

CASE REPORT

Allergic angina following wasp sting: Kounis syndrome

Prem Krishna Anandan*, Natesh B. Hanumanthappa, Prabhavathi Bhatt,
and Manjunath Nanjappa Cholenahally

Department of Cardiology, Sri Jayadeva Institute of Cardiovascular Research and Sciences,
Bangalore, Karnataka, India

*Correspondence address. Sri Jayadeva Institute of Cardiovascular Research and Sciences, Room No. 107, Doctors Accommodation, Bannerghatta Road, Jayanagar 9th Block, Bangalore, Karnataka 560069, India. Tel: +91-8861661048; Fax: +91-80-2297-7400; E-mail: premkrishna2k1@gmail.com

Abstract

Kounis syndrome is defined as an acute coronary syndrome triggered by the release of inflammatory mediators following an allergic insult characterized by acute onset of breathlessness, palpitations, diaphoresis and chest tightness (Gázquez V, Dalmau G, Gaig P, Gómez C, Navarro S, Mercé J. Kounis syndrome: report of 5 cases. *J Investig Allergol Clin Immunol* 2010;20:162–5). We report a 20-year-old male patient who presented with acute myocardial infarction following a wasp sting at our institute. ECG showed non-ST-elevation myocardial infarction. Symptoms settled with hydrocortisone and adrenaline. Coronary angiogram revealed normal epicardial coronaries.

INTRODUCTION

Myocardial infarction secondary to anaphylaxis is known as Kounis syndrome. Varied etiology has been described in the literature. Physicians should be aware of this entity since thrombolysis and percutaneous coronary intervention can be avoided in such situations. Serious allergic reactions may be the cause of acute coronary syndrome in patients with healthy or altered coronary arteries and no cardiovascular risk factors.

CASE REPORT

A 20-year-old man with no cardiovascular risk factors sustained a wasp sting over his left forearm. Fifteen minutes later, he experienced sudden shortness of breath, feeling of instability, palpitations, chest tightness and excessive sweating. On admission at our institute, patient was hypotensive (80/60 mmHg) with an arterial oxygen saturation of 85%. He was given 1 mg of subcutaneous adrenaline (1 : 1000) and 100 mg of intravenous hydrocortisone. The electrocardiogram revealed T-inversion in leads I, aVL, II, aVF and V1–V6 (Fig. 1). Echocardiogram showed anterior wall hypokinesia with an LVEF of 40%. Troponin T at admission was

elevated (1.1 ng/ml; ref. range <0.01 ng/ml). Following administration of adrenaline and corticosteroids, the patient became hemodynamically stable. Coronary angiogram (Fig. 2) after 8 h revealed normal epicardial coronaries. After 18 hours of admission abnormalities reverted to normal (Fig. 3) and the ejection fraction improved to 55% with no regional wall motion abnormality of anterior wall.

DISCUSSION

Kounis syndrome or allergic was first described in 1991 by Kounis and Zafras [1] as 'the coincidental occurrence of chest pain and allergic reactions accompanied by clinical and laboratory findings of classic angina pectoris caused by inflammatory mediators released during the allergic insult'. They called the progression from chest pain to acute myocardial infarction 'allergic myocardial infarction' [2, 3]. This occurs during episodes of anaphylaxis [4], and frequently in patients with prior coronary disease, although it has also been observed in patients with healthy coronary vessels. The main mechanism implicated is vasospasm of the coronary arteries [5].

Received: April 26, 2015. Revised: May 12, 2015. Accepted: May 28, 2015

© The Author 2015. Published by Oxford University Press.

This is an Open Access article distributed under the terms of the Creative Commons Attribution Non-Commercial License (<http://creativecommons.org/licenses/by-nc/4.0/>), which permits non-commercial re-use, distribution, and reproduction in any medium, provided the original work is properly cited. For commercial re-use, please contact journals.permissions@oup.com



Figure 1: ECG at admission showing deep T-inversion in anterior leads.

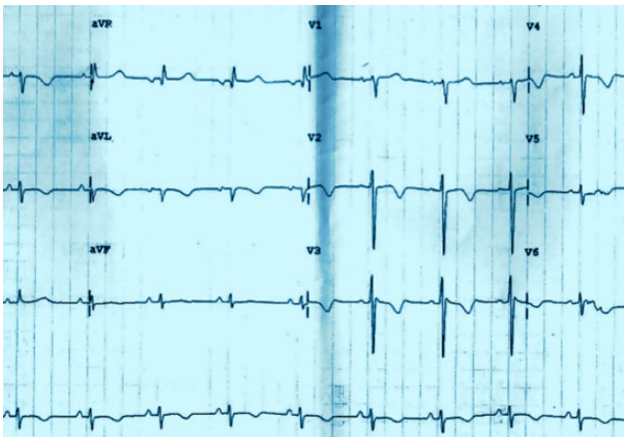


Figure 2: Coronary angiogram showing normal epicardial coronaries.

Three variants of syndrome have been described:

- (i) *Type I variant*—This applies to patients with normal or nearly normal coronary arteries without predisposing factors for coronary artery disease, and the acute mediator release can induce either coronary artery spasm [6] or coronary artery spasm progressing to acute myocardial infarction.
- (ii) *Type II variant*—This applies to patients with culprit, quiescent pre-existing atheromatous disease, and the mediator release can induce either coronary artery spasm or coronary artery spasm together with plaque erosion or rupture manifesting as acute myocardial infarction.
- (iii) *Type III variant* is seen in patients with stent thrombosis in whom thrombus harvested and stained with hematoxylin-eosin and Giemsa that show eosinophils and mast cells, respectively. Type III variant is diagnosed in patients with stent implantation who died suddenly and histological examination of coronary intima, media or adventitia adjacent to the stent is infiltrated by eosinophils and/or mast cells.

Following an allergic insult, arachidonic acid cascade is activated; histamine is released leading onto a series of functional and metabolic changes in the heart, termed as cardiac anaphylaxis [7, 8]. Authors have also described the role of renin released during anaphylaxis in causing cardiac dysfunction [9]. Mueller [10]



Figure 3: ECG after 18 hours showing almost normalization of T-inversions.

has reported the role of adrenaline in causing Kounis syndrome. Elevated serum tryptase will aid in differentiating Kounis syndrome from routine forms of acute coronary syndrome, provided that the blood sample is drawn between the first and second hour after onset of symptoms.

CONCLUSIONS

Kounis syndrome is not a rare disease but an infrequently diagnosed condition.

It should be borne in mind when diagnosing patients with no cardiovascular risk factors who experience acute coronary syndrome accompanied by symptoms of anaphylaxis.

CONFLICT OF INTEREST STATEMENT

None declared.

REFERENCES

1. Kounis NG, Zavras GM. Histamine-induced coronary artery spasm: the concept of allergic angina. *Br J Clin Pract* 1991; 45:121–8. Review.
2. Kounis NG, Zavras GM. Allergic angina and allergic myocardial infarction. *Circulation* 1996;94:1789.
3. Kounis NG, Grapsas GM, Goudevenos JA. Unstable angina, allergic angina and allergic myocardial infarction. *Circulation* 1999;100:e156.

4. Moneret-Vautrin DA, Morisset M, Flabbee J, Beaudouin E, Kanny G. Epidemiology of life-threatening and lethal anaphylaxis: a review. *Allergy* 2005;**60**:443–51.
5. Brener Z, Zhuravenko I, Bergman M. Acute myocardial injury following penicillin-associated anaphylactic reaction in a patient with normal coronary arteries. *Am J Med Sci* 2007;**334**:305–7.
6. Kounis NG. Kounis syndrome (allergic angina and allergic myocardial infarction): a natural paradigm? *Int J Cardiology* 2006;**110**:7–14.
7. Gázquez V, Dalmau G, Gaig P, Gómez C, Navarro S, Mercé J. Kounis syndrome: report of 5 cases. *J Investig Allergol Clin Immunol* 2010;**20**:162–5.
8. Bani D, Nistri S, Mannaioni PF, Masini E. Cardiac anaphylaxis: pathophysiology and therapeutic perspectives. *Curr Allergy Asthma Rep* 2006;**6**:14–9.
9. Reid A, Silver R, Levi R. Renin: at the heart of the mast cell. *Immunol Rev* 2007;**217**:123–40.
10. Mueller U. Cardiovascular disease and anaphylaxis. *Curr Opin Allergy Clin Immunol* 2007;**7**:337–41.