

Kounis Syndrome Presenting with Atrial Fibrillation and Shark-Fin ST Elevation After Multiple Bee Stings in an African Patient: A Case Report

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Background: Kounis syndrome is an acute coronary syndrome triggered by allergic or hypersensitivity reactions, mediated by inflammatory cascades and mast cell activation. It can manifest as coronary vasospasm, plaque rupture, or stent thrombosis. Electrocardiographic presentations vary and may mimic ST-elevation myocardial infarction, occasionally with atypical features such as atrial fibrillation or extreme ST-segment elevation patterns.

Case Presentation: We report a case of an adult African patient who developed acute chest pain, hypotension, and localized allergic manifestations (pruritus and soft tissue swelling at sting sites) following multiple bee stings. Notably, no generalized urticaria or angioedema was observed. Electrocardiography revealed atrial fibrillation with a “shark-fin” ST elevation pattern, a combination not previously reported in an African patient with Kounis syndrome suggestive of extensive myocardial ischemia. Emergent management included hydrocortisone, diphenhydramine, epinephrine infusion, and intracoronary nitroglycerin. Coronary angiography and clinical evolution supported a diagnosis of coronary vasospasm highly consistent with type I Kounis syndrome, in the absence of confirmatory biomarker testing.

Discussion: Kounis syndrome remains underrecognized, particularly in low-resource settings. The coexistence of atrial fibrillation and shark-fin ST elevation is rare and represents the distinguishing novelty of this case; it may lead to misdiagnosis as primary myocardial infarction or ventricular tachycardia. The confounding role of exogenous epinephrine administered during resuscitation must be acknowledged in interpreting the angiographic findings. Allergic mediators such as histamine and leukotrienes can induce coronary vasospasm and electrical instability. Early recognition is critical, as management requires simultaneous treatment of both allergic and cardiac components.

Conclusion: Kounis syndrome should be considered in patients presenting with acute coronary syndromes in the context of allergic triggers such as bee stings. The simultaneous occurrence of atrial fibrillation and shark-fin ST elevation is a rare, diagnostically challenging presentation. Prompt recognition and dual-pathway management can improve outcomes.

Keywords: Kounis syndrome, bee stings, coronary artery spasm, shark-fin ST elevation, atrial fibrillation, African patient

Introduction

Kounis syndrome is an acute coronary syndrome triggered by allergic or hypersensitivity reactions. It was first described by Kounis and Zavras in 1991 as “allergic angina” resulting from histamine-induced coronary vasospasm.^{1,2} Mast cell activation leads to the release of vasoactive and pro-inflammatory mediators, including histamine, leukotrienes, and cytokines, which can induce coronary vasospasm, plaque erosion or rupture, and stent thrombosis.^{1,2}

Three clinical variants are recognized. Type I occurs in patients without pre-existing coronary artery disease and is characterized by coronary vasospasm; Type II involves plaque rupture or erosion in patients with underlying atherosclerosis; and Type III is associated with stent thrombosis due to allergic inflammation.^{1,2}

Hymenoptera stings are established but uncommon triggers of Kounis syndrome. Most reported cases originate from Europe and Asia, with limited data from African populations likely reflecting underreporting rather than low prevalence, given high occupational exposure through subsistence farming, beekeeping, and outdoor agricultural activities. Clinical presentation can mimic acute myocardial infarction, and atypical electrocardiographic findings may complicate diagnosis.^{3–5}

We report a case of Kounis syndrome in a previously healthy African patient following multiple bee stings, complicated by ventricular fibrillation arrest, atrial fibrillation, and a “shark-fin” ST elevation pattern on electrocardiography. To our knowledge, the simultaneous occurrence of shark-fin ST elevation and atrial fibrillation in a case of Kounis syndrome following bee stings has not been previously reported in an African patient, and represents a uniquely challenging diagnostic constellation. The diagnosis is clinically and angiographically supported; however, the absence of serum tryptase data and the confounding effects of high-dose epinephrine administered during resuscitation preclude definitive biomarker confirmation. This case is reported in accordance with CARE guidelines.^{6,7}

Case Presentation

A 67-year-old African male subsistence farmer presented following multiple bee stings sustained while gardening at home. He had no prior history of asthma, atopy, or allergic reactions to drugs, foods, environmental exposures, or Hymenoptera venom. He had no known cardiovascular risk factors, including hypertension, diabetes mellitus, dyslipidemia, or smoking, and he was not on any regular medications. There was no family history of premature coronary artery disease, sudden cardiac death, or atopic disease.

His recent medical history was unremarkable. He had attended routine outpatient visits for age-appropriate screening, with no previously documented electrocardiographic abnormalities and no previously elevated inflammatory markers such as C-reactive protein or erythrocyte sedimentation rate, insofar as available records indicated.

The timeline of clinical events is summarized in [Table 1](#).

Table 1 Timeline of Clinical Events from Bee Sting to Hospital Course

Time from Sting	Event
0 min	Multiple bee stings while gardening; onset of malaise and pruritus.
~5–10 min	Fall with head impact and seizure-like activity witnessed by family.
~15 min	EMS arrival: patient found unresponsive, evolving to ventricular fibrillation; cardiopulmonary resuscitation initiated.
Pre-hospital	Multiple defibrillation attempts; epinephrine and amiodarone administered per ACLS protocols; transient transitions between ventricular fibrillation and ventricular tachycardia noted.
ED arrival	Ongoing episodes of ventricular fibrillation with repeated defibrillation; ROSC obtained and maintained on vasopressor infusion (epinephrine).
ED, initial ECG	12-lead ECG recorded showing atrial fibrillation with QRS–ST fusion producing shark-fin morphology in inferior leads and inverted shark-fin pattern in V2; initially interpreted as wide-complex tachycardia and possible STEMI.
+70 min	Repeat ECG showed sinus rhythm with residual ST-segment abnormalities.
Early in-hospital	Bedside echocardiography showed moderately reduced left ventricular ejection fraction (35–40%) with global hypokinesis.
Same day	Coronary angiography: severe (>95%) vasospasm of diagonal branch of proximal LAD and moderate vasospasm of mid RCA (both on epinephrine infusion), resolving completely after intracoronary nitroglycerin; no fixed obstructive stenosis identified.
24–48 h	Vasopressors weaned; high-sensitivity troponin I peaked at 23,000 ng/L.
Subsequent days	Clinical stabilization, progressive improvement in hemodynamics and respiratory function.
Follow-up	Planned outpatient review with repeat echocardiography, venom-specific IgE testing, and allergy evaluation.

Notes: Original data from patient medical records at Mulago National Referral Hospital, Kampala, Uganda.

Physical Examination

On emergency medical services arrival, the patient was unresponsive (GCS 7: E3 V2 M2) with spontaneous eye opening and incoherent vocalization. He rapidly deteriorated into pulseless ventricular fibrillation and was apneic with no palpable pulses. Pupils were mid-sized and sluggishly reactive. No cutaneous features of anaphylaxis such as urticaria were observed, and no generalized angioedema was present, though localized soft tissue swelling was noted at multiple bee sting sites.

Following return of spontaneous circulation (ROSC) in the emergency department, the patient was intubated and sedated. Vital signs revealed persistent hypotension with systolic blood pressure 80–90 mmHg requiring epinephrine infusion, and an irregularly irregular heart rhythm consistent with atrial fibrillation. Peripheral perfusion was markedly poor with cold extremities. Jugular venous pressure was not elevated. Lung auscultation demonstrated bilateral basal crackles. Cardiac examination revealed no murmurs, rubs, or gallops. Neurologic examination was limited by sedation, though pre-sedation GCS remained 7 (E3 V2 M2).

Diagnostic Assessment

Electrocardiographic Findings and Interpretation

The first 12-lead ECG (Figure 1), obtained shortly after return of spontaneous circulation (ROSC) in the emergency department, showed an irregularly irregular rhythm without discernible P waves, consistent with atrial fibrillation. QRS complexes appeared markedly widened due to fusion with massively elevated ST segments, creating the characteristic “shark-fin” morphology across inferior leads II, III, and aVF, with a reciprocal inverted shark-fin pattern in lead V2. This ECG was initially interpreted by the treating team as wide-complex tachycardia, prompting urgent activation of the catheterization laboratory for presumed ST-elevation myocardial infarction.

Several features support the diagnosis of atrial fibrillation with ischemia-related QRS–ST fusion, rather than ventricular tachycardia. First, the ventricular rate was grossly irregular, which is inconsistent with the regular or near-regular rate



Figure 1 Initial 12-lead ECG obtained shortly after return of spontaneous circulation, demonstrating an irregularly irregular rhythm without discernible P waves consistent with atrial fibrillation, and extreme shark-fin ST-segment elevation in inferior leads (II, III, aVF) with a reciprocal inverted shark-fin pattern in lead V2. The apparent QRS widening reflects QRS–ST fusion caused by massively elevated ST segments, initially misinterpreted as wide-complex tachycardia. Key distinguishing features from ventricular tachycardia include the grossly irregular ventricular rate and concordant multi-lead ST elevation with reciprocal changes.

characteristic of monomorphic ventricular tachycardia. Second, the QRS morphology varied on a beat-to-beat basis in a pattern inconsistent with a stable ectopic focus. Third, concordant ST elevation was present across multiple contiguous inferior leads with reciprocal changes in V2, a pattern reflecting transmural ischemia rather than the repolarization abnormalities typical of ventricular tachycardia. Fourth, and critically, the repeat ECG at 70 minutes demonstrated spontaneous conversion to sinus rhythm with progressive ST-segment normalization, a course consistent with reperfusion of ischemia-induced atrial fibrillation, not spontaneous termination of ventricular tachycardia. Recognition of QRS pseudo-widening due to QRS–ST fusion, rather than true bundle branch block or a ventricular rhythm, is critical; absence of distinct QRS termination, concordant multi-lead ST elevation, and rapid serial evolution distinguish this presentation from primary conduction disease or a ventricular arrhythmia.^{8,9}

A repeat ECG approximately 70 minutes later (Figure 2) demonstrated conversion to sinus rhythm with resolution of atrial fibrillation and marked improvement in ST-segment elevation, though residual ischemic changes persisted across inferior leads, supporting the diagnosis of transient severe ischemia.

Laboratory and Imaging Investigations

Initial laboratory evaluation revealed severe metabolic acidosis with elevated serum lactate, consistent with prolonged hypoperfusion during resuscitation. Serum tryptase was not obtained acutely, which is an important limitation in confirming mast cell activation. High-sensitivity cardiac troponin I demonstrated progressive elevation, peaking at 23,000 ng/L approximately 24–48 hours after presentation.

Urgent coronary angiography (Figure 3) demonstrated severe (>95%) diffuse vasospasm in the diagonal branch arising from the proximal left anterior descending (LAD) artery, and moderate vasospasm in the mid right coronary artery (RCA), both documented while the patient remained on high-dose epinephrine infusion for hemodynamic support. No fixed obstructive stenosis was identified in the RCA, left circumflex artery, or main LAD; only mild non-obstructive plaque was noted throughout the coronary tree. Administration of intracoronary nitroglycerin (200 mcg) resulted in immediate and complete resolution of all vasospasm with no residual obstructive lesions.



Figure 2 Follow-up 12-lead ECG obtained approximately 70 minutes after the initial tracing, demonstrating spontaneous conversion to sinus rhythm with marked improvement in ST-segment elevation and residual inferior lead ischemic changes, supporting the diagnosis of transient severe ischemia with reperfusion.

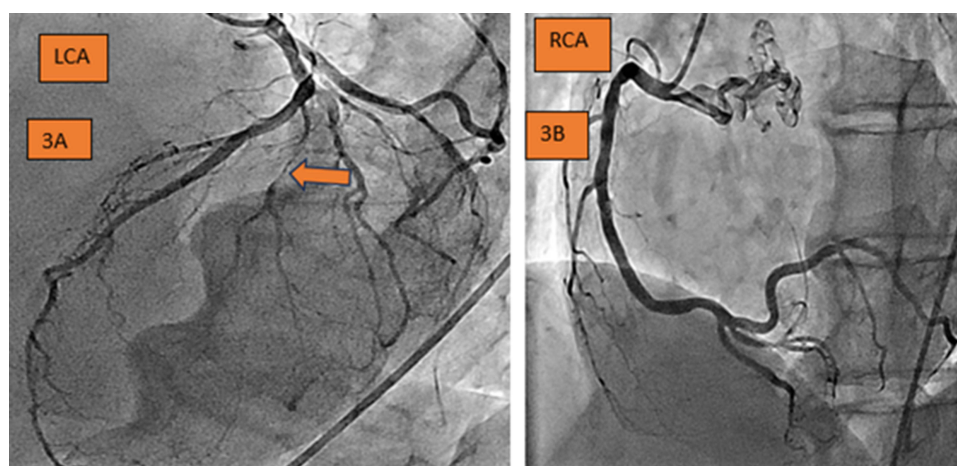


Figure 3 Coronary angiogram showing: (A) severe (>95%) diffuse vasospasm in the diagonal branch arising from the proximal left anterior descending (LAD) artery (Orange arrow indicates the site of maximal vasospasm); and (B) the right coronary artery (RCA) and left circumflex artery (LCx) demonstrating patent vessels with only mild non-obstructive plaque and no fixed obstructive stenosis. Both panels were obtained while the patient was on high-dose epinephrine infusion. Complete resolution of vasospasm occurred following intracoronary nitroglycerin administration.

Differential Diagnosis

The differential diagnosis included primary ST-elevation myocardial infarction with spontaneous reperfusion, catecholamine-induced myocardial injury and vasospasm from resuscitation epinephrine, stress cardiomyopathy, hypoxic-ischemic injury following cardiac arrest, and Kounis syndrome secondary to bee venom exposure.

Several findings favored Kounis syndrome as the most likely unifying diagnosis: the immediate temporal relationship to multiple bee stings while gardening, prodromal allergic symptoms of generalized pruritus and malaise, absence of fixed obstructive coronary artery disease, multivessel reversible vasospasm on angiography, and rapid improvement following spasm resolution. However, the extensive epinephrine administration during resuscitation represents a critical confounder, and absence of acute-phase serum tryptase measurement precludes definitive confirmation of mast cell degranulation.

Management

Acute Resuscitation and Coronary Intervention

Pre-hospital management involved bystander CPR followed by emergency medical services initiation of advanced cardiac life support protocols. The patient received multiple defibrillations for recurrent ventricular fibrillation, intravenous epinephrine boluses and amiodarone, and three rounds of ROSC with subsequent deterioration.

Upon emergency department arrival, the patient remained in refractory ventricular fibrillation requiring three additional defibrillations and initiation of epinephrine infusion at 0.1 mcg/kg/min for profound hypotension (systolic blood pressure 80–90 mmHg). He was emergently intubated and mechanically ventilated. Aspirin 325 mg and heparin were administered per acute coronary syndrome protocol given the initial ECG interpretation.

Due to concern for ST-elevation myocardial infarction, emergent coronary angiography was performed approximately 90 minutes after ED arrival. Severe multivessel coronary vasospasm affecting the proximal left anterior descending artery, mid right coronary artery, and diagonal branch was identified and treated with intracoronary nitroglycerin (200 mcg), resulting in complete angiographic resolution within seconds. No stents or other interventions were required.

Management of Allergic Component

Once the allergic trigger was recognized and coronary vasospasm confirmed angiographically as the ischemic mechanism, treatment was broadened to address the hypersensitivity reaction. Intravenous hydrocortisone 100 mg and diphenhydramine 50 mg were administered. The epinephrine infusion, initiated during initial resuscitation for refractory ventricular fibrillation arrest, was gradually weaned as hemodynamics stabilized post-nitroglycerin, avoiding further catecholamine-mediated vasoconstriction when possible.

Beta-blockers, morphine, and additional vasopressors were withheld due to concern for exacerbation of coronary spasm. Dual antiplatelet therapy beyond aspirin was deferred following negative angiography. The patient was transferred to the cardiac intensive care unit for ongoing mechanical ventilation, targeted temperature management (33–36°C for 24 hours), and neuroprotection per post-cardiac arrest protocols.

Follow-Up and Outcomes

The patient remained mechanically ventilated in the cardiac intensive care unit with initial persistent hemodynamic instability requiring low-dose epinephrine infusion. Over the subsequent 12 hours, vasopressors were successfully weaned as coronary vasospasm resolution improved cardiac output.

Serial ECGs showed progressive normalization of ST segments with T-wave inversion across inferior leads by hospital day 2. No recurrent ventricular arrhythmias occurred. High-sensitivity troponin I peaked at 23,000 ng/L at 24–48 hours before gradual decline.

Transthoracic echocardiography on day 3 demonstrated improvement in left ventricular systolic function with ejection fraction recovering to 45–50%. The patient was successfully extubated on hospital day 4 with no focal neurological deficits despite prolonged resuscitation.

Prior to discharge on hospital day 7, the patient and family received extensive counselling regarding Kounis syndrome, bee sting avoidance strategies, and recognition of anaphylactic symptoms. Arrangements were made for outpatient allergology evaluation including venom-specific IgE testing and consideration of Hymenoptera venom immunotherapy, along with cardiology follow-up with repeat echocardiography and stress testing.

Discussion

Kounis syndrome represents an acute coronary syndrome triggered by allergic or hypersensitivity reactions. First described by Kounis and Zavras in 1991 as histamine-mediated coronary vasospasm occurring during allergic events,^{1,2} it results from mast cell activation releasing histamine, tryptase, leukotrienes, and platelet-activating factor, which induce coronary vasospasm, endothelial dysfunction, and plaque destabilization.^{1,2,10} While drugs such as antibiotics and contrast media represent the most frequent triggers, Hymenoptera stings are well-recognized but less common precipitants.^{1,2} Most reported cases originate from Europe and Asia, with sparse documentation from sub-Saharan Africa despite frequent exposure to stinging insects.^{3–5,11}

This case is distinguished by the simultaneous occurrence of shark-fin ST elevation and atrial fibrillation in the setting of suspected Kounis syndrome following bee stings a combination not previously reported in an African patient and rarely described in the global literature. The shark-fin ECG pattern, reflecting fusion of the QRS complex, ST segment, and T wave into a characteristic triangular morphology, is a prognostic red flag associated with extensive transmural ischemia, high risk of malignant ventricular arrhythmias, and cardiogenic shock.^{8,9} Its co-occurrence with atrial fibrillation creates a particularly challenging diagnostic scenario in which the rhythm may be misidentified as ventricular tachycardia and the ST changes attributed to a primary conduction abnormality rather than ischemia.

In the present case, the initial ECG was interpreted as wide-complex tachycardia. However, several features are consistent with atrial fibrillation with QRS–ST fusion rather than ventricular tachycardia: the grossly irregular ventricular rate, beat-to-beat QRS morphological variability, concordant multi-lead ST elevation with reciprocal changes, and spontaneous conversion to sinus rhythm on the repeat tracing at 70 minutes. This distinction has important therapeutic implications: ventricular tachycardia would ordinarily prompt immediate cardioversion and antiarrhythmic therapy, whereas atrial fibrillation in the context of active ischemia should prompt treatment of the underlying ischemic insult.^{8,9} Clinicians encountering similar post-arrest ECG patterns should consider ischemia-induced atrial fibrillation with QRS–ST fusion as a differential diagnosis.

Atrial fibrillation in this context likely resulted from a combination of atrial ischemia, inflammatory mediator release during mast cell activation, catecholamine surge, and metabolic derangements associated with cardiac arrest. Histamine and other vasoactive mediators released during anaphylaxis are well-recognized triggers of atrial arrhythmias through direct electrophysiological effects on atrial myocytes and increased adrenergic tone.^{12,13} Dogan et al reported that arrhythmic complications, including atrial fibrillation, occur in a significant proportion of Kounis syndrome cases and are associated with more complex clinical courses and higher in-hospital mortality, underscoring the severity of this presentation.¹⁴

Coronary angiography confirmed the diagnosis by demonstrating severe, reversible multivessel vasospasm involving the diagonal branch of the proximal LAD and the mid RCA, in the presence of only mild non-obstructive atherosclerosis.^{1,2,4} The spasm resolved completely following intracoronary nitroglycerin administration. Echocardiography showed global hypokinesis with moderately reduced systolic function, rather than regional wall-motion abnormalities, supporting a diffuse ischemic process. High-sensitivity troponin peaked at 23,000 ng/L, reflecting combined injury from coronary vasospasm, post-arrest myocardial stunning, and possible catecholamine-mediated toxicity.¹⁵ These findings are consistent with type I Kounis syndrome, in which vasospasm occurs in structurally normal or minimally diseased coronary arteries.^{1,2,4}

A major diagnostic challenge in this case is distinguishing allergic-mediated coronary vasospasm from catecholamine-induced vasoconstriction. Epinephrine, which is essential in the management of anaphylaxis and cardiac arrest, can induce coronary vasospasm through alpha-adrenergic effects, increase myocardial oxygen demand, and precipitate arrhythmias.^{16–18} In this patient, epinephrine was administered repeatedly during resuscitation and continued as an infusion during angiography, and coronary spasm was documented while the infusion was ongoing. The confounding effect of exogenous catecholamines cannot be excluded.

Nevertheless, several features support Kounis syndrome as the primary mechanism. Symptoms occurred immediately after bee stings, preceding epinephrine administration. Clinical features of hypersensitivity, including pruritus and localized tissue swelling, were present. Angiography demonstrated multivessel coronary vasospasm, which is more consistent with systemic mediator release than a focal catecholamine effect. The magnitude of myocardial injury was also disproportionate to arrest duration alone.^{3–5,11} A synergistic contribution of endogenous stress catecholamines and exogenous epinephrine nonetheless cannot be excluded,^{16–18} this reflects real-world complexity, where life-saving therapy may exacerbate coronary vasoconstriction.

The patient exhibited features of mixed cardiogenic and distributive shock. Left ventricular systolic dysfunction from ischemia and myocardial stunning contributed to the cardiogenic component, while systemic vasodilation from anaphylaxis produced a distributive component. The cardiac arrest most likely resulted from primary ischemic ventricular fibrillation triggered by severe coronary vasospasm. The rapid progression from sting exposure to collapse supports a primary arrhythmic mechanism rather than secondary deterioration.^{19,20}

These findings have important clinical implications. Kounis syndrome requires simultaneous management of the allergic and ischemic components.^{1,2,21} Treatment of anaphylaxis includes oxygen, antihistamines, corticosteroids, and carefully titrated epinephrine when indicated. Coronary vasospasm should be treated with nitrates and calcium-channel blockers.^{1,2,21} Beta-blockers and morphine should be avoided due to the risk of exacerbating vasospasm and histamine release, respectively.^{1,2,21} In resource-limited settings with high Hymenoptera exposure, clinicians should maintain a high index of suspicion. A temporal relationship between allergen exposure and cardiac symptoms, dynamic electrocardiographic changes, and early measurement of serum tryptase within 30–120 minutes of symptom onset can support diagnosis.^{10,22} Early recognition is critical, as delayed diagnosis may lead to extensive myocardial injury or death.^{1,2,14,21}

Despite compelling evidence, immediate symptom onset after bee stings, absence of obstructive coronary disease, and angiographic demonstration of reversible multivessel vasospasm, this case remains highly suggestive rather than definitively confirmed Kounis syndrome. Key limitations include: the absence of serum tryptase measurement (ideally obtained within 30–120 minutes of symptom onset), the lack of allergen-specific IgE testing, the confounding effect of high-dose exogenous epinephrine on coronary vasomotor tone, and the inherent interpretive challenge of a post-cardiac arrest ECG in which rhythm identification may be uncertain. The initial ECG interpretation, in particular, illustrates the diagnostic difficulty of distinguishing atrial fibrillation with ischemia-related QRS–ST fusion from ventricular tachycardia in critically ill patients. Nonetheless, this case contributes to the limited African literature on Kounis syndrome, highlights the diagnostic significance of the shark-fin ECG pattern combined with atrial fibrillation, and underscores the complexity of managing overlapping allergic and ischemic mechanisms in resource-limited settings.

Conclusion

Kounis syndrome should be considered in any patient presenting with acute coronary syndrome immediately following an allergic or hypersensitivity trigger, including Hymenoptera stings, even in the absence of prior allergy history. The simultaneous occurrence of shark-fin ST elevation and atrial fibrillation the distinguishing novelty of this case creates

a particularly complex diagnostic scenario that may be mistaken for ventricular tachycardia or a primary conduction abnormality. Careful ECG interpretation, with attention to rhythm regularity and serial evolution, is essential.

Management requires simultaneous attention to the allergic reaction and ischemic insult: antihistamines, corticosteroids, and cautious epinephrine for anaphylaxis, combined with nitrates and calcium-channel blockers for coronary spasm. Beta-blockers and morphine should be avoided. Early serum tryptase sampling and allergen-specific testing should be prioritized where available.

This report adds to the limited African literature on Kounis syndrome and emphasizes the need for heightened clinical awareness, prompt dual-pathway management, and robust post-event allergy evaluation, particularly in low-resource settings where Hymenoptera exposure is high.

Patient Perspective

The patient's family expressed gratitude for the rapid response by emergency medical services and the emergency department team. They noted that the patient had been gardening peacefully moments before the sudden collapse and were initially unaware that bee stings could trigger such a severe cardiac event. Following recovery, the patient reported no recollection of the event itself but expressed determination to avoid bee exposure and seek immediate medical attention for any future allergic reactions. The family emphasized the importance of raising awareness about Kounis syndrome in rural communities where bee stings are common.

Informed Consent and Ethics Statement

Written informed consent for publication of this case report and accompanying electrocardiographic and imaging figures was obtained from the patient in accordance with institutional policy at Mulago National Referral Hospital, Kampala, Uganda, and in compliance with the CARE guidelines. In accordance with the research ethics policy of Mulago National Referral Hospital, individual case reports are exempt from formal Research and Ethics Committee review; formal institutional approval for publication was therefore not required.

Acknowledgments

The authors thank the emergency medical services personnel, emergency department staff, cardiology team, and intensive care unit staff at the Department of Internal Medicine, Mulago National Referral Hospital, Kampala, Uganda, for their dedicated care of this patient. We also acknowledge the patient and his family for permitting publication of this case to advance medical knowledge.

Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

Funding

No funding was received for this case report.

Disclosure

The authors declare no competing interests in this work.

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