Journal of Acute Medicine 10(4): 156-160, 2020
DOI:10.6705/j.jacme.202012_10(4).0004
Case Report

Acute Myocardial Infarction in Anaphylactic Shock After an Ant Bite: Case Report

Yun-Wu Deng[1,*], Yi-Wei Tsuei[2]

[1]Department of Emergency Medicine, Landseed International Hospital, Taoyuan, Taiwan
[2]Department of Emergency Medicine, Taoyuan Armed Forces General Hospital, Taoyuan, Taiwan

A 72-year-old woman with an ant bite on her hand presented with anaphylaxis at the scene and chest pain in the emergency department. Electrocardiography showed a myocardial infarction with inferior and lateral wall ST elevation. She received percutaneous coronary intervention. The results revealed left anterior descending branch and right coronary artery obstruction. After stenting, she was discharged in good condition. Kounis syndrome was diagnosed. Emergency physicians should be alert to acute coronary syndrome following anaphylaxis.

Key words: *allergy, anaphylaxis, myocardial infarction, Kounis syndrome*

Introduction

Insect bites can induce allergy symptoms in some people. Anaphylaxis can induce general edema, itching, and even hypotension. However, chest pain and myocardial infarction are seen less often in these cases. Anaphylaxis with acute coronary syndrome is rare. Herein, we report a 72-year-old woman with anaphylaxis after an ant bite on her hand with a subsequent acute coronary syndrome.

Case Report

A 72-year-old woman (156 cm, 63 kg) with a history of coronary artery disease with stent deployment, diabetes, and hypertension received an ant bite on her right dorsal hand at a farm. She felt a general weakness. At the scene, paramedics recorded the Glasgow coma scale score of E4V5M6, blood pressure of 68/35 mmHg, respiratory rate of 24/min, pulse rate of 138/min, oxygen saturation of 98%, and body temperature of 35.2°C. Physical examination revealed a general skin rash. Anaphylactic shock was diagnosed. She was given oxygen via a mask, and an intramuscular (IM) injection of epinephrine 0.3 mL in the deltoid muscle area of the left arm. She was sent to the emergency department. There, her coma scale score was E4V5M6, blood pressure was 153/68 mmHg, respiratory rate was 22/min, pulse rate was 107/min, and body temperature was 35.2°C. Her blood pressure had obviously improved. However, she complained of left side chest pain 15 minutes later. Electrocardiography (ECG) showed ST elevation in the inferior wall, and mild ST elevation in the lateral wall (Fig. 1). The blood test results were within normal limits. Acute myocardial infarction was diagnosed. The emergency coronary angiography showed total occlusion of the proximal and middle one-third of the right coronary artery (Fig. 2). After thrombi aspiration and balloon dilation, residual stenosis was under 10% (Figs. 3 and 4). Stent implantation was performed in the anterior descending branch of the left coronary artery and the proximal right coronary artery without in-stent restenosis. There was 70% stenosis of the proximal left coronary artery. The tro-

Received: May 15, 2019; Revised: January 6, 2020 (3rd); Accepted: February 11, 2020.
*Corresponding author: Yun-Wu Deng, MD, Department of Emergency Medicine, Landseed International Hospital, No. 77, Guangtai Rd., Pingzhen Dist., Taoyuan City 324, Taiwan. E-mail: deng888168@hotmail.com

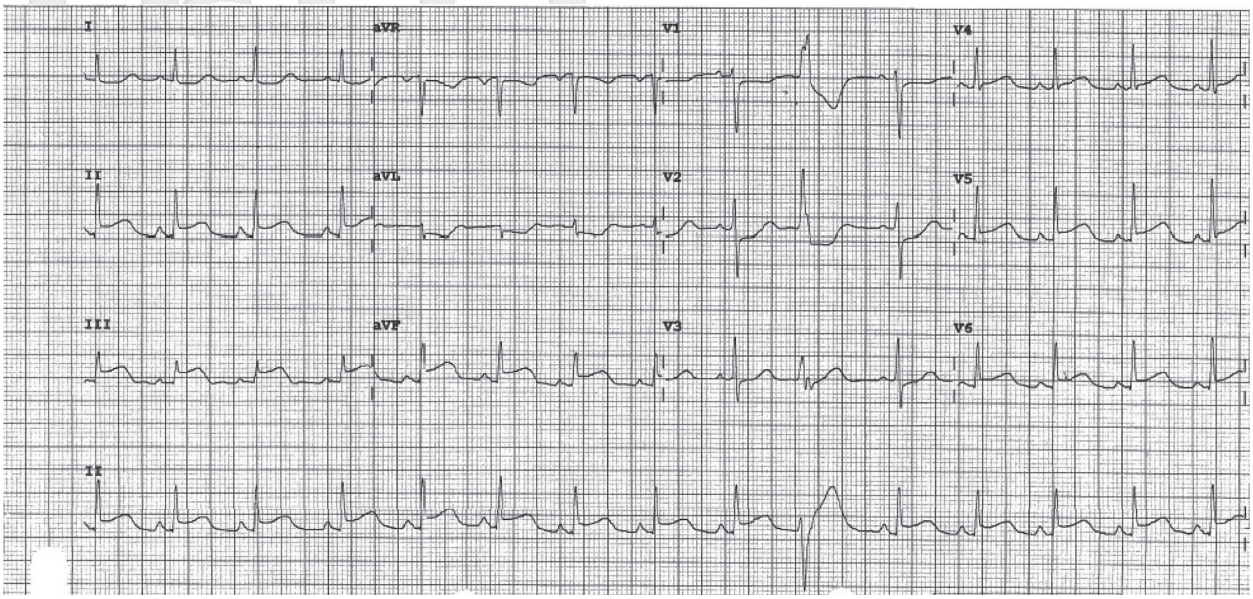

Fig. 1. Electrocardiography reveals ST elevation in leads II, III and aVF and mild ST elevation in V5–V6.

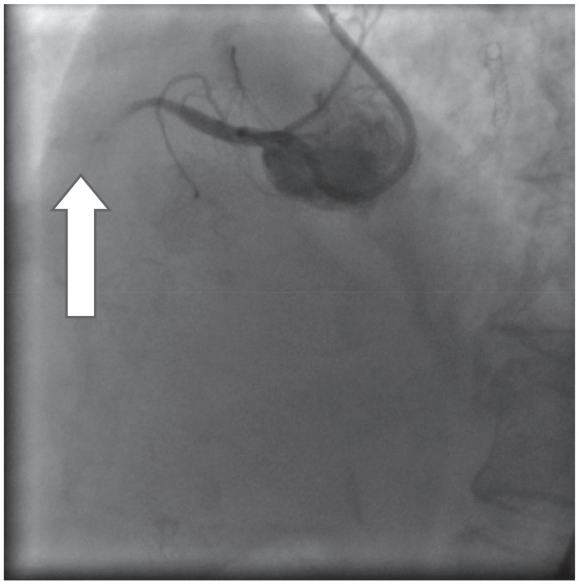

Fig. 2. Arteriography shows total occlusion of the proximal right coronary artery (white arrow).

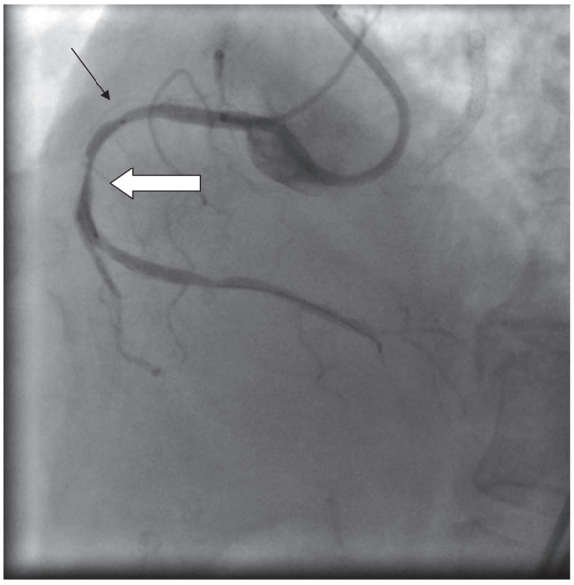

Fig. 3. Arteriography shows stenosis in the proximal (black arrow) and the middle (white arrow) right coronary artery.

ponin-I level was within normal limits and the ratio of eosinophils to total white blood cells was 0.9%. This patient was stable and was discharged after 8 days of hospitalization.

Discussion

In 1991, Kounis in Greece first described a coronary syndrome (angina and myocardial infarction) associated with allergic reactions.[1] In 2016, Kounis revised the definition of Kounis syndrome (KS) to acute coronary syndrome associated with mast-cell and platelet activation in the setting of hypersensitivity and allergic or anaphylactic insults.[2] The pathophysiology of KS involves coronary artery spasm and/or atheromatous plaque erosion or rupture during an allergic reaction.[2]

KS can be classified into three types. In type I, the release of inflammatory mediators induces coro-

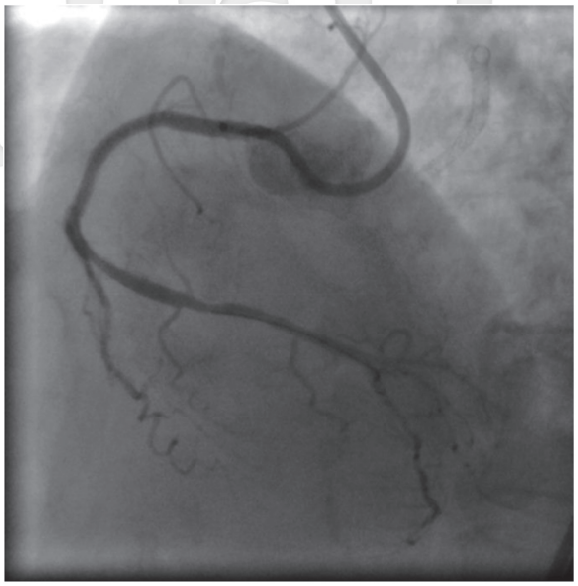

Fig. 4. Arteriography shows the right coronary artery is completely patent after stenting.

nary artery spasm with or without increases in cardiac enzymes and troponins. In type II, the release of inflammatory mediators induces coronary artery spasm together with plaque erosion or rupture manifesting as acute MI. Type III includes patients with coronary artery stent thrombosis as a result of an allergic reaction. Our patient had type III KS.

KS is rare. A search of MEDLINE by Abdelghany et al. through March 31, 2016 found a total of 175 patients whose cases fulfilled the definition of one of the three variants of KS.[2] Men were affected more often than women (74.3%). The most common age group was 40–70 years (68%). Risk factors for KS included an allergy history, hypertension, smoking, diabetes, and hyperlipidemia. The most common trigger of KS was antibiotics (27.4%). In 80% of patients, the symptoms of acute coronary syndrome happened within 1 hour of allergic insults. The most common symptom was chest pain (86.8%). Type I was found in 72.6% of patients, type II was 22.3%, and type III was 5.1%. The inferior wall was most commonly affected (66.9%). The most common complication was cardiac arrest (6.3%) (Table 1).[2]

There have been some reports of KS in Taiwan. Wong et al. reported a 38-year-old woman with anaphylactic shock following the intrathecal injection of contrast for the lumbar spine myelography in 1996. She had no peripheral pulses. ECG initially revealed ventricular tachycardia, and later, ST depression with inverted T waves in the inferior wall. Finally, the patient was stabilized and discharged.[3] She most likely had KS type I. Yang et al. reported a 70-year-old man who sustained KS induced by cisatracurium administration following induction of general anesthesia in 2008.[4] Bradycardia, hypotension, and wheezing were noted. ECG showed diffuse ST-T changes suggestive of acute inferior and posterior wall myocardial infarction. Finally, the patient was stabilized and discharged. This was most likely KS type II. In 2012, Hsu et al. reported a 54-year-old man with dizziness, weakness, chest tightness, and generalized pruritus with skin erythema after eating onions. Initial ECG revealed first-degree atrioventricular block and right bundle branch block. The creatine kinase-MB and troponin levels were within normal limits. Later the ECG showed the inferior wall ST elevation. Selective cardiac angiography revealed total occlusion of the distal right coronary artery.[5] Balloon angioplasty was successfully performed. This case was most likely KS type II. The anaphylactic inducers in these reported cases were different from that in our case.

Epinephrine injection is a treatment option in anaphylactic shock. However, epinephrine is a strong vasoconstrictor and is associated with adverse cardiovascular (CV) events. Campbell et al. compared the IM, subcutaneous, and intravenous (IV) routes of administration in patients with adverse CV events associated with the anaphylaxis management with epinephrine. There were adverse CV events in 3 of 30 doses by IV bolus compared with 4 of 316 doses by IM injection (10% vs. 1.3%, $p = 0.006$).[6] These data support the safety of IM epinephrine.

Because most patients with KS (types I and II) have only coronary vasospasm without thrombus obstruction, some authors suggest a coronary angiography may be not necessary.[1] Single-photon emission computer tomography has recently been used in the diagnosis of type I variant KS.[2]

In conclusion, allergic reactions sometimes result in complications such as respiratory or circulatory compromise. Acute coronary syndrome is a possible CV problem. The differential diagnosis of chest pain should also include pulmonary emboli, myocarditis, and aortic dissection. KS is one of these complications, but it has a good prognosis for complete recovery. However, caution should be used because some conditions in the differential diagnosis carry a high risk of mortality.

Table 1. Epidemiology, clinical presentations, diagnostic findings and complications in 175 patients with Kounis syndrome in the literature[2]

Variable	n (%)	Variable	n (%)
Sex		Type of Kounis syndrome	
Male	130 (74.3)	Type I	127 (72.6)
Female	45 (25.7)	Type II	39 (22.3)
Age		Type III	9 (5.1)
0–10	3 (1.7)	Troponin	
11–20	13 (7.4)	Elevated	106 (60.6)
21–30	8 (4.6)	Normal	44 (25.1)
31–40	14 (8.0)	Not documented	25 (14.3)
41–50	26 (14.9)	ECG manifestation	
51–60	52 (29.7)	ST elevation	133 (76.0)
61–70	41 (23.4)	Inferior leads	89 (66.9)
71–80	15 (8.6)	Anterior leads	22 (16.5)
81–90	3 (1.7)	Antero-lateral leads	10 (7.5)
Past medical history		Lateral leads	7 (5.3)
Allergy	44 (25.1)	Diffuse elevation	3 (2.3)
Hypertension	32 (18.3)	Unknown	2 (1.5)
Smoking	23 (13.1)	ST depression	30 (17.1)
Diabetes	21 (12.0)	Inferior leads	10 (33.3)
Hyperlipidemia	20 (11.4)	Anterior leads	10 (33.3)
Triggers		Lateral leads	6 (20.0)
Antibiotics	48 (27.4)	Antero-lateral leads	4 (13.3)
Penicillins	27	Normal	3 (1.7)
Cephalosporins	13	Unknown	9 (5.1)
Insect bites	41 (23.4)	Echocardiography	
Bee	18	Abnormal	45/78 (57.7)
Wasp	14	Normal	33/78 (42.3)
Others	86 (49.2)	Complications	
Triggers to onset time in hours		Cardiogenic shock	4 (2.3)
0–1	105/131 (80.0)	Cardiac arrest	11 (6.3)
1–2	2/131 (1.5)	Death	5 (2.9)
2–3	6/131 (4.6)	Ventricular fibrillation	2 (1.1)
3–4	2/131 (1.5)	Anterior STEMI	2 (1.1)
4–5	2/131 (1.5)	Interior STEMI	1 (0.6)
5–6	2/131 (1.5)		
> 6	12/131 (9.2)		
Clinical presentation			
Chest pain	152 (86.8)		
Anaphy laxis	93 (53.0)		
Rash	47 (26.8)		
Wheezing	25 (14.0)		
Pulmonary edema	9 (5.1)		

ECG: electrocardiography; STEMI: ST elevation myocardial infarction.

Conflicts of Interest Statement

The authors declare no conflicts of interest.

References

1. Kounis NG, Zavras GM. Histamine-induced coronary artery spasm: the concept of allergic angina. *Br J Clin Pract* 1991;45:121-128.
2. Abdelghany M, Subedi R, Shah S, Kozman H. Kounis syndrome: a review article on epidemiology, diagnostic findings, management and complications of allergic acute coronary syndrome. *Int J Cardiol* 2017;232:1-4. doi:10.1016/j.ijcard.2017.01.124
3. Wong YK, Cheng HC, Law HW, Liu PH, Hao KL. Anaphylaxis followed by reversible myocardial ischemia: a case report. *J Emerg Crit Care Med* 1996;7:27-31. doi:10.30018/JECCM.199603.0005
4. Yang YL, Huang HW, Yip HK, Jawan B, Tseng CC, Lu HF. Acute coronary syndrome in cisatracurium-induced anaphylactic shock: Kounis syndrome. *Acta Anaesthesiol Taiwan* 2008;46:184-186. doi:10.1016/S1875-4597(09)60007-2
5. Hsu LW, Hou SW, Wu BH, Wang TL, Hung TY. Onion-induced anaphylactic shock rapidly evolving to allergic right ventricular myocardial infarction and subsequent cardiogenic shock. *J Acute Med* 2012;2:117-120. doi:10.1016/j.jacme.2012.08.002
6. Campbell RL, Bellolio MF, Knutson BD, et al. Epinephrine in anaphylaxis: higher risk of cardiovascular complications and overdose after administration of intravenous bolus epinephrine compared with intramuscular epinephrine. *J Allergy Clin Immunol Pract* 2015;3:76-80. doi:10.1016/j.jaip.2014.06.007