

A case of possible Kounis syndrome as a complication of scombroid syndrome

Stefano Rusconi¹, Antonio Villa¹

Affiliations:

¹ Department of Emergency, ASST Monza, PO Desio, Monza, Italy. Via G. Mazzini,1
20832 Desio (MB), Italy.

Corresponding author:

Dr. Antonio Villa, ASST Monza, PO Desio, Via Fiuggi,56 20159 Milan, Italy.
E-mail: antonio_villa@fastwebnet.it

Abstract

Kounis syndrome is defined as the concurrence of acute coronary syndromes such as coronary spasm or acute myocardial infarction with conditions associated with activation of inflammatory mediators such histamine, arachidonic acid and various cytokines and chemokines. Recently, a variety of unusual etiologies have been reported, including scombroid syndrome. We present a case of a woman without previous history of cardiac diseases or cardiovascular risk factors, who presented to emergency department after the onset of flushing, asthenia, palpitations, burning sensation in the mouth having just eaten tuna. The electrocardiogram revealed a sinus tachycardia with diffuse ST segment depression. After therapy, in a short time symptoms recovered and a second electrocardiogram no longer showed any ST changes. These electrocardiographic changes observed in our case were probably due to transitory coronary vasospasm as described in type I variant of Kounis syndrome.

KEY WORDS: Electrocardiographic changes; histamine poisoning; Kounis syndrome; myocardial ischemia; scombroid syndrome.

Riassunto

La sindrome di Kounis consiste nell'insorgenza di sindromi coronariche acute come lo spasmo coronarico o l'infarto miocardico acuto secondarie all'attivazione di mediatori infiammatori come l'istamina, l'acido arachidonico, varie citochine e chemochine. Recentemente, è stata descritta una varietà di eziologie inusuali, inclusa la sindrome sgombroide. Presentiamo il caso di una donna con anamnesi negativa per patologie cardiache o fattori di rischio cardiovascolari, arrivata in pronto soccorso per l'insorgenza di arrossamento cutaneo, astenia, palpitazioni, sensazione di bruciore in bocca dopo aver mangiato tonno. L'elettrocardiogramma rivelò una tachicardia sinusale con diffuso sottoslivellamento del segmento ST. Dopo la terapia, in breve tempo i sintomi regredirono ed un secondo elettrocardiogramma non mostrò più le modificazioni del tratto ST. Questi cambiamenti elettrocardiografici osservati nel nostro caso potrebbero essere probabilmente dovuti a vasospasmo coronarico transitorio come descritto nella variante tipo 1 della sindrome di Kounis.

TAKE-HOME MESSAGE

Scombroid syndrome is often misdiagnosed and cardiac involvement related to scombroid poisoning is relatively rare, but it could be complicated by vasospasm as described in Kounis syndrome. Physicians should be aware of the existence of Kounis syndrome in order to obtain an early and correct diagnosis and apply appropriate diagnostic and therapeutic measures

Competing interests - none declared.

Copyright © 2017 Stefano Rusconi et al. FS Publishers

This is an open access article distributed under the Creative Commons Attribution (CC BY 4.0) License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited. See <http://www.creativecommons.org/licenses/by/4.0/>.

Cite this article as: Rusconi S, Villa A. A case of possible Kounis syndrome as a complication of scombroid syndrome. J Health Soc Sci. 2017;2(3):329-335

DOI 10.19204/2017/csfp9

Received: 05/10/2017

Accepted: 11/10/2017

Published: 15/11/2017

INTRODUCTION

Scombroid syndrome is a histaminic poisoning, due to histamine-induced reaction because of the ingestion of histamine-contaminated fish [1, 2]. Usually, the course is self-limiting, but in rare cases the poisoning can be serious and severe cardiac complications have been described [1–5]. Allergic angina and allergic myocardial infarction caused by chemical mediators released through mast-cell activation have been described as Kounis syndrome [6–8]. In this syndrome hypersensitivity coronary disorders are induced by various types of environmental exposures, drugs or conditions. Recently, a variety of unusual etiologies have been reported, including scombroid syndrome [9].

We report a case of scombroid poisoning with electrocardiographic changes associated with transitory myocardial ischemia rapidly recovered after antihistaminic therapy. This case could represent a type I variant of Kounis syndrome.

CASE REPORT

A 59-year-old woman without previous history of cardiac diseases, allergies or other relevant diseases and without cardiovascular risk factors, presented to the emergency department after the onset of flushing, asthenia, palpitations, and burning sensation in the mouth having just eaten tuna. She had not chest pain. The patient was treated by the emergency service with hydrocortisone 200 mg IV. In the emergency department, on the clinical examination she had a blood pressure of 100/50 mmHg, tachycardia (heart rate = 124 beats/min), tachypnea (respiratory rate = 28 breaths/min), oxygen saturation of 88%, and a diffuse skin erythematous rash. The first electrocardiogram (hour: 10.10 PM) revealed a sinus tachycardia with diffuse ST segment depression (Figure 1).

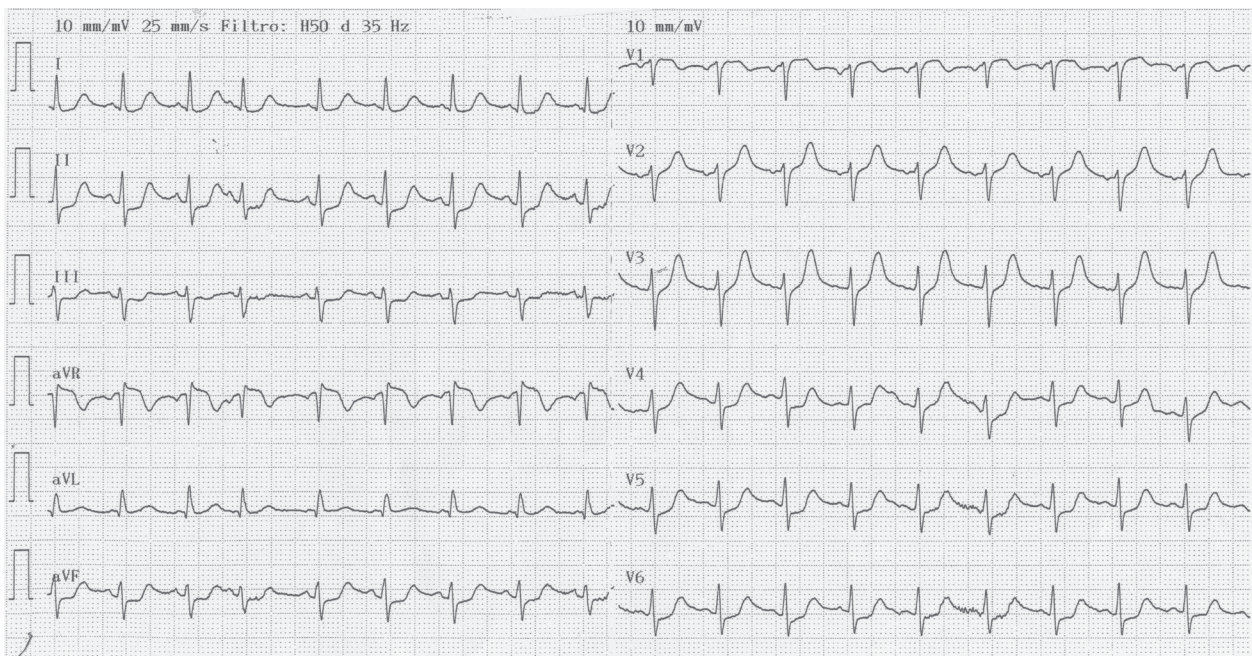


Figure 1. First electrocardiogram showing sinus rhythm with some supraventricular ectopic beats and ST segment depression in leads II, III, aVF, and V2-V6.

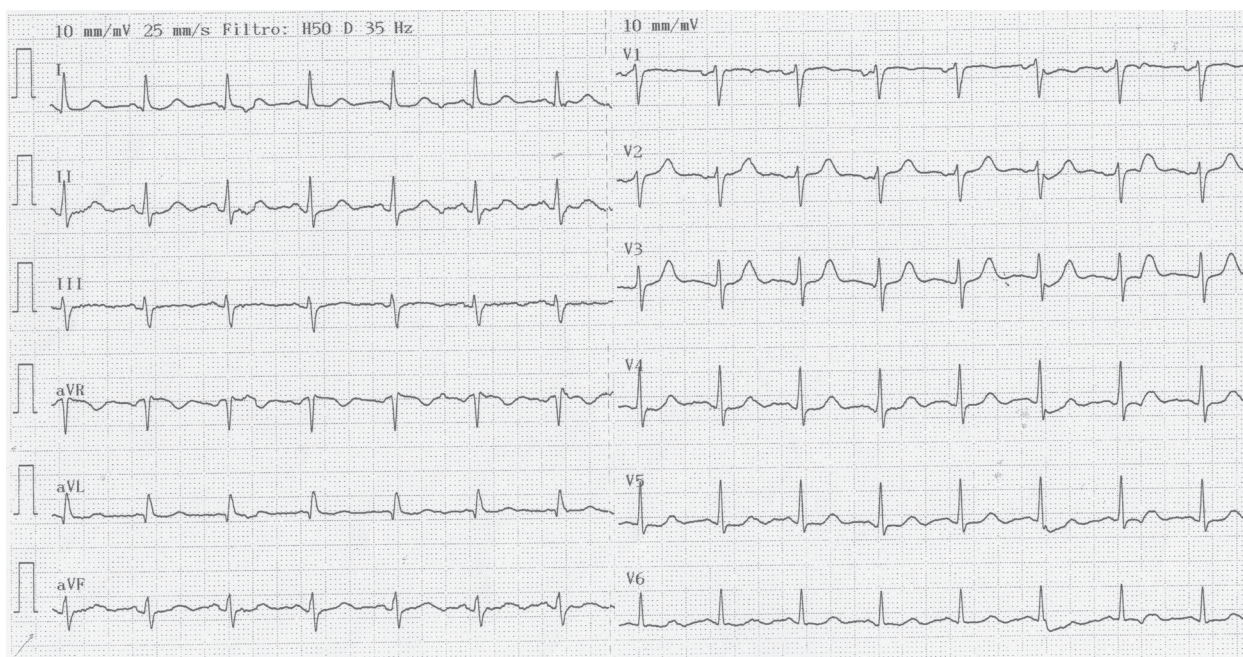


Figure 2. Second electrocardiogram showing the disappearance of ST segment depression.

She received chlorpheniramine 10 mg IV and ranitidine 50 mg IV. In a short time symptoms recovered, vital signs became normal (blood pressure = 130/60 mmHg; heart rate = 88 beats/min; respiratory rate = 14 breaths/min; oxygen saturation = 98%), and a second electrocardiogram (hour: 10.50 PM) no longer showed any ST changes (Figure 2).

DISCUSSION

Scombroid syndrome/histamine poisoning occurs worldwide and it is considered one of – if not – the most common form of toxicity, caused by fish consumption [10]. Scombroid poisoning is triggered by ingestion of poorly preserved fish of Scombroideae and Scomberosidae families, such as tuna, bonito, mackerel, albacore, skipjack, herrings, sardines, anchovies, bluefish, sea urchins, and mahi-mahi; also salmon can be implicated, but more rarely [2].

Histamine is not present in fish in normal condition, but it is produced by histidine decarboxylase present in bacteria resident in fish gills and gastrointestinal tract [11]. This enzyme is formed after few hours of fish exposition at room temperature and is inactivated by temperature of 0°C or lower. Storage at 0°C should be performed immediately after

fishing because, once activated, the histidine carboxylase is still functioning even after bacteria are not more viable [12]. Indeed, the produced histamine is not inactivated by any kind of food processing or cooking [2].

Scombroid syndrome is usually a self-limiting and benign disease. Its symptomatology occurs with a rapid onset, within 10-30 min after fish ingestion. The clinical manifestations spontaneously resolves within 24 hours [13].

The most common clinical manifestations are abdominal pain, diarrhea, nausea and vomiting; facial or generalized flushing, sometimes with hives and/or edema; headache or dizziness; dry mouth associated occasionally with metallic, pepper or bitter taste; palpitations [1, 2, 13].

Notably, some severe cases of scombroid syndrome are reported in literature. These cases include cardiovascular and respiratory complications such as arrhythmias (ventricular fibrillation) and acute coronary syndrome [4, 5, 14-16]; cardiogenic shock associated with acute pulmonary edema [3, 17]; severe bronchoconstriction and hypotension [18, 19].

In literature it has been documented the occurrence of angina or myocardial infarction caused by chemical mediators such histamine,

leukotrienes, and neutral proteases released during an allergic episode. This phenomenon is known as Kounis syndrome and represents a cause of coronary spasm or atheromatous plaque rupture due to allergic etiology [8, 20]. Further, since 1991, allergic angina has been inserted in a subgroup of dynamic lesions of coronary occlusion [6]. The chemical mediators act on smooth vascular coronary muscles, and can induce vasospastic angina or myocardial infarction.

Kounis syndrome is defined as 'the concurrence of acute coronary syndromes such as coronary spasm, acute myocardial infarction, and stent thrombosis, with conditions associated with mast-cell and platelet activation involving interrelated and interacting inflammatory cells in the setting of allergic or hypersensitivity and anaphylactic or anaphylactoid insults' [21]. It is caused by inflammatory mediators such as histamine, neutral, proteases, arachidonic acid products, platelet-activating factor, and a variety of cytokines and chemokines released during the activation process [22–24].

A variety of electrocardiographic changes, ranging from ST-segment elevation or depression to any degree of heart block and cardiac arrhythmias, may be observed [21]. A high index of suspicion regarding this syndrome is of paramount importance. Although it is not a rare disease, its diagnosis is sparse and easily overlooked [21].

Three variants of Kounis syndrome have been described [23]: the type I variant includes normal or nearly normal coronary arteries without risk factors for coronary artery disease, and with the acute release of inflammatory mediators that may induce either coronary artery spasm without increased cardiac enzymes and troponins or coronary artery spasm progressing to acute myocardial infarction with raised cardiac enzymes and troponins. The type II variant includes culprit but quiescent preexisting atheromatous disease in which the acute release of inflammatory mediators may induce either coronary artery spasm with normal cardiac enzymes and troponins or coronary artery spasm together

with plaque erosion or rupture manifesting as acute myocardial infarction. The type III variant includes coronary artery stent thrombosis in which aspirated thrombus specimens stained with hematoxylin-eosin and Giemsa demonstrate the presence of eosinophils and mast cells, respectively [21]. In patients with type I variant, treatment of the allergic event alone may abolish symptoms. The use of hydrocortisone and H1 and H2 antihistamines is adequate [21].

New etiologies of Kounis syndrome are being detected each year [9] and several novel offenders have recently been reported to induce this syndrome, including scombroid poisoning [5, 9, 15, 25, 26].

Electrocardiographic changes observed in our case report were probably due to transitory coronary vasospasm as described in type I variant of Kounis syndrome. The limitation of this case report consists in the lack of troponin dosage, but the quick recovery of symptoms and the rapid normalization of electrocardiographic changes with antihistaminic therapy have driven us to avoid further investigations.

CONCLUSIONS

Scombroid syndrome is often misdiagnosed and therefore under reported; it is believed that the incidence is high because many cases are not reported since the symptoms may last for a short time.

Cardiac involvement related to scombroid poisoning is relatively rare, but it could be complicated by vasospasm as described in Kounis syndrome. In our case report, we showed a case of cardiac complication of scombroid syndrome. Since scombroid fish poisoning can easily be confused with food allergy, the physicians should stay aware assessing the previous consumption of fish that must alert to the possibility of this syndrome and its complications [3]. Moreover, physicians should be aware of the existence of Kounis syndrome in order to obtain an early and correct diagnosis and apply appropriate diagnostic and therapeutic measures [9].

References

1. Hungerford JM. Scombroid poisoning: a review. *Toxicol.* 2010;56(2):231–243.
2. Feng C, Teuber S, Gershwin ME. Histamine (scombroid) fish poisoning: a comprehensive review. *Clin Rev Allergy Immunol.* 2016;50(1):64–69.
3. D'Aloia A, Vizzardì E, Della Pina P, Bugatti S, Del Magro F, Raddino R, et al. A scombroid poisoning causing a life-threatening acute pulmonary edema and coronary syndrome in a young healthy patient. *Cardiovasc Toxicol.* 2011;11(3):280–283.
4. Grinda JM, Bellenfant F, Brivet FG, Carel Y, Deloche A. Biventricular assist device for scombroid poisoning with refractory myocardial dysfunction: a bridge to recovery. *Crit Care Med.* 2004;32(9):1957–1959.
5. Coppola G, Caccamo G, Bacarella D, Corrado E, Caruso M, Cannavò MG, et al. Vasospastic angina and scombroid syndrome: a case report. *Acta Clin Belg.* 2012;67(3):222–225.
6. Kounis NG, Zavras GM. Histamine-induced coronary artery spasm: the concept of allergic angina. *Br J Clin Pract.* 1991;45(2):121–128.
7. Kovanen PT, Kaartinen M, Paavonen T. Infiltrates of activated mast cells as the site of coronary atheromatous erosion or rupture in myocardial infarction. *Circulation.* 1995;92(5):1084–1088.
8. Kounis NG. Kounis syndrome (allergic angina and allergic myocardial infarction): a natural paradigm? *Int J Cardiol.* 2006;110(1):7–14.
9. Kounis NG, Giannopoulos S, Soufras GD, Kounis GN, Goudevenos J. Foods, drugs and environmental factors: novel Kounis syndrome offenders. *Intern Med.* 2015;54(13):1577–1582.
10. Dalgaard P, Emborg J, Kjolby A, Sorensen ND, Ballin NZ. Histamine and biogenic amines: formation and importance in seafood. In: *Improving seafood products for the consumer*, pp 292–324. Cambridge: Borresen T Eds, British Welding Research Association; 2008.
11. Tsai YH, Kung HF, Lee TM, Lin GT, Hwang DF. Histamine related hygienic qualities and bacteria found in popular commercial scombroid fish fillets in Taiwan. *J Food Prot.* 2004;67(2):407–412.
12. Cattaneo P. Scombroid syndrome: histamine poisoning. *Food In.* 2011;1(2):5–80. *Italian.*
13. Smolinska S, Jutel M, Cramerì R, O'Mahony L. Histamine and gut mucosal immune regulation. *Allergy.* 2014;69(3):273–281.
14. Tortorella V, Masciari P, Pezzi M, Mola A, Tiburzi SP, Zinzi MC, et al. Histamine poisoning from ingestion of fish or scombroid syndrome. *Case Rep Emerg Med.* 2014;2014:482531.
15. Cucunato M, Cannavà G, Currò A. Acute coronary syndrome and scombroid syndrome. *Int J Cardiol.* 2015;187:317–318.
16. McInermey J, Sahgal P, Vogel M, Rahn E, Jonas E. Scombroid poisoning. *Ann Emerg Med.* 1996;28(2):235–238.
17. Ascione A, Barresi LS, Sarullo FM, De Silvestre G. Two cases of “scombroid syndrome” with severe cardiovascular compromise. *Cardiologia.* 1997;42(12):1285–1288.
18. Iannuzzi M, D'Ignazio N, Bressy L, De Sio A. Severe scombroid fish poisoning syndrome requiring aggressive fluid resuscitation in the emergency department: two case reports. *Minerva Anestesiol.* 2007;73(9):481–483.
19. Borade PS, Ballary CC, Lee DK. A fish cause of sudden near fatal hypotension. *Resuscitation.* 2007;72(1):158–160.
20. Zavras GM, Papadaki PJ, Kokkinis CE, Kalokairinov K, Kouni SN, Batsolaki M, et al. Kounis syndrome secondary to allergic reaction following shellfish ingestion. *Int J Clin Pract.* 2003;57(7):622–624.
21. Kounis NG. Coronary hypersensitivity disorder: the Kounis syndrome. *Clin Ther.* 2013;35(5):563–571.
22. Biteker M. Current understanding of Kounis syndrome. *Expert Rev Clin Immunol.* 2010;6(5):777–788.
23. Kounis NG, Mazarakis A, Tsigkas G, Giannopoulos S, Goudevenos J. Kounis syndrome: a new twist on

- an old disease. *Future Cardiol.* 2011;7(6):805–824.
24. Kounis NG, Giannopoulos S, Tsigkas GG, Goudevenos J. Eosinophilic responses to stent implantation and the risk of Kounis hypersensitivity associated coronary syndrome. *Int J Cardiol.* 2012;156(2):125–132.
 25. Ridolo E, Martignago I, Senna G, Ricci G. Scombroid syndrome: it seems to be fish allergy but ... it isn't. *Curr Opin Allergy Clin Immunol.* 2016;16(5):516–521.
 26. De Gennaro L, Brunetti ND, Locuratolo N, Ruggiero M, Resta M, Diaferia G, et al. Kounis syndrome following canned tuna fish ingestion. *Acta Clin Belg.* 2017;72(2):142–145.