

A case of type I variant Kounis syndrome with Samter-Beer triad

Jayesh S Prajapati, Kapil M Virpariya, Ashok S Thakkar, Atul D Abhyankar

Jayesh S Prajapati, Kapil M Virpariya, Department of Cardiology, UN Mehta Institute of Cardiology and Research Center, Ahmedabad 380016, Gujarat, India

Ashok S Thakkar, Department of Clinical Trials, Sahajanand Medical Technologies Pvt. Ltd., Surat 395004, India

Atul D Abhyankar, Department of Cardiology, Shree BD Mehta Mahavir Heart Institute, Athwagate, Surat 395001, India

Author contributions: Prajapati JS, Virpariya KM, Thakkar AS and Abhyankar AD designed the research and wrote the paper; Prajapati JS and Virpariya KM performed the research.

Correspondence to: Jayesh S Prajapati, MD, DM, Associate Professor of Cardiology, Department of Cardiology, UN Mehta Institute of Cardiology and Research Center, BJ Medical College and Civil Hospital Campus, Asarwa, Ahmedabad 380016, Gujarat, India. drjsprajapati@yahoo.co.in

Telephone: +91-79-26464343 Fax: +91-79-22682092

Received: January 27, 2013 Revised: March 4, 2013

Accepted: March 15, 2013

Published online: April 26, 2013

Core tip: When there is a young individual with no predisposing factors of atherosclerosis and apparent coronary lesion, with or without electrocardiography and biochemical markers of infarction, the possibility of Kounis syndrome should be kept in mind. In such a situation, intracoronary vasodilators, nitrates, nicorandil or diltiazem should be used before proceeding with a coronary intervention. An urgent eosinophil count should be done before proceeding with a coronary intervention to rule out coronary spasm.

Prajapati JS, Virpariya KM, Thakkar AS, Abhyankar AD. A case of type I variant Kounis syndrome with Samter-Beer triad. *World J Cardiol* 2013; 5(4): 112-114 Available from: URL: <http://www.wjgnet.com/1949-8462/full/v5/i4/112.htm> DOI: <http://dx.doi.org/10.4330/wjc.v5.i4.112>

Abstract

Kounis syndrome is defined as the coexistence of acute coronary syndromes with situations associated with allergy or hypersensitivity, as well as anaphylactic or anaphylactoid reactions, to a variety of medical conditions, environmental and medication exposures. We report a case of Kounis-Zavras syndrome type I variant in the setting of aspirin-induced asthma, or the Samter-Beer triad of asthma, nasal polyps and aspirin allergy. When there is a young individual with no predisposing factors of atherosclerosis and apparent coronary lesion, with or without electrocardiography and biochemical markers of infarction, the possibility of Kounis syndrome should be kept in mind.

© 2013 Baishideng. All rights reserved.

Key words: Kounis syndrome; Samter-Beer triad; Nasal polyps; Coronary spasm; Aspirin allergy

INTRODUCTION

Kounis syndrome is defined as the coexistence of acute coronary syndromes with situations associated with allergy or hypersensitivity, as well as anaphylactic or anaphylactoid reactions, to a variety of medical conditions, environmental and medication exposures. Patients undergoing stent implantation receive several substances which have antigenic properties. Many etiologies have been reported^[1,2], including drugs (antibiotics, analgesics, antineoplastics, contrast media, corticosteroids, intravenous anesthetics, non-steroidal anti-inflammatory drugs, skin disinfectants, thrombolytics, anticoagulants), various conditions (angioedema, bronchial asthma, rhinitis, nasal polyp, urticaria, food allergy, exercise-induced allergy, mastocytosis, serum sickness), environmental exposure (stings of ants, bees, wasps and jellyfish, grass cuttings, millet allergy, poisoning, latex contact, eating shellfish, viper venom poisoning) and stent implantation (nickel, chromium, manganese, titanium, molybdenum, polymers), which can induce allergy,

either separately or synergistically^[3].

CASE REPORT

A 19 year old male presented with history of dyspnoea. Over the past 2 d, he had chest pain and the first episode of syncope and was admitted to hospital. On physical examination, he was sweaty but hemodynamically stable. He had no family history of coronary artery disease. Electrocardiography (ECG) showed ventricular tachycardia (VT). On arrival, his Troponin-T was elevated (0.26 ng/mL, reference range: 0-0.03 ng/mL). A diagnosis of non-ST segment myocardial infarction with complete heart block was made. Cardiac catheterization demonstrated 99% lesion in mid right coronary artery (RCA). The ejection fraction was 60%. The percutaneous coronary intervention was performed *via* the right femoral artery access route. A sirolimus-eluting stent (3.5 mm × 23 mm) was deployed with an excellent angiographic result. The patient was discharged on the fifth day.

Two months later, the patient was again hospitalized with a second episode of syncope arrest and chest pain. In the brain magnetic resonance image, no significant focal intracranial abnormality was detected. An absolute eosinophil count was 645 cumm/ μ L (reference range: 40-440 cumm/ μ L). Check angiogram revealed a well flowing RCA stent with 40% *de novo* lesion. The remaining coronary arteries were normal. The patient was recommended for medical management and was discharged on the seventh day.

Five months later, the patient had a 3rd episode of syncope, and hence was rehospitalized for clinical evaluation with twenty-four hour electrocardiographic (Holter) monitoring, which was normal. At this time, ECG and echocardiography were normal. Patient developed VT during hospitalization which was reverted with direct current shock and beta blockers were started. During the hospital stay, on the 3rd day, the patient had chest pain again. Troponin-I was positive. A 12-lead electrocardiogram showed ST elevation in anterior leads, suggestive of hyper acute stage of anterior wall myocardial infarction. The patient was transferred to the intensive cardiac care unit and there the ST elevation disappeared. Echocardiography showed mid apical septum and apex hypokinesia. Glycoprotein IIb/IIIa inhibitors were given.

DISCUSSION

When the fourth check angiography was done, it revealed a mid to distal left anterior descending (LAD) 90% discrete lesion with sluggish flow and a well flowing RCA stent. The patient was taken for percutaneous transluminal coronary angioplasty (PTCA) to LAD after 3 d. During coronary angiography, prior to PTCA, another 80% lesion proximal to previous 90% lesion was revealed where previously no plaque was present. After repeated administered nicorandil and nitrates, both lesions became insignificant. Hence, we suspect it might due to coronary

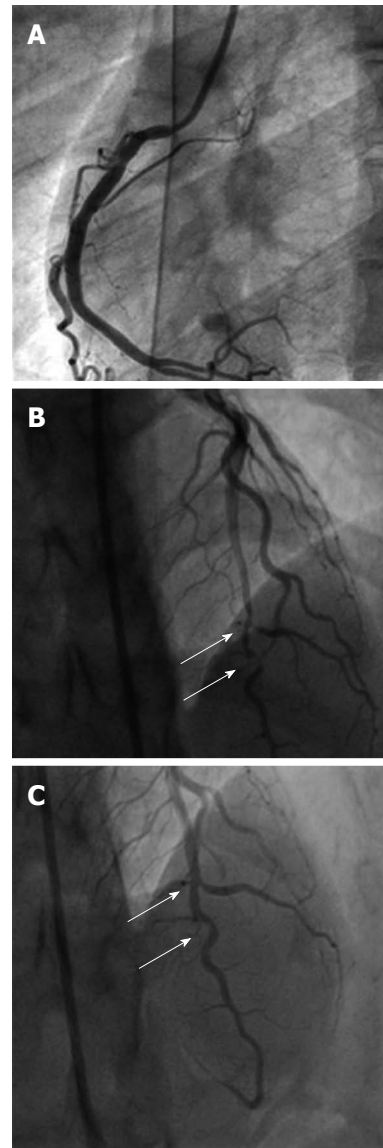


Figure 1 Angiographic images. A: Well flowing patent right coronary artery; B: Angiography revealed mid left anterior descending (LAD) two consecutive lesions (arrows) at D2 bifurcation; C: After *iv* nicorandil and nitroglycerin, mid LAD lesions (arrows) at D2 bifurcation.

spasm. The patient was discharged under treatment with oral nicorandil, nitrates, diltiazem and antiplatelets. Beta blockers were omitted (Figure 1).

After discharge, patient had difficulty in breathing due to some nasal obstruction, so an Ear Nose and Throat (ENT) surgeon consultation was done. Subsequently, a nasal ethmoidal polyp was detected by the ENT surgeon on the basis of a computed tomography scan. The chest physician's opinion was taken and pulmonary function tests were done, which were suggestive of mild obstructive lung disease. Bilateral functional endoscopic sinus surgery was also done (3.5 cm × 2.5 cm, reference range: 0.2 cm × 0.2 cm up to 1.4 cm × 0.8 cm). The histopathological examination of polyps was suggestive of inflammatory nasal polyps. An immunoglobulin E level was 396 kU/L (reference range: 20-100 kU/L). Normal range for

all allergens is less than 0.35 U/L. Within food, cucumber 1.70 U/L, wheat 2.00 U/L, groundnut 1.80 U/L and yeast 1.60 U/L induce mid high allergy. Within inhalants, house dust 1.90 U/L, dog dander 1.10 U/L and paper dust 1.10 U/L induce mid high allergy, while house dust mite 3.50 U/L induces high allergy. Within contact, perfume 1.40 U/L induces mid high allergy. Within drugs, ciprofloxacin 1.70 U/L, cloxacillin 1.30 U/L and diclofenac 1.10 U/L induce mid high allergy, while oxacillin 0.90 U/L, tetracycline 0.60 U/L and norfloxacin 0.80 U/L induce mild allergy. During allergic screening tests (by immune-enzyme immune assay), it was found that the patient was allergic to contact, drugs, food and inhalants. The patient was advised to avoid these allergens and put on topical steroids, cetirizine and montelukast. Aspirin was omitted. To date, the patient has been doing well for the last 9 mo.

Today, allergic angina and allergic myocardial infarction are referred as “Kounis syndrome”. Aspirin-induced asthma was first described by Widal *et al* in 1922 and later by Samter *et al*^[4] in 1967. The term Samter’s triad (asthma, aspirin sensitivity and nasal polyps) became popular. The Samter-Beer triad generally starts as chronic rhinitis with development of nasal polyposis. Salicylate intolerance and asthma develop over 1 to 5 years^[5].

When there is a young individual with no predisposing factors of atherosclerosis and apparent coronary le-

sion, with or without ECG and biochemical markers of infarction, the possibility of Kounis syndrome should be kept in mind. In such situations, intracoronary vasodilators, nitrates, nicorandil or diltiazem should be used before proceeding with a coronary intervention. An urgent eosinophil count should be done before proceeding with coronary interventions to rule out coronary spasm.

REFERENCES

- 1 **Yanagawa Y**, Nishi K, Tomiharu N, Kawaguchi T. A case of takotsubo cardiomyopathy associated with Kounis syndrome. *Int J Cardiol* 2009; **132**: e65-e67 [PMID: 18031840 DOI: 10.1016/j.ijcard.2007.08.022]
- 2 **Kounis GN**, Kounis SA, Hahalis G, Kounis NG. Coronary artery spasm associated with eosinophilia: another manifestation of Kounis syndrome? *Heart Lung Circ* 2009; **18**: 163-164 [PMID: 19081300 DOI: 10.1016/j.hlc.2008.09.008]
- 3 **Chen JP**, Hou D, Pendyala L, Goudevenos JA, Kounis NG. Drug-eluting stent thrombosis: the Kounis hypersensitivity-associated acute coronary syndrome revisited. *JACC Cardiovasc Interv* 2009; **2**: 583-593 [PMID: 19628178 DOI: 10.1016/j.jcin.2009.04.017]
- 4 **Samter M**, Beers RF. Concerning the nature of intolerance to aspirin. *J Allergy* 1967; **40**: 281-293 [PMID: 5235203 DOI: 10.1016/0021-8707(67)90076-7]
- 5 **Szczeklik A**, Nizankowska E, Duplaga M. Natural history of aspirin-induced asthma. AIANE Investigators. European Network on Aspirin-Induced Asthma. *Eur Respir J* 2000; **16**: 432-436 [PMID: 11028656]

P-Reviewer Kounis NG **S-Editor** Gou SX
L-Editor Roemmele A **E-Editor** Zhang DN

