



Letter to the Editor

Anaphylactic shock-associated cardiomyopathy

Maximilian Hartmann, Christoph Bode, Andreas Zirlik*

Department of Cardiology and Angiology, University of Freiburg, Breisacherstrasse 33, 79106 Freiburg, Germany

Received 29 January 2007; accepted 23 April 2007

Abstract

Acute heart failure in young people may have other etiologies than myocarditis. We report about a twenty-year-old female developing acute heart failure following an anaphylactic reaction after a Prick test. Initially her left ventricular ejection fraction was severely compromised on echocardiography but normalized quickly. We postulated an anaphylactic shock-associated cardiomyopathy due to the close temporal association to an anaphylactic reaction, the presence of hypereosinophilia, and the absence of specific serologic, microbiologic, and immunologic abnormalities. Anaphylactic-associated cardiomyopathy very likely represents a common cause of acute heart failure and should be thought of in young people with known atopy.

© 2007 Elsevier Ireland Ltd. All rights reserved.

Keywords: Acute heart failure; Anaphylactic shock; Myocarditis; Case report

A twenty-year-old female underwent a Prick test showing strong reactions on several allergens in an outpatient dermatology service. After the test she complained of dyspnea and rapidly developed an anaphylactic reaction with hypotension, tachycardia, and vomiting. Administration of 0.3 mg epinephrine, 250 mg prednisone, and 1000 ml NaCl resulted in complete relief of symptoms. An hour later, the patient went home in stable cardio-respiratory condition. In the afternoon, dyspnea, nausea, and weakness recurred resulting in admission to a community hospital. Physical examination revealed a presystolic gallop and discrete pulmonary crepitations. The ECG showed sinus tachycardia (120 bpm) with normal repolarisation. The chest X-ray displayed signs of mild pulmonary edema. Arterial blood gas analysis revealed hypoxemia (PaO₂ 61 mm Hg, SaO₂ 92%) and mild metabolic acidosis with respiratory compensation. Laboratory testing showed an elevated Troponin T (1.42 ng/dl) and leukocytosis (16,300/μl). Left ventricular ejection fraction was severely compromised (20–25%) with posterior-lateral akinesia as assessed by echocardiography. Subsequently, the patient was transferred by helicopter to the cardiac intensive care unit at our center. Shortly after arrival her

circulation further weakened requiring dobutamine treatment. Pro-BNP rose to 4273 pg/ml and differential blood count showed hypereosinophilia (9%).

On detailed questioning, the patient disclosed a tick bite 10 days prior to the Prick test. She had it inspected by a general practitioner at this time. No subsequent erythema developed. We started an antibiotic regimen with intravenous administration of 2 g ceftriaxone per day. During the following 42 h at our ICU the patient quickly stabilized hemodynamically and could be weaned of catecholamines. A second echocardiography showed an improved moderately reduced LV ejection fraction (35–40%) with global hypokinesia.

After transfer to a regular cardiac ward, an ace-inhibitor and betablocker was initiated. Her condition further improved over the next few days and a third echocardiography showed a near-normal LV ejection fraction (50%). WBC, Troponin T-, and pro-BNP levels quickly normalized. Extensive serologic and immunologic testing revealed no abnormalities. Antibiotic therapy with ceftriaxone was discontinued after two consecutive negative serologies for borrelia. A cardiac multi-slice computed tomography and magnetic resonance tomography excluded morphologic abnormalities of the coronary arteries and showed normal cardiac structure and function. The patient was discharged in excellent condition and returned for follow-up visits after one and four weeks. She reported normal

* Corresponding author. Tel.: +49 761 270 7059; fax: +49 761 270 7045.

E-mail address: andreas.zirlik@uniklinik-freiburg.de (A. Zirlik).

Table 1
Differential diagnosis of temporary heart failure in young people

Myocarditis	Viral (adeno- and enteroviruses)
	Bacterial (mycobacteria)
	Parasitic (toxoplasmosis, trypanomiasis)
	Immunologic (SLE)
Myocardial infarction	Allergic/toxic (hypereosinophilic syndrome)
	Congenital coronary anomalies
	Spontaneous dissection of coronary arteries
	Drug abuse (cocaine)
Anaphylactic shock-associated cardiomyopathy	Vasculitis (Takayasu arteriitis)
	Allergen-mediated type 1 reaction

physical strength and the absence of symptoms. Examination was unremarkable and follow-up echocardiography, stress echocardiography, and MRI were normal.

1. Discussion

Temporary heart failure in young people may have several reasons. Table 1 summarizes the major entities. Clinical presentation may vary and includes symptoms of forward failure, such as weakness, syncope, or dizziness as well as signs of backward failure such as dyspnea, pulmonary edema, or cyanosis. In young people infectious myocarditis is the leading cause of heart failure. Viral infection most commonly represents the underlying disorder in Westernized countries. The most frequently identified viruses are adenoviruses and enteroviruses. In contrast, in developing countries non-viral infections with e.g., *Trypanosoma cruzi* or HIV-associated opportunistic infections with toxoplasma gondii and mycobacteria play the dominant etiologic role. In rare cases, myocarditis may be found in immunologic disorders such as systemic lupus erythematoses (SLE) or in hypereosinophilic syndrome, e.g. following administration of certain drugs. Cocaine abuse, vasculitis (e.g., Takayasu arteriitis), and spontaneous dissection or congenital abnormalities of the coronary arteries may trigger acute heart failure when leading to acute myocardial infarction.

In our patient, infectious myocarditis had to be considered. Since she suffered a tick bite ten days earlier, cardiac bor-

reliosis was possible. However, we did not detect typical serologic changes and the onset of symptoms was very sudden and clearly associated with an anaphylactic reaction.

Anaphylaxis is a severe systemic response initiated by exposure to a specific antigen in a sensitized organism. The cause of acute circulatory failure is controversial. It may be due to a sudden fall of arterial pressure caused mainly by decreased systemic vascular resistance or due to an increase in resistance in the pulmonary vascular system resulting in a decreased left ventricular preload [1]. In addition to that, previous studies in rats and dogs suggested direct myocardial impairment, potentially caused by direct effects of mediators such as leukotrienes, histamines, and platelet-activating factors [2,3]. Indeed, there is clinical evidence that allergic reactions may lead to acute heart failure in humans. Ito and Ohata reported the case of a 14-year-old girl with sudden onset of severe atopic dermatitis followed by temporary acute heart failure with pulmonary congestion [4]. As in our case laboratory testing showed a leukocytosis with hypereosinophilia suggesting that the mechanism may be similar to those in hypereosinophilic syndrome. The hypereosinophilia, the clear temporal association with an anaphylactic reaction, the rapid clinical course, and the absence of specific serologic, microbiologic, and immunologic abnormalities render it most likely that our patient suffered from anaphylactic shock-associated cardiomyopathy.

Anaphylactic shock-associated cardiomyopathy is a disorder one should think of in patients with known atopy presenting with acute symptoms of heart failure.

References

- [1] Mitsuhashi H, Hasome N, Saitoh J, Takeuchi H, Horiguchi Y, Shimizu R. Evaluation of left ventricular diastolic function in IgE-mediated anaphylaxis in dogs. *Acta Anaesthesiol Scand* 1995;39(6):791–6.
- [2] Correa E, Mink S, Unruh H, Kepron W. Left ventricular contractility is depressed in IgE-mediated anaphylactic shock in dogs. *Am J Physiol* 1991;260(3 Pt 2):H744–51.
- [3] Sun S, Weil MH, Tang W, et al. Cardiac anaphylaxis in the Sprague–Dawley rat. *J Lab Clin Med* 1992;120(4):589–96.
- [4] Ito T, Ohata T. Acute myocarditis with transient eosinophilia and serum hyper-IgE-emia in a patient with atopic dermatitis. *Heart Vessels* 2001;16(1):28–31.