

Acute Myocardial Ischemia Following Bee Sting in an Adolescent Male: A Case Report

Authors' Contribution:
Study Design A
Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
Literature Search F
Funds Collection G

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Conflict of interest: Publication support in part through ROAPP Grants through the University of Illinois

Patient: Male, 16-year-old
Final Diagnosis: Anaphylaxis
Symptoms: Chest pain • pulmonary edema
Medication: —
Clinical Procedure: —
Specialty: Critical Care Medicine

Objective: Unusual clinical course
Background: Epinephrine for anaphylactic shock is the standard life-saving treatment in the emergency department. Cardiac symptoms after epinephrine administration in a child with no prior cardiac history are often not suspected. We describe a presentation of diastolic cardiac dysfunction after anaphylaxis from a bee sting in an adolescent male.
Case Report: A 16-year-old male with no prior history of allergy presented with anaphylaxis following a bee sting. The patient received an inadvertent intravenous rather than intramuscular dose of 1: 1000 epinephrine, leading to myocardial ischemia. Diastolic dysfunction resulting from myocardial ischemia and fluid resuscitation led to development of pulmonary edema. The patient required epinephrine drip for hemodynamic support and BiPAP for respiratory support.
Conclusions: This case highlights the risk of giving a rapid intravenous push of epinephrine, which converted an anaphylactic reaction to cardiogenic shock. Anaphylaxis-related coronary ischemia (Kounis) syndrome is another less likely etiology for our patient's presentation.

MeSH Keywords: Anaphylaxis • Emergencies • Intensive Care Units, Pediatric

Full-text PDF: <https://www.amjcaserep.com/abstract/index/idArt/922120>

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Background

Anaphylaxis and anaphylactic shock are very frequent presentations in any emergency department. Epinephrine steroids and volume resuscitation are standard management strategies with excellent response rates [1]. Cardiac symptoms after anaphylaxis or after treatment with epinephrine are, however, not often suspected. In this case report, we describe an unusual presentation of diastolic cardiac dysfunction after bee sting anaphylaxis and treatment with intravenous epinephrine.

Case Report

A 16-year-old male (weight 56.7 Kg) with a past medical history of attention deficit hyperactivity disorder, presented to the Emergency Department (ED) after being stung by an insect (possibly a bee) behind his right ear. His initial symptom was swelling at the site and “feeling weird”. There were no complaints of chest pain or shortness of breath. EMS was called, and paramedics placed an IV and gave the patient 50 mg of IV Benadryl and inadvertently administered 0.3 mg of 1: 1000 epinephrine by rapid push. Initial emergency vital signs were systolic blood pressure 103/69 mmHg and heart rate 105. He was sleepy but arousable. He received an additional 20 mL per kilo fluid for hypotension, as well as H2 blocker and steroids. Shortly after arrival at the ED, he reported feeling chest pain and had an EKG done, which showed T wave inversion in inferior leads, suggestive of acute ischemia (Figure 1).

He received aspirin based on the EKG. The initial troponin was 0.169 ng/mL and brain natriuretic peptide (BNP) was 13 pg/mL. The patient became progressively hypotensive in the ED and received an additional 20 mL/kg crystalloid (lowest BP recorded 78/58). The peripheral exam was significant for cold extremities with poor perfusion. A bedside echo done in the ED showed qualitatively normal systolic function. He also became progressively hypoxemic and required high-flow nasal cannula of up to 40 L 100% oxygen to maintain oxygen at equal to or more than 90%. A chest x-ray was done, which showed pulmonary edema (Figure 2). Peripheral venous blood gas showed pH 7.22/PCO₂ 52.6/BD -6 and lactate of 3.7 mmol/L. Because of persisting hypotension, cold extremities, and pulmonary edema, an epinephrine drip was started, which was up-titrated up to 0.1 mcg/kg/min. An echo done on epinephrine of 0.1 mcg/kg/min showed a dilated left atrium with an overall normal left ventricle systolic function (EF Teicholz: 62.9%) and relatively hyperdynamic apical segments but normal contractility of basal segments in the presence of tachycardia, moderate mitral valve regurgitation, and abnormal inflow Doppler to the mitral valves with fused E and A waves suggesting diastolic dysfunction. The origins of coronary arteries were normal (Videos 1, 2). Because of the significant hypoxemia, the patient was switched to non-invasive BiPAP ventilatory support of 18/8. His serum lactate level peaked at 5.0 mmol/L, 6 h after the bee sting, and troponin peaked at 1.708 ng/mL (12 h after the bee sting). Our patient’s clinical shock fluctuated between “cold” and “warm” in the initial hours of therapy, although exact systemic vascular resistance (SVR) determination was not

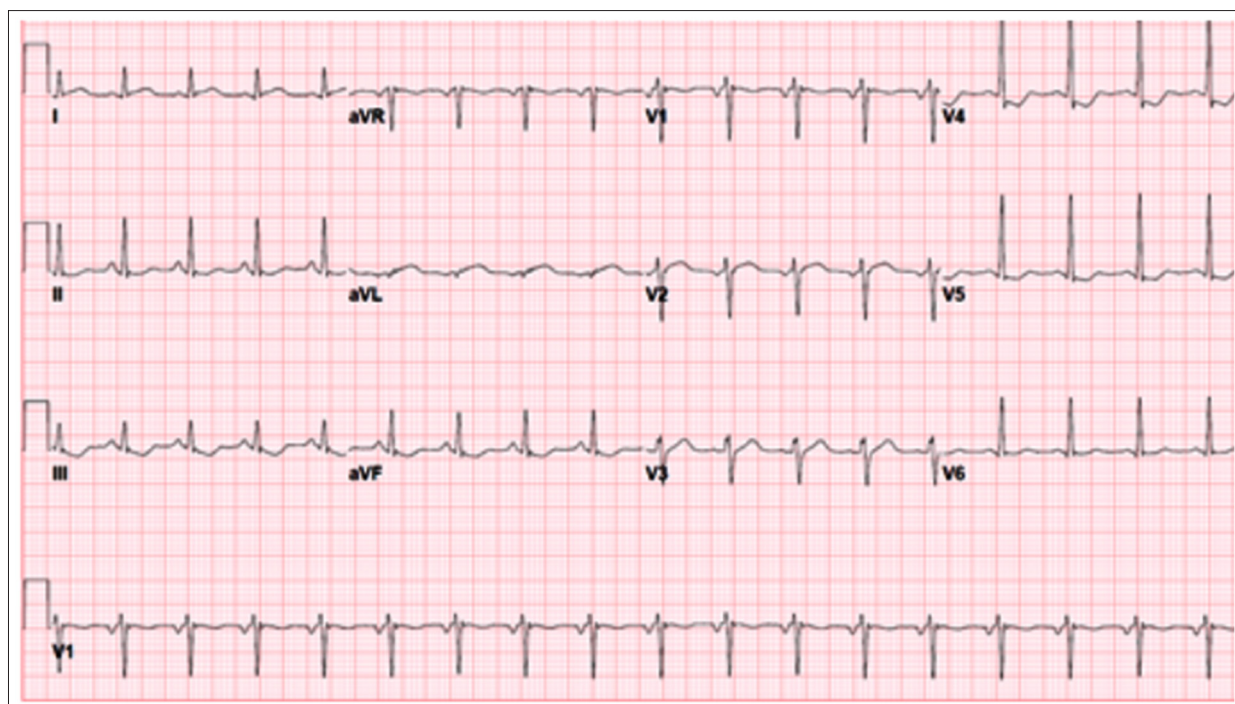


Figure 1. 12-lead EKG on admission: Sinus tachycardia 105 bpm. T wave inversion in inferior leads suggestive of acute ischemia.

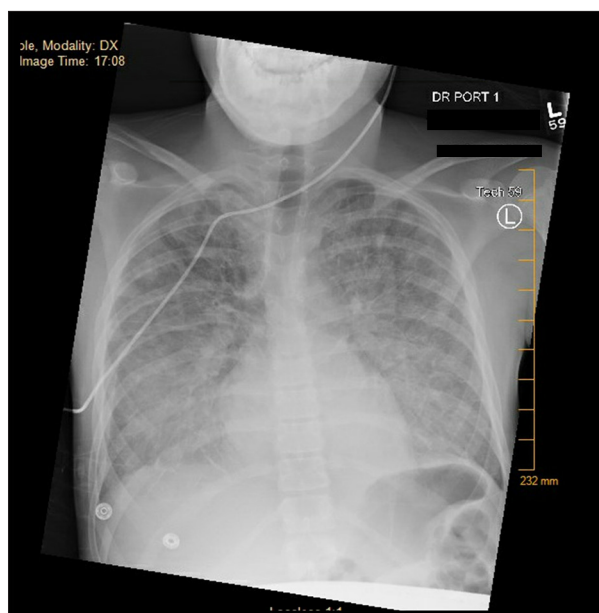


Figure 2. CXR showing pulmonary edema.

possible due to lack of invasive monitoring. Overnight, the patient gradually recovered normal blood pressure. Epinephrine was weaned off by the morning. A repeat EKG done 1 day after admission showed resolution of T wave inversion (Figure 3). The pulmonary edema gradually resolved over 2 days. A repeat echo done the next day and EKG done 3 days after admission showed normalization of systolic and diastolic left ventricle function while off inotropic support and improved mitral regurgitation. The patient was discharged on hospital admission day 4 with a prescription of EpiPen. The time sequence of events is shown in Figure 4.

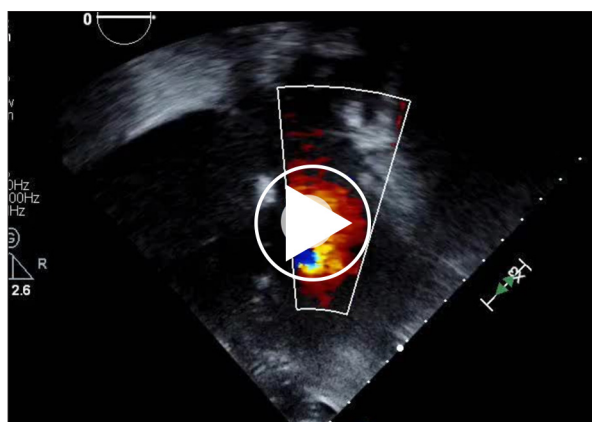
Discussion

We have described a case of anaphylaxis with a bee sting, which was complicated by an inadvertent intravenous administration of 1: 1000 epinephrine at standard dosing. The intravenous injection of the intramuscular concentration of epinephrine led to myocardial ischemia and diastolic dysfunction. The resulting hypotension and fluid resuscitation lead to development of pulmonary edema.

Epinephrine given intramuscularly or subcutaneously is the standard life-saving treatment for anaphylaxis. Intravenous epinephrine has also been advocated in small doses (0.002 mg/kg) for hypotension and 0.01 mg/kg for the presence of cardiovascular collapse [1]. Intravenous epinephrine is associated with more complications compared to the intramuscular route. Campbell et al. studied 573 emergency department patients treated for anaphylaxis, finding that adverse effects included arrhythmia, cardiac ischemia, stroke, angina, and hypertension.



Video 1. Four-chamber view of the LV systolic function, showing good function, without evidence of regional wall motion abnormality.



Video 2. ECHO showing moderate mitral regurgitation.

The incidence of the adverse effects was significantly higher in patients who received intravenous vs. intramuscular administration (10% vs. 1.3%, $p=0.006$). Similarly, overdose occurred with 13.3% of the IV bolus compared to 0% of the intramuscular doses [2]. Intravenous epinephrine should always be given diluted to 1: 10 000 or higher and should always be given slowly [3]. Rare reports of iatrogenic epinephrine overdose [4] describe myocardial stunning with akinetic basal and mid segments of the left ventricle but hyperdynamic apical segments, and severely abnormal diastolic function. It is associated with relatively modest elevation of biochemical markers of myocardial necrosis. Increased myocardial oxygen demand and decreased supply (with hypotension related to anaphylaxis) complicates myocardial oxygen delivery, leading to ischemia. The increased left ventricle filling pressure leads to pulmonary venous hypertension, and fluid exudates into the alveolar space, resulting in pulmonary edema [5]. Sporadic case reports of epinephrine administered for treatment of anaphylaxis leading to myocardial infarction have been reported both in the adult and pediatric literature, although more commonly in adults with pre-existing coronary vascular disease [3,6].

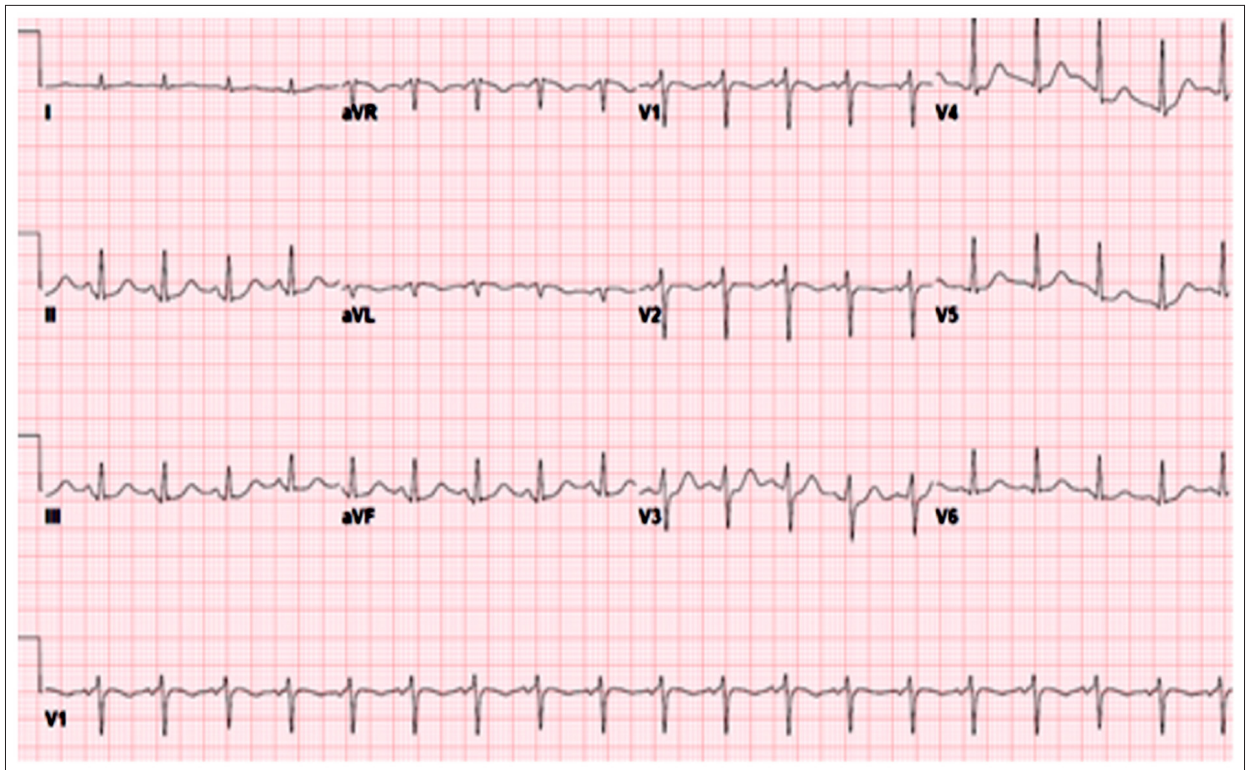
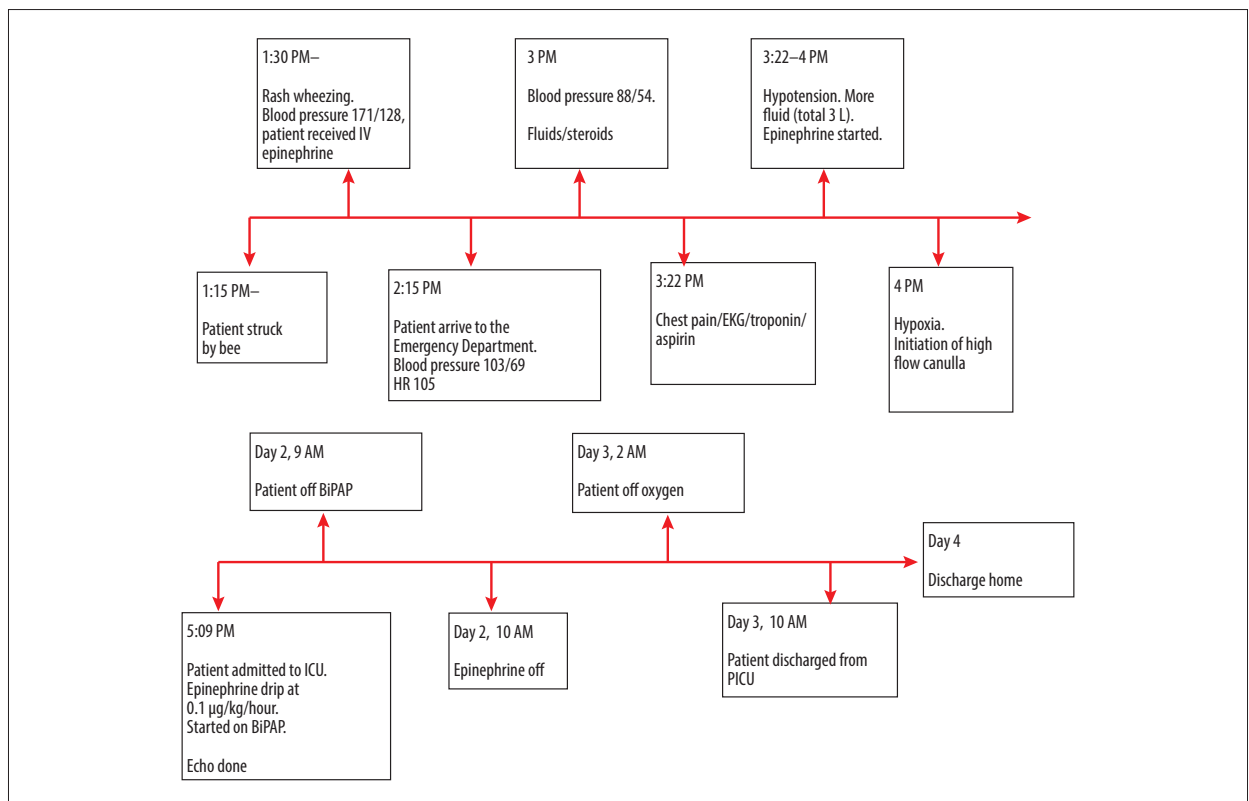


Figure 3. 12-lead ECG 1 day after admission. Sinus tachycardia 115 bpm. T wave inversion in inferior leads is now resolved. QTc prolongation 460 ms. (QTc normalized on discharge ECG).



A case report of myocardial infarction after nebulized epinephrine for croup in an 11-year-old boy has also been described [7].

It is also possible that the anaphylaxis itself leads to coronary ischemia. Acute myocardial infarction after a wasp sting has also been described in the literature [8]. Nicholas Kounis first described a syndrome of “allergic angina” or coronary spasm; this has been termed Kounis syndrome [9]. This represents a manifestation of endothelial dysfunction or microvascular angina and sometimes progresses to an allergic acute myocardial infarction, and may be related to histamine- and leukotriene-induced coronary vasoconstriction, as well as platelet activation [10]. All factors that can trigger anaphylaxis can also trigger Kounis syndrome, including several kinds of food, drugs, and environmental exposures. Even prednisolone given for the treatment of anaphylaxis has been shown to cause acute myocardial infarction resembling Kounis syndrome [11]. Three variants of this syndrome have been described. Type I variant (coronary spasm) includes patients with healthy or near-normal coronary arteries, Type II variant includes patients with pre-existing atheromatous disease, and Type III variant includes patients with coronary artery stent thrombosis in the setting of anaphylaxis [12]. It is possible that clinical ischemia with anaphylaxis is often unrecognized. A prospective study of 138 911 emergency department patients showed Kounis syndrome had a prevalence rate of 19.4/100 000, and 3.4% of allergy patients had Kounis syndrome [13].

Our patient either had an anaphylaxis-related coronary ischemia or coronary ischemia related to an intravenous dose of epinephrine. However, since our patient did not report chest pain prior to receiving epinephrine, it is more likely that his coronary ischemia was related to epinephrine overdose rather than Kounis syndrome. Also, echocardiography showed no

segmental wall motion abnormalities corresponding to coronary distribution seen in Kounis syndrome, but it showed a relatively hyperdynamic apex compared to basal segments, which is more characteristic of epinephrine overdose. In retrospect, it can be argued that most of the morbidity with this patient was iatrogenic. The patient had an allergic reaction to a bee sting; however, a later IV dose of epinephrine may have caused him to develop cardiogenic shock. This was unrecognized at that time, and he was treated for anaphylactic shock, at least initially, with volume resuscitation. In the setting of diastolic dysfunction with coronary ischemia, volume resuscitation leads to pulmonary edema.

Conclusions

Our case demonstrates the consequences of an intravenous epinephrine overdose leading to severe diastolic dysfunction of the left ventricle, which converted an anaphylactic reaction to cardiogenic shock. It is essential to be cognizant of this possibility in patients with anaphylaxis who received multiple doses of epinephrine. Kounis syndrome should also be considered in patients with anaphylaxis presenting with chest pain, as misdiagnosis of this condition can be fatal and a high index of suspicion is of paramount importance.

Department and Institution where work was done

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Conflict of interest

None.

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