Acute Myocardial Infarction Induced by Anaphylaxis

Rui Tang, Jin-Lu Sun

Department of Allergy, Peking Union Medical College Hospital, Peking Union Medical College, Chinese Academy of Medical Sciences, Beijing 100730, China

To the Editor: A 65-year-old male patient was admitted to our hospital on September 9th, 2016, because of wheals involving the whole body 1 h after ingesting bread and milk. His coexisting symptoms included itchiness, chest discomfort, dyspnea, wheezing, abdominal pain, nausea, vomiting, palpitation, sweating, pale complexion, dizziness, and syncope. His consciousness recovered after taking the Suxiao Jiuxin pill, and then he was sent to our hospital.

The patient's history included hyperlipidemia and hypertension for 5 years. He had undergone percutaneous coronary intervention. One stent had been placed at the site between the distal left main (LM) coronary artery and the proximal left anterior descending (LAD) coronary artery, and another stent had been placed at the site between the LM and the proximal segment of the LAD. He complained of repeated wheals involving the whole body, accompanied by chest discomfort, abdominal pain, and dizziness in the past 2 years without definitive precipitating factors. Such symptoms had occurred five times previously up to the current presentation. The patient did not pay attention to these symptoms or receive further tests and treatment.

The physical examination on admission revealed that the patient's BP was 51/21 mmHg, heart rate (HR) was 95 beats/min, and oxygen saturation was 92%. Bilateral lungs were clear on auscultation. Cardiac examination revealed a regular rhythm without murmur. The electrocardiogram showed a sinus rhythm with a HR of 88 beats/min. ST elevation was noticed on the II, III, aVF, and V5–V9 leads. Cardiac enzymes showed that the creatine kinase (CK) level was 114 U/L, CK-myocardial band (CK-MB) was 1.0 μg/L, cardiac troponin I (cTnI) was 0.000 μg/L, and N-terminal pro b-type natriuretic peptide was 132 pg/ml. The allergen test revealed a Grade 2 (2.11 kUA/L) for wheat and Grade 0 for milk.

The diagnosis of anaphylaxis and acute ST-elevated myocardial infarction was made on admission. Epinephrine at a dose of 0.3 mg was injected subcutaneously, and hydrocortisone was administered intravenously. Other drugs administered subcutaneously included ticagrelor at a dose of 180 mg and Clexane at a dose of 6000 U. After treatment, BP returned to 107/70 mmHg, with a HR of 82 beats/min. The myocardial biomarkers were measured at 14:00, showing that the CK level was 255 U/L, the CK-MB level was 9.1 μg/L, and the cTnI level was 3.015 μg/L. Coronary angiography was performed, which revealed lesions in the left trunk and the three coronary branches. The stent was unobstructed. The elevated ST segment decreased postoperatively, returning to normal 14 h after the operation. The myocardial biomarkers reached a peak at 13 h after the operation and returned to normal gradually thereafter. The CK level increased to 411 U/L and 486 U/L, then decreased to 128 U/L. The CK-MB level increased to 18.2 μg/L and 21.5 μg/L, then decreased to 1.1 μg/L. The cTnI level increased to 8.728 μg/L and 9.478 μg/L, then dropped to 2.726 μg/L.

There are no reports on acute myocardial infarction (AMI) induced by anaphylaxis in China. Anaphylaxis is a severe, life-threatening, and systematic allergic disease, with an incidence rate of between 0.05% and 2%.[1] It is a Type I allergic disease induced by IgE. Clinical manifestations of the disease can involve the skin, respiratory, cardiovascular, and digestive systems. Anaphylaxis may present rapid hypotension within minutes or hours, and it can cause death if not promptly treated. The patient had preexisting cardiovascular disease, but there was no evidence of coronary artery occlusion by coronary angiography. The AMI was considered to be caused by coronary artery spasm and hypoperfusion during anaphylaxis, rather than by lesions on the coronary artery.[2] Epinephrine can be a triggering factor of AMI. Epinephrine can contract blood vessels in anaphylaxis to ensure perfusion to vital organs. However, vascular contraction may cause local cardiac ischemia, leading to AMI.[3] Jiang *et al.* have reported that wheat is a significant allergen in the Chinese population, and physical activity after eating wheat can exacerbate the disease.[4] The patient had reported similar symptoms before this acute episode, which was attributable to the ingestion of an allergenic food. However, he did not pay attention to this condition. The acute episode was induced after ingesting wheat followed by walking. The symptoms involved the skin, respiratory, digestive, and cardiovascular systems. Hypotension was a cardinal presentation. According to the American Academy of Allergy, Asthma, and Immunology criteria,[5] the diagnosis of anaphylaxis was determined, and an allergen test revealed that wheat was the culprit. The disease was wheat-dependent exercise-induced anaphylaxis, which further induced AMI. Allergens and exercise should be avoided for those who are allergic to certain foods and induced by exercise so that

Address for correspondence: Dr. Jin-Lu Sun, Department of Allergy, Peking Union Medical College Hospital, Peking Union Medical College, Chinese Academy of Medical Sciences, Beijing 100730, China
E-Mail: 18611382050@163.com

Access this article online

Quick Response Code:

Website: www.cmj.org

DOI: 10.4103/0366-6999.231518

Received: 20-12-2017 **Edited by:** Li-Shao Guo
How to cite this article: Tang R, Sun JL. Acute Myocardial Infarction Induced by Anaphylaxis. Chin Med J 2018;131:1251-2.

catastrophic conditions such as AMI can be prevented in this way. For elderly patients with preexisting cardiovascular disease, close monitoring should be instituted after administering epinephrine. In addition, cardiovascular conditions should be identified as soon as possible to save lives.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient has given his consent for his images and other clinical information to be reported in the journal. The patient understands that his name and initial will not be published and due efforts will be made to conceal his identity, but anonymity cannot be guaranteed.

Financial support and sponsorship

This work was supported by a grant from CAMS Innovation Fund for Medical Sciences (No. 2016-I2M-1003).

Conflicts of interest

There are no conflicts of interest.

References

1. Lieberman P, Nicklas RA, Oppenheimer J, Kemp SF, Lang DM, Bernstein DI, *et al.* The diagnosis and management of anaphylaxis practice parameter: 2010 update. J Allergy Clin Immunol 2010;126:477-80.e1-42. doi: 10.1016/j.jaci.2010.06.022.
2. Kounis NG, Mazarakis A, Bardousis C, Patsouras N. The heart and coronary arteries as primary target in severe allergic reactions: Cardiac troponins and the Kounis hypersensitivity-associated acute coronary syndrome. Int J Cardiol 2015;198:83-4. doi: 10.1016/j.ijcard.2015.06.142.
3. Park JS, Min JH, Kang JH, In YN. Acute myocardial infarction due to stent thrombosis after administration of intravenous epinephrine for anaphylaxis. Chin Med J 2015;128:2692-3. doi: 10.4103/0366-6999.166020.
4. Jiang N, Yin J, Wen L, Li H. Characteristics of anaphylaxis in 907 Chinese patients referred to a tertiary allergy center: A Retrospective study of 1,952 episodes. Allergy Asthma Immunol Res 2016;8:353-61. doi: 10.4168/aair.2016.8.4.353.
5. Lieberman P, Nicklas RA, Randolph C, Oppenheimer J, Bernstein D, Bernstein J, *et al.* Anaphylaxis – A practice parameter update 2015. Ann Allergy Asthma Immunol 2015;115:341-84. doi: 10.1016/j.anai.2015.07.019.