Tako-tsubo cardiomyopathy (TTC) is a rare non-ischaemic, transient, reversible left ventricular dysfunction, which is often stress-related and is therefore also known as broken-heart-syndrome or stress cardiomyopathy. Due to the typical morphological changes seen in echocardiography (Fig. 1A) the condition was named “tako tsubo” (relating to the shape of Japanese octopus traps, Fig 1B) as well as “transient apical ballooning syndrome”. Clinical symptoms and electrocardiography (ECG) changes are suggestive of an anterior wall myocardial infarction. Besides emotional stress, exogenous catecholamines and rare cases of anaphylactic reactions have been reported as causes of TTC (1, 2). There is no specific therapy known for TTC, the prognosis is favourable, although left ventricular free wall rupture has been reported (3).

We report here the case of an 81-year-old man with mastocytosis who developed a life-threatening anaphylactic reaction after a Hymenoptera sting, followed by TTC.

CASE REPORT

Immediately after being stung by an unknown Hymenoptera, 0.3 mg adrenaline was injected intramuscularly by the patient. However, because of unconsciousness, absent pulses and gasping he was intubated and resuscitated. The ECG showed initially widened QRS complexes and a heart rate of 50 bpm. He was administered a second dose of adrenaline (1 mg, intravenously) as well as amiodarone 300 mg, prednisolone 250 mg, dimetindene 8 mg and ranitidine 150 mg. As ventricular fibrillation manifested he was defibrillated, which was successful after the second round. The patient recovered quickly and was extubated the next day. Because of persisting QT-interval prolongation and elevated troponin blood levels, the patient underwent a coronary catheterization and an echocardiography. The results showed a markedly decreased left ventricular output with apical wall motion hypokinesia, and no coronary artery disease. TTC was diagnosed.

The patient could not specify the insect responsible for the anaphylactic reaction. He reported two further anaphylactic reactions due to Hymenoptera stings several years previously. He had been prescribed an injection pen containing adrenaline. However, no allergological examinations had been performed.

Skin inspection revealed brown macula on the patient’s medial thighs and lower back which had not been noticed before. Cutaneous mastocytosis was diagnosed histologically. The basal serum tryptase level, measured six month after the anaphylactic reaction, was elevated (20.8 µg/l, normal 11.4 µg/l). There was no evidence of a systemic manifestation of the mastocytosis, including chest X-ray, abdominal ultrasound, oesophagogastroduodenoscopy, coloscopy and bone density measurement. A bone marrow biopsy was declined by the patient. Serum IgE levels were normal, but elevated venom-specific IgE antibodies to wasp (4.48 kU/l, normal < 0.35 kU/l) and bee (1.00 kU/l, normal < 0.35 kU/l) were detected. Recombinant allergen-based testing was performed to differentiate between a bee and/or wasp allergy: rVes v 5 (wasp) was elevated, with 4.75 kU/l (normal < 0.35 kU/l), r Api m 1 (bee) was negative, with 0.0 kU/l (normal < 0.35 kU/l). Intracutaneous testing showed a positive reaction to wasp venom and no reaction to bee venom up to a concentration of 1 µg/ml. The patient is currently undergoing desensitization to wasp venom.

DISCUSSION

Mastocytosis is a well-known risk factor for severe anaphylactic reactions, especially in elderly patients (4, 5). Therefore, patients with a history of anaphylaxis should be examined for mastocytosis, including measurement of serum tryptase levels (6).

In cases of severe anaphylactic reactions, such as was seen in our patient, catecholamine administration is required. Exogenous catecholamines, as well as rare cases of anaphylactic reactions, have been reported as causes of TTC (1, 2). It remains unclear whether our patient developed the TTC due to the adrenaline that was administered, or because of the severe anaphylaxis, which is a stressful event itself. However, with regard to the course of the ECG changes, initially already showing widened QRS complexes, the anaphylaxis is the more likely reason. After the patient received adrenaline he developed ventricular fibrillation, which probably aggravated the stress situation. It is possible that the combination of anaphylaxis and adrenaline application was decisive. Allergologists should be aware of TTC, which may follow anaphylaxis.

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