Journal of the Postgraduate Institute of Medicine 2025; 12(2): E276 1-7

https://doi.org/10.4038/jpgim.v12i2.8576

Case Report

Myocardial infarction triggered by a giant honeybee sting without atopy or anaphylaxis: A case of suspected Kounis syndrome

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Key words: **Kounis syndrome (KS), myocardial infarction, bee sting, Apis dorsata, Hymenoptera venom**

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Received: 25 Jun 2025, Accepted: 10 Sep 2025, Published: ~~01 Oct~~ 2025

Competing Interests: Authors have declared that no competing interests exist

Introduction

Giant honeybee (*Apis dorsata*) envenomation is a common health hazard frequently reported in hill country and North Central Province of Sri Lanka. Although most cases are limited to minor local reactions or mild anaphylaxis reactions, sometimes it has led to severe reactions and complications such as severe anaphylactic shock, disseminated intra vascular coagulation and acute kidney injury, cardiac complications, encephalitis and multi organ failure. Although acute myocardial infarction is a well-known complication of wasp bite, the case reports in the medical literature are scarce [1,2,3]. With the wide distribution of wasp colonies in treetops in mountainous areas, rocks and ancient pilgrimages, the potential target groups such as travelers, tea estate workers, school children and farmers have been reported in mass casualties in Sri Lanka which highlights the importance of awareness on this issue among clinicians on associated potential complications and its management.

We report a case of a 61-year-old male, presented with ischemic type chest pain associated with autonomic symptoms, admitted to the cardiology unit who was subsequently diagnosed with acute ST elevation myocardial infarction. The patient was stung by 50-60 giant honeybees while working in a rubber crop near a reservoir in Western Province, Sri Lanka. Two hours later, the event was followed by the onset of cardiac symptoms. Acute inferior myocardial infarction was confirmed with electrocardiography findings and elevated cardiac troponin I Torres. Further evaluation with echocardiogram confirmed the myocardial dysfunction of inferior territory (EF=50%) and coronary angiography showed thrombus formation in the right coronary artery.

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Case Presentation

A 61-year-old male with a history of diabetes mellitus presented to the Base Hospital Wathupitiwala in December 2024. He had been attacked by multiple bee stings while working on a rubber estate near a reserved forest in Wewaldeniya, Gampaha (Figure 01). Around 8.45 in the morning, the patient was stung by giant honey bees, approximately 50-60 stings. After the arrival to the hospital within an hour, he complained of severe localized pain and swelling at the sting sites, including his lips, but did not exhibit any signs of wheezing, vomiting, or itching. Initial treatment at the Primary Care Unit (PCU) involved the removal of 34 stingers, where 12 additional stingers were removed in the ward.

Figure 01: Giant honeybees found at the site of bee attack

However, by 2.30 in the evening, the patient had developed central chest tightness, nausea (without vomiting), and shortness of breath, though no wheezing was noted during the initial investigation. The 12-lead electrocardiogram (ECG) revealed an acute inferior myocardial infarction through ST segment elevations in leads II, III, and aVF, (Figure 02). After 45 minutes, the patient was thrombolysed with Tenecteplase. Despite this, the patient left the hospital against medical advice and sought care at Sri Jayewardenepura General Hospital at 11.30 p.m. on the same day.

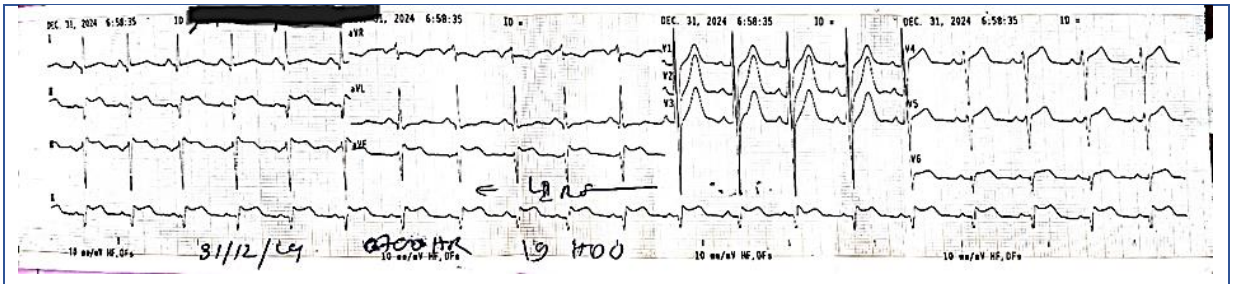

Figure 02: The 12-lead electrocardiogram (ECG)

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Upon re-admission, the patient did not report symptoms such as chest pain, chest tightness, nausea, or shortness of breath. On examination, he was alert, oriented, and rational. Multiple bite marks with erythematous patches were noted across his body (Figure 03).

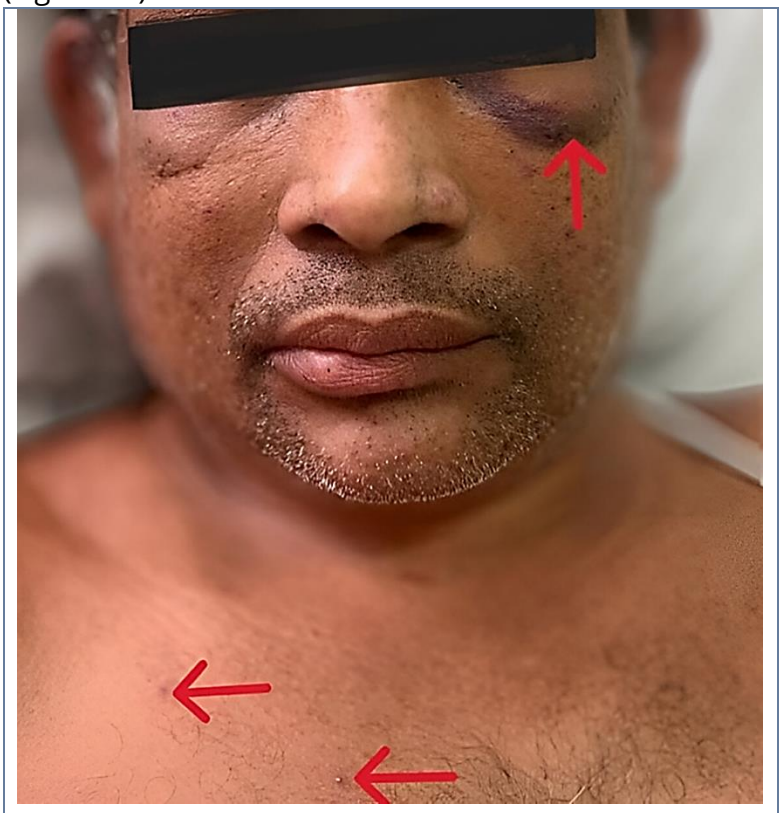

Figure 03: Bite marks with erythematous patches

He was hemodynamically stable, with a radial pulse rate of 99 beats per minute, blood pressure of 128/90 mmHg, and oxygen saturation of 98% with room air (Table 01). Repeat ECG was performed which confirmed acute inferior ST elevation myocardial infarction. His Troponin I was >50.0 (red <0.036). Full blood count showed white blood cell (WBC) count of 22,730 (4000-11000) cells/mm^{-3} with predominant neutropenia, hemoglobin of 14.4 g/dL and platelet count of 298,000 cells/mm^{-3}. His serum electrolytes were normal with sodium of 139 mmol/L and potassium of 3.8 mmol/L. His blood urea was 30 mg/dL, and serum creatinine was 85 mmol/L. His liver enzymes were elevated with aspartame amino transferase of 497 U/L (8-48 U/L) and alanine amino transferase of 251 U/L (7-55 U/L). His Pt/ INR was 1.04 (1.0-1.4) (Table 01). Echocardiogram showed ejection fraction of 50% with hypokinetic inferior wall.

Table 01: Results of lab investigations

Parameters	Patient Values	Reference range
Pulse rate	99 bpm	60 – 100 bpm
Blood pressure	128/90 mmHg	90/60 – 120/80 mmHg
Oxygen saturation	98%	95 – 100%
Troponin I	>50.0	<0.036
WBC count	22,730 cells/mm^{-3}	4000-11000 cells/mm^{-3}
Hemoglobin	14.4 g/dL	14 – 18 g/dL
Platelet count	298,000 cells/mm^{-3}	150,000 – 400,000 cells/mm^{-3}
Serum Sodium	139 mmol/L	135 – 147 mmol/L
Serum Potassium	3.8 mmol/L	3.6 – 5.2 mmol/L
Blood urea	30 mg/dL	17 - 43 mg/dL
Serum creatinine	85 mmol/L	60 – 100 mmol/L
Aspartame amino transferase	497 U/L	8-48 U/L
Alanine amino transferase	251 U/L	7-55 U/L
Pt/ INR	1.04	1.0-1.4

The patient was started on subcutaneous enoxaparin and continued cardiac monitoring. On day 2 of the admission, a coronary angiogram was performed, and it revealed a dominant large vessel with mild disease in the mid-vessel and a thrombus in the distal posterior left ventricular artery (PLV) (Figure 04). Based on the findings, he was diagnosed with single-vessel disease. He was managed conservatively with medical therapy, including enoxaparin for three days, antiplatelet agents, and statins. The patient was discharged in stable condition after five days of successful inpatient treatment.

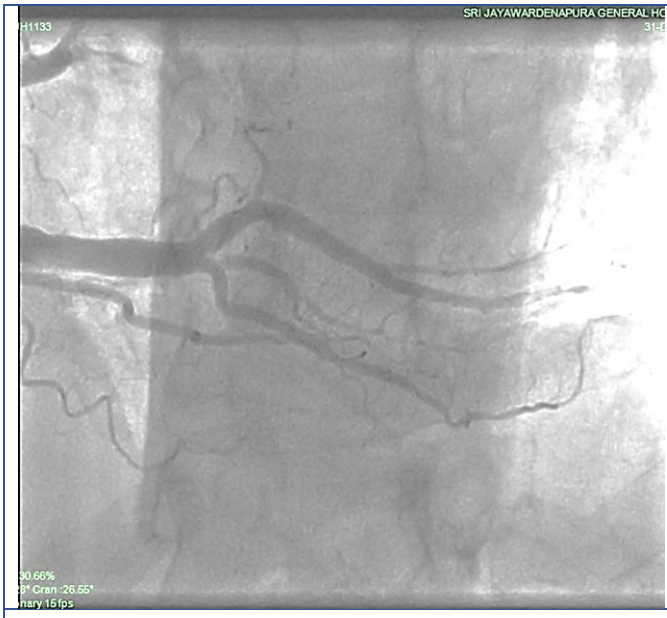

Figure 04: Coronary angiogram of thrombus in the distal posterior left ventricular artery

https://doi.org/10.4038/jpgim.8576

Discussion

Kounis syndrome (KS) is defined as cardiovascular symptoms (acute coronary syndrome -ACS) that occur secondary to an allergic or hypersensitivity reaction [4]. It was first described in 1991 by N. G. Kounis in a case report of allergic angina [5], where he describes the key role of histamine released secondary to activation and degradation of mast cells following induction by IgE. Following the first description of KS, it has now evolved to be described in 3 variants according to the pathophysiology. The first type is ACS secondary to coronary spasms, while 'coronary thrombosis' and 'ACS in drug-eluting stents' are described as the second and third types, respectively [6]. In our case report, it is the type 2 ALS where it revealed thrombus formation in invasive coronary angiogram. A wide range of precipitants of KS has been described in medical literature, including a wide range of medications, clinical conditions such as atopy, bronchial asthma, antipodes and food allergies, Churg-Strauss syndrome, serum sickness, and exercise-induced anaphylaxis. Further, an interesting category of environmental exposures includes viper venom, shellfish eating, jellyfish stings, limpet ingestion, and a wide range of insect stings, including ants and Hymenoptera species [7].

Hymenoptera venom is a complex mixture of simple organic molecules, proteins, peptides and other bioactive molecules [8]. Its clinical manifestations range from mild local reactions to rarely life-threatening complications including severe anaphylaxis and organ dysfunction. So far, the anaphylaxis is the most common manifestation, accounting for ~95% of cases, though the unusual systemic reactions, such as serum sickness, diffuse alveolar hemorrhages, cerebral stroke, glomerulonephritis, acute kidney injury, disseminated intravascular coagulation and thrombocytopenia purpura, are reported [9,10].

Hymenoptera venom induced myocardial infarction can occur either by inducing anaphylactic reaction or by direct action of venom [11]. IgE mediated anaphylactic reaction induced by venom results in activation of mast cells which causes degranulation and release of mediators such as histamine, leukotrienes and cytokines into the circulation [12]. Mast cells are prominently found in coronary arteries during coronary spasms and during coronary plaque ruptured events [13,14]. Hymenoptera venom induced myocardial infarction could be severe or prolonged in individuals with atopy and in some cases, anaphylaxis may occur in association [15]. In the second mechanism, it is postulated that the mast cell activation triggers release of proteinases capable of activating matrix metalloproteinases results in coronary plaque rupture [13].

In the diagnosis of ACS following a bee sting, a high index of clinical suspicion is required due to the wide range of clinical symptoms ranging from silent ischemia [16] to atypical angina pectoris and the unpredictable wide range of time intervals between the sting and the onset of clinical features. In case of suspected myocardial infarction, it is important to perform early ECGs and biochemical confirmation with cardiac troponin. ECG changes may vary from normal to ST elevation, ST depression or even arrhythmias.

The initial management should include supplementary oxygen, analgesics, corticosteroids, adequate hydration and inotrope support depending on the individual's clinical condition. Early removal of stings would be helpful in lessening further envenomation. It is recommended to avoid beta blockers as their alpha-adrenergic properties may precipitate coronary spasms. Further, morphine should be avoided as it may trigger the release of histamine, resulting in paradoxical worsening of the clinical course. Further management should consist of the re-establishment of coronary perfusion immediately with primary PCI or thrombolysis based on available resources. Our patient was managed with thrombolysis with tenecteplase and an early angiogram performed with the intention of early PCI (percutaneous coronary intervention), but abandoned due to unsuitable coronary anatomy.

In summary, we emphasize the importance of early identification of ACS in a patient presenting following a bee bite with high index of suspicion, which may result in minimizing cardiac complications. Further, early application of appropriate pharmacological management and interventions is essential.

Acknowledgements

We would like to express our sincere gratitude to the patient for his cooperation and consent to share his case.

We also thank Professor Inoka Karunaratne of the Department of Zoology, Faculty of Science, University of Peradeniya, for her expert guidance in identifying the honeybee species involved in this case report.

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