primarily by released histamine for 3 reasons. First, the arrhythmias involved above can be reproducible by the administration of exogenous histamine. Second, the extent, incidence, and duration of arrhythmias are proportional to the amount of histamine during anaphylaxis. Third, antihistamines can prevent the development of these arrhythmias.

Because anaphylaxis is a life-threatening reaction to many allergens, it is critical to avoid touching or eating them. However, numerous anaphylactic reactions are unanticipated and may lead to airway obstruction or cardiac diseases, so immediate management should be taken to prevent possible damage. Performing a focused examination and providing a stable airway and intravenous access are the critical initial steps. Administrations most commonly used to treat anaphylaxis are histamine H1 receptor antagonists and corticosteroids, especially epinephrine. The management of epinephrine is the most important treatment for anaphylaxis. It can activate the α_1 , β_1 , and β_2 adrenergic receptors, leading to bronchodilation, lower airway obstruction, increased peripheral vessel resistance, vasoconstriction, and increased cardiac inotropy. Now an commercially made auto-injector is preferred as emergency treatment. Intravenous fluid administration is also critical for patients with hypotension, especially if he/she has shows reaction to epinephrine. In adults, 2 L of normal saline and even more may be required because of vascular dilation and increased permeability [9].

As a whole, anaphylaxis as a systemic response to a specific allergen is life-threatening and needs more attention, especially in the emergency department. This case emphasizes the need for cardiac monitoring in patients with anaphylaxis to identify malignant ventricular arrhythmias early.

☆ Conflicts of interest: None.

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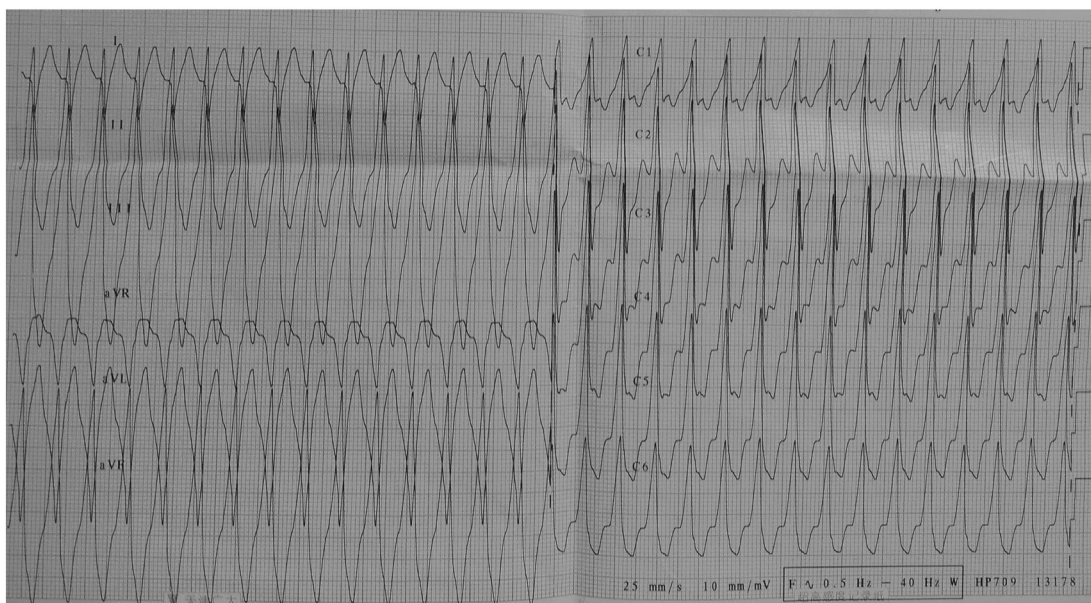


Fig. Admission electrocardiogram showing sustained VT.

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