



HONEY BEE STING – A MIMIC OF ACUTE MYOCARDIAL INFARCTION – CASE SERIES

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INTRODUCTION

Acute myocardial infarction (AMI) due to honeybee sting has been extensively documented in literature. Numerous authors have discussed the relationship between honey bee sting, anaphylactic shock and myocardial infarction. 1-4 Bee venoms can act in promoting acute coronary artery thrombosis via platelet aggregation and hypotension. The allergic reaction secondary to the stings trigger various inflammatory mediators and can induce acute coronary syndrome. Many studies have reported single cases of honeybee bite and their manifestations¹.

Herein, we report multiple cases with clinical manifestations mimicking acute myocardial infarction following honeybee sting and review the literature.

CASE REPORTS

CASE 1

A 48 years old male non hypertensive, non diabetic with no prior history of cardiac disorders, farmer by occupation presented with history of honey bee bites while working in the fields. The patient an ex smoker left the habit 2 years back.

The patient started complaining of dyspnoea on exertion and chest pain NYHA class II after 2 days of the incident. The patient was taken to a local doctor for the complaints and was then referred to this hospital for further management. The patient was evaluated for the symptoms in the form of investigations of ECG (Figure 1), Echocardiography and cardiac enzymes all of which were found to be normal.

The patient underwent Treadmill Exercise testing (figure 2) which was positive for inducible ischaemia. In view of the symptoms of presentation and TMT positive the patient was taken up for diagnostic coronary angiography which revealed triple vessel disease.

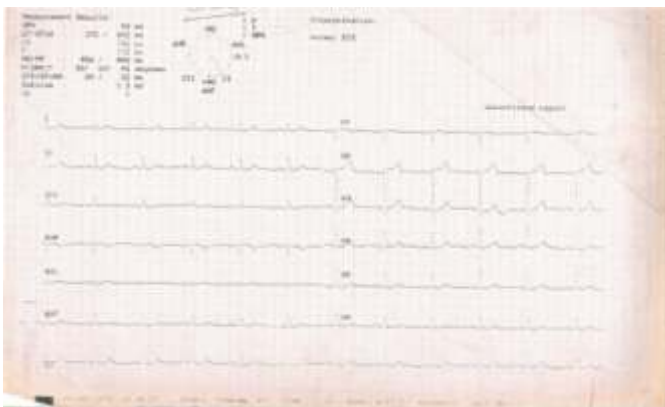


FIGURE 1

Stage	Stage Speed (km/hr) Time (min)	HR bpm	BP mmHg	RPR % CO2	VEG L/min	ST mV	Stage Comments
Rest	0:00 0.00/0.00	70	120/80	1	1.00	0.00	
Stage 1	0:05 0.40/0.40	75	120/80	1	1.00	-0.05	
Stage 2	0:10 0.80/0.80	80	120/80	1	1.00	-0.10	
Stage 3	0:15 1.20/1.20	85	120/80	1	1.00	-0.15	
Stage 4	0:20 1.60/1.60	90	120/80	1	1.00	-0.20	
Stage 5	0:25 2.00/2.00	95	120/80	1	1.00	-0.25	
Stage 6	0:30 2.40/2.40	100	120/80	1	1.00	-0.30	
Stage 7	0:35 2.80/2.80	105	120/80	1	1.00	-0.35	
Stage 8	0:40 3.20/3.20	110	120/80	1	1.00	-0.40	
Stage 9	0:45 3.60/3.60	115	120/80	1	1.00	-0.45	
Stage 10	0:50 4.00/4.00	120	120/80	1	1.00	-0.50	
Stage 11	0:55 4.40/4.40	125	120/80	1	1.00	-0.55	
Stage 12	1:00 4.80/4.80	130	120/80	1	1.00	-0.60	
Stage 13	1:05 5.20/5.20	135	120/80	1	1.00	-0.65	
Stage 14	1:10 5.60/5.60	140	120/80	1	1.00	-0.70	
Stage 15	1:15 6.00/6.00	145	120/80	1	1.00	-0.75	
Stage 16	1:20 6.40/6.40	150	120/80	1	1.00	-0.80	
Stage 17	1:25 6.80/6.80	155	120/80	1	1.00	-0.85	
Stage 18	1:30 7.20/7.20	160	120/80	1	1.00	-0.90	
Stage 19	1:35 7.60/7.60	165	120/80	1	1.00	-0.95	
Stage 20	1:40 8.00/8.00	170	120/80	1	1.00	-1.00	
Stage 21	1:45 8.40/8.40	175	120/80	1	1.00	-1.05	
Stage 22	1:50 8.80/8.80	180	120/80	1	1.00	-1.10	
Stage 23	1:55 9.20/9.20	185	120/80	1	1.00	-1.15	
Stage 24	2:00 9.60/9.60	190	120/80	1	1.00	-1.20	
Stage 25	2:05 10.00/10.00	195	120/80	1	1.00	-1.25	
Stage 26	2:10 10.40/10.40	200	120/80	1	1.00	-1.30	
Stage 27	2:15 10.80/10.80	205	120/80	1	1.00	-1.35	
Stage 28	2:20 11.20/11.20	210	120/80	1	1.00	-1.40	
Stage 29	2:25 11.60/11.60	215	120/80	1	1.00	-1.45	
Stage 30	2:30 12.00/12.00	220	120/80	1	1.00	-1.50	
Stage 31	2:35 12.40/12.40	225	120/80	1	1.00	-1.55	
Stage 32	2:40 12.80/12.80	230	120/80	1	1.00	-1.60	
Stage 33	2:45 13.20/13.20	235	120/80	1	1.00	-1.65	
Stage 34	2:50 13.60/13.60	240	120/80	1	1.00	-1.70	
Stage 35	2:55 14.00/14.00	245	120/80	1	1.00	-1.75	
Stage 36	3:00 14.40/14.40	250	120/80	1	1.00	-1.80	
Stage 37	3:05 14.80/14.80	255	120/80	1	1.00	-1.85	
Stage 38	3:10 15.20/15.20	260	120/80	1	1.00	-1.90	
Stage 39	3:15 15.60/15.60	265	120/80	1	1.00	-1.95	
Stage 40	3:20 16.00/16.00	270	120/80	1	1.00	-2.00	
Stage 41	3:25 16.40/16.40	275	120/80	1	1.00	-2.05	
Stage 42	3:30 16.80/16.80	280	120/80	1	1.00	-2.10	
Stage 43	3:35 17.20/17.20	285	120/80	1	1.00	-2.15	
Stage 44	3:40 17.60/17.60	290	120/80	1	1.00	-2.20	
Stage 45	3:45 18.00/18.00	295	120/80	1	1.00	-2.25	
Stage 46	3:50 18.40/18.40	300	120/80	1	1.00	-2.30	
Stage 47	3:55 18.80/18.80	305	120/80	1	1.00	-2.35	
Stage 48	4:00 19.20/19.20	310	120/80	1	1.00	-2.40	
Stage 49	4:05 19.60/19.60	315	120/80	1	1.00	-2.45	
Stage 50	4:10 20.00/20.00	320	120/80	1	1.00	-2.50	
Stage 51	4:15 20.40/20.40	325	120/80	1	1.00	-2.55	
Stage 52	4:20 20.80/20.80	330	120/80	1	1.00	-2.60	
Stage 53	4:25 21.20/21.20	335	120/80	1	1.00	-2.65	
Stage 54	4:30 21.60/21.60	340	120/80	1	1.00	-2.70	
Stage 55	4:35 22.00/22.00	345	120/80	1	1.00	-2.75	
Stage 56	4:40 22.40/22.40	350	120/80	1	1.00	-2.80	
Stage 57	4:45 22.80/22.80	355	120/80	1	1.00	-2.85	
Stage 58	4:50 23.20/23.20	360	120/80	1	1.00	-2.90	
Stage 59	4:55 23.60/23.60	365	120/80	1	1.00	-2.95	
Stage 60	5:00 24.00/24.00	370	120/80	1	1.00	-3.00	
Stage 61	5:05 24.40/24.40	375	120/80	1	1.00	-3.05	
Stage 62	5:10 24.80/24.80	380	120/80	1	1.00	-3.10	
Stage 63	5:15 25.20/25.20	385	120/80	1	1.00	-3.15	
Stage 64	5:20 25.60/25.60	390	120/80	1	1.00	-3.20	
Stage 65	5:25 26.00/26.00	395	120/80	1	1.00	-3.25	
Stage 66	5:30 26.40/26.40	400	120/80	1	1.00	-3.30	
Stage 67	5:35 26.80/26.80	405	120/80	1	1.00	-3.35	
Stage 68	5:40 27.20/27.20	410	120/80	1	1.00	-3.40	
Stage 69	5:45 27.60/27.60	415	120/80	1	1.00	-3.45	
Stage 70	5:50 28.00/28.00	420	120/80	1	1.00	-3.50	
Stage 71	5:55 28.40/28.40	425	120/80	1	1.00	-3.55	
Stage 72	6:00 28.80/28.80	430	120/80	1	1.00	-3.60	
Stage 73	6:05 29.20/29.20	435	120/80	1	1.00	-3.65	
Stage 74	6:10 29.60/29.60	440	120/80	1	1.00	-3.70	
Stage 75	6:15 30.00/30.00	445	120/80	1	1.00	-3.75	
Stage 76	6:20 30.40/30.40	450	120/80	1	1.00	-3.80	
Stage 77	6:25 30.80/30.80	455	120/80	1	1.00	-3.85	
Stage 78	6:30 31.20/31.20	460	120/80	1	1.00	-3.90	
Stage 79	6:35 31.60/31.60	465	120/80	1	1.00	-3.95	
Stage 80	6:40 32.00/32.00	470	120/80	1	1.00	-4.00	
Stage 81	6:45 32.40/32.40	475	120/80	1	1.00	-4.05	
Stage 82	6:50 32.80/32.80	480	120/80	1	1.00	-4.10	
Stage 83	6:55 33.20/33.20	485	120/80	1	1.00	-4.15	
Stage 84	7:00 33.60/33.60	490	120/80	1	1.00	-4.20	
Stage 85	7:05 34.00/34.00	495	120/80	1	1.00	-4.25	
Stage 86	7:10 34.40/34.40	500	120/80	1	1.00	-4.30	
Stage 87	7:15 34.80/34.80	505	120/80	1	1.00	-4.35	
Stage 88	7:20 35.20/35.20	510	120/80	1	1.00	-4.40	
Stage 89	7:25 35.60/35.60	515	120/80	1	1.00	-4.45	
Stage 90	7:30 36.00/36.00	520	120/80	1	1.00	-4.50	
Stage 91	7:35 36.40/36.40	525	120/80	1	1.00	-4.55	
Stage 92	7:40 36.80/36.80	530	120/80	1	1.00	-4.60	
Stage 93	7:45 37.20/37.20	535	120/80	1	1.00	-4.65	
Stage 94	7:50 37.60/37.60	540	120/80	1	1.00	-4.70	
Stage 95	7:55 38.00/38.00	545	120/80	1	1.00	-4.75	
Stage 96	8:00 38.40/38.40	550	120/80	1	1.00	-4.80	
Stage 97	8:05 38.80/38.80	555	120/80	1	1.00	-4.85	
Stage 98	8:10 39.20/39.20	560	120/80	1	1.00	-4.90	
Stage 99	8:15 39.60/39.60	565	120/80	1	1.00	-4.95	
Stage 100	8:20 40.00/40.00	570	120/80	1	1.00	-5.00	
Stage 101	8:25 40.40/40.40	575	120/80	1	1.00	-5.05	
Stage 102	8:30 40.80/40.80	580	120/80	1	1.00	-5.10	
Stage 103	8:35 41.20/41.20	585	120/80	1	1.00	-5.15	
Stage 104	8:40 41.60/41.60	590	120/80	1	1.00	-5.20	
Stage 105	8:45 42.00/42.00	595	120/80	1	1.00	-5.25	
Stage 106	8:50 42.40/42.40	600	120/80	1	1.00	-5.30	
Stage 107	8:55 42.80/42.80	605	120/80	1	1.00	-5.35	
Stage 108	9:00 43.20/43.20	610	120/80	1	1.00	-5.40	
Stage 109	9:05 43.60/43.60	615	120/80	1	1.00	-5.45	
Stage 110	9:10 44.00/44.00	620	120/80	1	1.00	-5.50	
Stage 111	9:15 44.40/44.40	625	120/80	1	1.00	-5.55	
Stage 112	9:20 44.80/44.80	630	120/80	1	1.00	-5.60	
Stage 113	9:25 45.20/45.20	635	120/80	1	1.00	-5.65	
Stage 114	9:30 45.60/45.60	640	120/80	1	1.00	-5.70	
Stage 115	9:35 46.00/46.00	645	120/80	1	1.00	-5.75	
Stage 116	9:40 46.40/46.40	650	120/80				

period was uneventful and patient was discharged with advice to follow up after 4 weeks.



FIGURE 3



FIGURE 4A

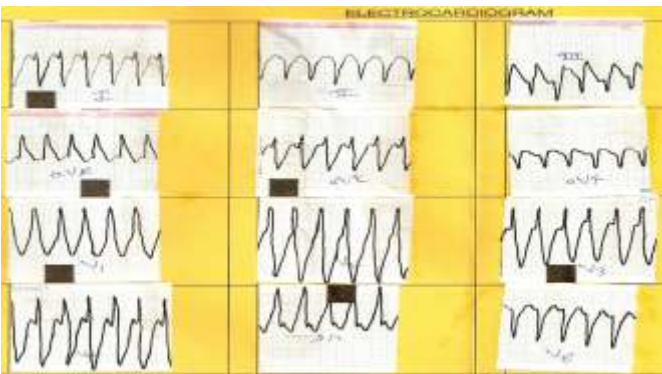


FIGURE 4B



FIGURE 5



FIGURE 6A, 6B



FIGURE 7

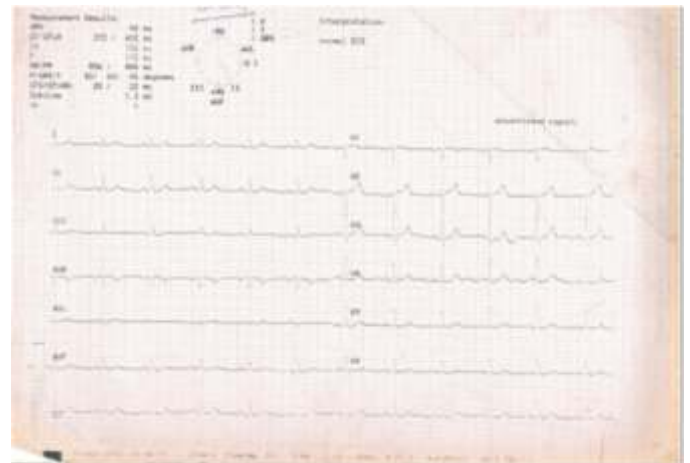


FIGURE 8

CASE 3

A 60 year old male (Fig 7) presented to hospital with complaints of chest pain squeezing type, diffuse in nature, precordial in origin radiating to the arms. The history of honey bee bite 4 hours prior to the symptoms was revealed during the history taking. The patient had no dyspnoea, no palpitation, no sweating associated with the chest pain. No history of diabetes, hypertension or any prior cardiac or medical illness was obtained. Cardiac examination was normal. ECG (Fig 8) and Echocardiography were normal although cardiac enzymes Trop I was raised on day of admission which showed a rising trend with repeat samples. The patient underwent coronary angiography in view of the raised cardiac enzymes and symptoms at presentation. CAG revealed normal coronaries.

DISCUSSION

The term “enormous animals” is usually applied to a creature capable of producing a poison in a secretory gland and delivering that toxin during biting or stinging. Arthropods, such as spiders, scorpions, and hymenoptera (bees, wasps, yellow jacket) are found worldwide, and some of them are venomous animals.

Bee sting venom generally consists of complex mix of proteins, peptides and enzyme. The venom is mixed with water, so the actual

composition of the substance it injects into you is around 88% water and 12% venom. The main toxic component of bee venom, also referred to as apitoxin, is melittin. Melittin is a peptide that comprises around 50-55% of dry venom, and is a compound that can break up cell membranes, resulting in the destruction of cells. However, it's not considered the most harmful component of bee venom; that prize goes to an enzyme that makes up around 10-12%, phospholipase A. This enzyme destroys phospholipids, and also breaks down the membranes of blood cells, resulting in cell destruction; additionally, unlike the majority of larger molecules in the venom, it causes the release of pain-inducing agents. Yet another enzyme, hyaluronidase, aids the action of the venom by catalysing the breakdown of protein-polysaccharide complexes in tissue, allowing the venom to penetrate further into the flesh.

Other, smaller molecules can also contribute towards painful effects. A small amount of histamine is found in bee venom; histamine is one of the compounds released by the body during the allergic response, and can cause itchiness and inflammation. The proteins in the sting can cause an allergic reaction, leading to the release of even more histamine, and possible anaphylaxis. MCD peptide, another minor component of the venom, can also cause mast cells in the body to release more histamine, worsening inflammation.

Hymenoptera (bees, wasps) stings or bites are responsible for far more deaths than those due to all other poisonous creatures. The most frequent clinical events are due to immediate type of hypersensitivity reactions leading to hypotension, dyspnea, anaphylactic shock and angioedema. Acute myocardial infarction occurs very rarely after an arthropod envenomation. There are a few cases of AMI due to bee or scorpion bites reported in the literature.¹⁻⁴ In these patients, coronary arteries were normal or non-significantly stenotic.

However, the exact mechanism of AMI caused by arthropod envenomation is unclear. Several reports deal with the cardiovascular complications after hymenoptera stings.^{2,5,6,7} The important medical problem posed by the stings is the development of anaphylactic shock. Anaphylaxis leads to hypotension causing vasodilation and decrease of intravascular volume.^{2,5,6,7}

Many pharmacologically active constituents of venoms have been isolated.⁸ These substances can provoke ischemia and even myocardial injury due to profound hypotension or by increasing oxygen demands through direct inotropic and chronotropic effects in the presence of compromised myocardial supply.

Electrocardiographic changes consistent with acute myocardial ischemia or infarction, including ST depression or elevation and even the appearance of pathologic Q-waves, have been recorded in people after stings.^{2,9,10}

Rhythm abnormalities such as supraventricular arrhythmias, VPC's, junctional rhythm and right bundle branch block have been recorded during initial stages after the sting.^{2,6,9} Animal studies of bee venom have shown that such ECG changes may be due to direct cardiotoxic effect. However, the mechanism is still not clear.

Laboratory parameter, such as elevated CPK, SGOT, Trop I and T have been noted in people with stings which indicates myocardial injury.^{2,6,9} The rise in serum creatine kinase and creatine kinase-MB levels may be attributed to myocardial injury and/or rhabdomyolysis caused by extremely high sympathetic discharge. Elevation of the more specific marker for myocardial injury like cardiac troponin I or T following sting indicates direct or indirect myocardial injury.

Transthoracic echocardiography showing regional wall motion abnormalities (hypokinesia and akinesia) and left ventricular dysfunction after a bite has been reported.⁴ The stunned myocardium is known as a prolonged postischemic LV dysfunction after brief myocardial ischemia and represents a reversible LV dysfunction.⁸ Abrough et al. reported gradual normalization of wall motion abnormality in the left ventricle and septum, and also complete restoration of the systolic function in cases with severe bites.¹⁰

The change in echocardiographic findings of our cases are similar to that of above reported cases.^{4,10} Moreover, clinical, electroca-

rdiographic, laboratory and echocardiographic findings in our cases excluded the possibility of myocarditis or pericarditis.

Various differential diagnosis that could be possible with such a clinical and laboratory presentation are Hereditary Thrombophilia, Kouniss syndrome, Takotsubo cardiomyopathy.

Two cases are described with the possible association between hereditary thrombophilia and arthropod bite giving rise to AMI without any evidence of atherosclerotic heart diseases.⁴ The authors therefore recommend that patients presenting with AMI following an arthropod bite should be screened for any inherited thrombophilia.⁴ Another two patients who were stung by wasps and honeybee, respectively, developed Kounis syndrome as a consequence of allergic reaction.¹¹ Kounis syndrome is the concurrence of acute coronary syndrome with mast cell activation induced by allergic or hypersensitivity and anaphylactic of anaphylactoid reactions.

Takotsubo cardiomyopathy (Takotsubo CM) is a novel cardiac syndrome characterized by transient and severe LV apical ballooning and basal hyperkinesia in acute stage.^{12,13} Although Takotsubo cardiomyopathy shows striking initial manifestations mimicking AMI, the minimal change of cardiac enzymes are not consistent with the extent of LV change in acute stage, and unusual LV morphology was restored to normal, usually within several weeks, in most cases.^{12,13} In patients with Takotsubo CM, the ECG in acute stage shows concave ST-segment elevation, usually in leads V3-6, there is less dynamic change for days, followed by T-wave inversion and resolved in approximately 2-3 weeks^{6,16} associated with QTc prolongation.¹⁶ Abnormal Q-wave and reciprocal changes are rarely seen.¹⁴

It is well known that systemic anaphylaxis with bronchospasm, larynx edema and hypotension may ensue following hymenoptera stinging. In the most severe cases, the symptoms of cardiovascular system are predominant; therefore, stenocardial troubles and accelerated and irregular heart rate may develop.¹⁵

The electrocardiographic (ECG) changes^{16,17,18} as well as chest pain¹⁹ were described. The acute myocardial infarction following an insect sting of Hymenoptera (bees, wasps, hornets) occurs rarely.²⁰⁻²⁵ Anaphylactic reactions after different insects sting may induce cardiovascular events, including acute myocardial infarction. These substances are responsible for direct venom cardiotoxicity causing vasoconstriction and platelet aggregation.^{20,26}

Several of the venom proteins and peptides are allergenic. These allergens, especially phospholipase A2, can cause endogenous amine release from mast cells during anaphylactic reaction.

The main mechanisms responsible for myocardial infarction might be coronary arterial spasm and/or secondary in situ thrombosis.^{22,18,28-30}

Our report focuses on cases of acute myocardial infarction after a bee sting. Bee venom contains epinephrine, dopamine, leukotrienes and thromboxanes, which cause severe platelet aggregation and direct vasoconstriction, therefore paradoxical vasoconstriction is a possible explanation as an underlying mechanism. Severe coronary arterial spasm or secondary in situ thrombosis may also play a role in such cases.³¹ Clinical and pathophysiological background of AMI after bee sting are generally related with three different mechanisms; AMI due to anaphylaxis and shock, a typical AMI occurring in patients with coronary atherosclerosis and an AMI occurring in subjects without significant coronary artery disease in whom coronary thrombosis and vasoconstriction are enhanced by intoxication.⁵ Several cases of AMI were reported after envenomation with different animals such as snakes, wasps and several different insects. Authors have postulated that venom constituents can cause endogenous amine release and vasodilatation leading to endothelial dysfunction³², also postulated that it was possible that adverse effects of therapeutic doses of epinephrine could be responsible for the reaction. Primary coronary artery vasospasm (usually associated with chest pain and an ischemic pattern on the ECG) was postulated to be the alternative pathophysiological hypothesis. Matucci et al. also recommend a consequence of an immunoglobulin E-related allergic reaction as another potential mechanism.³³ Clinical presentation may be quite different in AMI patients after bee stings. It may be completely silent³²

or ECG changes with overt ST wave elevation may take place several hours after admission of the patient, as in our cases. Therefore higher grade clinical suspicion is absolutely necessary in order to come up with the correct diagnosis. Also, serial ECG recordings with assessment of laboratory parameters are recommended in every patient who had encountered chest pain regardless of the severity of a patient's reaction to a bee sting.

In conclusion, hymenoptera (bee) venom can cause acute coronary syndrome by several pathogenetic mechanisms: release of allergenic proteins, vasoactive, inflammatory, and thrombogenic peptides and amine constituents (histamine, serotonin, bradykinin, leukotrienes, thromboxane), which act on the coronary vasculature and induce coronary artery vasospasm and facilitate platelet aggregation as well as thrombosis; direct cardiotoxic effect of the venom; and anaphylactic reactions.

REFERENCES

1. Gueron M, Stern J, Cohen W. Severe myocardial damage and heart failure in scorpion sting: report of five cases. *Am J Cardiol* 1967;19:719-26.
2. Levine HD. Acute myocardial infarction following wasp sting. *Am Heart J* 1976;91:365-74.
3. Wagdi P, Mehan VK, Burgi H, Salzmann C. Acute myocardial infarction after wasp stings in a patient with normal coronary arteries. *Am Heart J* 1994;128:820-3.
4. Kayikcioglu M, Eroglu Z, Kosova B, et al. Acute myocardial infarction following an arthropod bite: Is hereditary thrombophilia a contributing factor? *Blood Coagulation & Fibrinolysis* 2006;17:581-3.
5. Ceyhan C, Ercan E, Tekten T, Kirilmaz B, Onder R. Myocardial infarction following a bee sting. *Int J Cardiol* 2001; 80:251-3.
6. Brasher GW, Sanchez SA. Reversible electrocardiographic changes associated with wasp sting anaphylaxis. *JAMA* 1974;229: 1210-1.
7. Maguire JH, Spielman A. Ectoparasite Infestations and Arthropod Bites and Stings, In: Harrison's Principles of Internal Medicine. *New York: McGraw Hill* 1988:2251
8. Braunwald E, Kloner RA. The stunned myocardium: prolonged, postischemic ventricular dysfunction. *Circulation* 1982;66:1146-9.
9. Law DA, Beto RJ, Dulaney J, et al. Atrial flutter and fibrillation following bee stings. *Am J Cardiol* 1997;80:1255.
10. Abrough F, Ayari M, Nouira S, et al. Assessment of left ventricular function in severe scorpion envenomation: Combined hemodynamic and echo-doppler study. *Int Care Med* 1995;21:629-35.
11. Kogias J, Sideris S, Anifadis S. Kounis syndrome associated with hypersensitivity to hymenoptera stings. *International Journal of Cardiology* 2007;114:252-5
12. Tsuchihashi K, Ueshima K, Uchida T, et al. Transient left ventricular apical ballooning without coronary artery stenosis: a novel heart syndrome mimicking acute myocardial infarction. Angina Pectoris-Myocardial Infarction Investigations in Japan. *J Am Coll Cardiol* 2001;38:11-8.
13. Chiou CS, Chang NC, Shih CM, et al. Takotsubo cardiomyopathy associated with jet-lag syndrome in a Taiwanese elderly woman: a case report and literature review. *Taiwan Geriatr Gerontol* 2006; 2:130-41.
14. Ogura R, Hiasa Y, Takahashi T, et al. Specific findings of the standard 12-lead ECG in patients with 'Takotsubo' cardiomyopathy: comparison with the findings of acute anterior myocardial infarction. *Circ J* 2003;67:687-90.
15. Fisher BA, Antonios TF. Atrial flutter following a wasp sting. *J Postgrad Med* 2003; 49(3): 254-5.
16. Castberg T, Schwartz M. Changes in the electrocardiogram during allergic shock. *Acta Med Scand* 1974; 126: 459-71.
17. Brasher GW, Sanchez SA. Reversible electrocardiographic changes associated with wasp sting anaphylaxis. *JAMA* 1974;229(9): 1210 1.
18. Epelde F, SáenzCusi L, Alvarez Auñón A. Myocardial ischemia after a wasp sting. *An Med Interna* 2001; 18(4): 219. (Spanish)
19. Milne MD. Unusual case of coronary thrombosis. *Br Med J* 1949; 1(4616): 1123.
20. Levine HD. Acute myocardial infarction following wasp sting. Report of two cases and critical survey of the literature. *Am Heart J* 1976; 91(3): 365 74.
21. Jones E, Joy M. Acute myocardial infarction after a wasp sting. *Br Heart J* 1988; 59(4): 506 8.
22. Wagdi P, Mehan VK, Burgi H, Salzmann C. Acute myocardial infarction after wasp stings in a patient with normal coronary arteries. *Am Heart J* 1994; 128(4): 820-3.
23. Larsen SL. Acute myocardial infarction following a wasp sting in a patient with normal coronary vessels. *Ugeskr Laeger* 2000; 162(36): 4819-20.
24. Calveri G, Bertelli Y, Caico SI, Ermolli NC, Torretta M, Lattanzio M, et al. Acute myocardial infarction after wasp sting. *Ital Heart J Suppl* 2002; 3(5): 555 7. (Italian)
25. Ceyhan C, Ercan E, Tekten T, Kirilmaz B, Onder R. Myocardial infarction following a bee sting. *Int J Cardiol* 2001; 80(2-3): 251-3.
26. Moffitt JE. Allergic reactions to insect stings and bites. *South Med J* 2003; 96(11): 1073 9.
27. Massing JL, Bentz MH, Schlessner P, Dumitru C, Louis JP. Myocardial infarction following a bee sting. Apropos of a case and review of the literature. *Ann Cardiol Angeiol* 1997; 46(5-6): 311-5. (French)
28. Wong S, Greenberger PA, Patterson R. Nearly fatal idiopathic anaphylactic reaction resulting in cardiovascular collapse and myocardial infarction. *Chest* 1990; 98(2): 501-3.
29. Antonelli D, Koltun B, Barzilay J. Transient ST segment elevation during anaphylactic shock. *Am Heart J* 1984; 108(4 Pt 1): 1052-4.
30. Erbilien E, Gulcan E, Albayrak S, Ozveren O. Acute myocardial infarction due to a bee sting manifested with ST wave elevation after hospital admission. *South Med J* 2008; 101(4): 448.
31. Massing JL, Bentz MH, Schlessner P, Dumitru C, Louis JP. Myocardial infarction following a bee sting. Apropos of a case and review of the literature. *Ann Cardiol Angiol (Paris)* 1997; 46: 311-5.
32. Lombardi A, Vandelli R, Cere E, Di Pasquale G. Silent acute myocardial infarction following a wasp sting. *Ital Heart J* 2003; 4: 638-41.
33. Matucci A, Rossi O, Cecchi L, et al. Coronary vasospasm during an acute allergic reaction. *Allergy* 2002; 57: 867