Acute myocardial infarction (AMI) after bee sting has been rarely reported in the literature. Bee venom contains epinephrine, dopamine, leukotrienes and thromboxanes, which cause severe platelet aggregation and direct vasoconstriction, therefore can lead to an acute myocardial infarction. Here, we describe a 44 year old man with acute myocardial infarction with ST-segment elevation after a bee sting treated with primary percutaneous coronary intervention (PCI).

Key Words: Myocardial infarction; bee venom

One hour after admission his chest pain was increased substantially. ECG was repeated and demonstrated ST wave elevation on chest leads (Figure 1B). The diagnosis of acute anterior myocardial infarction was concluded and primary percutaneous coronary intervention (PCI) was decided. Aspirin 300 mg was given. Coronary angiography was performed which revealed total occlusion of proximal left anterior descending artery (LAD) (Figure 2-left panel) and non critical lesions in right coronary artery (RCA). The culprit lesion in the infarct related artery was predilated with 2.0x20 mm balloon and a 4.0x20 mm Ephesos (Nemed Corp, Istanbul, TURKEY) stent was implanted successfully in the proximal LAD without any complications (Figure 2-right panel). The patient’s chest pain relieved completely after PCI and elevated ST waves were restored by 70%. Echocardiographic examination showed anterior hypokinesis. The ejection fraction of the left ventricle was 50%. In-hospital stay was without any complications and he was discharged on seventh day of admission.

Our report focuses on a case 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 role in such cases.²

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 vasospasm enhanced by intoxication.¹

Several cases of AMI were reported after envenomation with different animals such as snakes, wasps and several different insects. One of these cases was a 67-year-old male patient who had a silent AMI after wasp sting.³ The authors postulated that venom constituents can cause endogenous amine release and vasodilatation leading to endothelial dysfunction. They also postulated that it was possible that adverse effects of therapeutic doses of epinephrine could be responsible for the reaction. This case history does suggest that the episode of hypotension (syncope) could have been a major factor in the pathogenesis of the myocardial infarction as in our case.
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.\(^4\)

Clinical presentation may be quite different in AMI patients after bee stings. It may be completely silent\(^3\) or ECG changes with overt ST wave elevation may take place several hours after admission of the patient, as in our case. Therefore higher grade clinical suspicion is absolutely necessary in order to come up with the correct diagnosis. Also, serial ECG recordings are recommended in every patient who had encountered chest pain regardless of the severity of a patient’s reaction to a bee sting.

**REFERENCES**