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Transient ST-Segment Elevation Following Snake Envenomation and Anti-snake Venom Administration: A Case Report of Suspected Kounis Syndrome

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Abstract

Kounis syndrome is an acute coronary syndrome associated with a hypersensitivity reaction, potentially involving mast-cell mediator release and coronary vasomotor dysfunction. Snake envenomation and anti-snake venom (ASV) administration have been reported as possible triggers in isolated cases.

We report a 55-year-old man with hypertension and type 2 diabetes mellitus who presented approximately three hours after a suspected viper bite to the right ankle with a prolonged whole blood clotting time (WBCT) (>20 minutes) and received 10 vials of ASV. Shortly after ASV administration, he developed chest pain, palpitations, and breathlessness with ST-segment elevation in leads I, augmented vector left (aVL), and V2-V3 and reciprocal ST depression in leads II, III, and augmented vector foot (aVF), together with elevated troponin-T and creatine kinase-MB (CK-MB). The ECG changes resolved within 10 minutes of treatment with adrenaline, hydrocortisone, and pheniramine. Echocardiography showed a hypokinetic infero-posterior wall with an ejection fraction of 45-50% and indeterminate chronicity, and coronary angiography performed after stabilization showed normal epicardial coronary arteries. Kounis syndrome, provisionally type I, is one possible explanation, alongside direct venom-induced myocardial injury and other causes of myocardial injury; the diagnosis remains suspect because venom and ASV could not be distinguished as triggers and alternative causes cannot be fully excluded. Clinicians should consider Kounis syndrome in snake-bite patients with chest pain during or after ASV administration, while recognizing that adrenaline remains first-line treatment for anaphylaxis and should be given with cardiac monitoring.

Categories: Cardiology, Internal Medicine, Allergy/Immunology
Keywords: allergic myocardial infarction, anti-snake venom, coronary vasospasm, kounis syndrome, snake envenomation, st-segment elevation

Introduction

Kounis syndrome is an acute coronary syndrome associated with a hypersensitivity reaction, potentially involving mast-cell mediator release and coronary vasomotor dysfunction. It was first described by Kounis and Zavras in 1991 as “allergic angina” [1]. Three variants are recognized: type I, vasospastic allergic angina without underlying coronary atherosclerosis; type II, allergic myocardial infarction in a patient with pre-existing but previously silent atheromatous disease, in whom the hypersensitivity reaction precipitates plaque erosion or rupture; and type III, allergen-associated stent thrombosis [2]. The proposed mechanism involves the release of inflammatory mediators, including histamine, leukotrienes, platelet-activating factor, and various cytokines, from activated mast cells, producing coronary vasospasm, increased vascular permeability, and, in susceptible individuals, plaque destabilization [2,3].

Cardiovascular involvement after snakebite is uncommon but includes myocardial infarction, ventricular dysfunction, hypotension, cardiac arrest, and myocarditis [4]. Direct venom-induced myocardial injury is, therefore, an important competing explanation for any cardiac event after envenomation. Hypersensitivity-associated coronary vasospasm (Kounis syndrome) is a distinct mechanism that has been reported after snakebite and, less commonly, after anti-snake venom (ASV) administration, but only in isolated case reports [5-7]; the evidence for these associations is therefore limited. Because the presenting features overlap substantially with a primary acute coronary syndrome and with direct venom toxicity, distinguishing these mechanisms is clinically important. Management is further complicated because adrenaline, the first-line treatment for anaphylaxis, may theoretically aggravate coronary vasospasm, and beta-blockers and morphine have also been cautioned against in suspected Kounis syndrome [2,8]. Adrenaline should, nevertheless, still be given for genuine anaphylaxis, with cardiac monitoring. We describe a patient with hypertension and type 2 diabetes mellitus, without known coronary artery disease, who developed transient ST-segment elevation temporally associated with ASV administration following a suspected viper bite, and we discuss why Kounis syndrome is a possible but unproven explanation.

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Case Presentation

A 55-year-old man, a chronic alcohol user and tobacco chewer with known hypertension and type 2 diabetes mellitus and no known ischemic heart disease, was brought to the casualty department by his son after a snake bite.

History

The patient had been asymptomatic until 6:00 AM, when he was bitten on the right lower limb near the ankle at a farmhouse. The bite was painless, and no fang marks were initially visible. He was first taken to a government hospital and subsequently referred to our casualty department, arriving approximately three hours after the bite. The snake was not captured or definitively identified but was suspected to be a viper. No ASV or other treatment had been administered before arrival.

There was no local swelling or bleeding at the bite site; no numbness, tingling, ptosis, or blurring of vision; no dysarthria, dysphagia, or breathlessness at presentation; and no vomiting, abdominal pain, hematuria, or other bleeding manifestations. He was a known hypertensive and type 2 diabetic on regular treatment, with no history of ischemic heart disease, coronary intervention, pulmonary tuberculosis, or bronchial asthma. He reported chronic daily alcohol use for 30 years (last consumed three days before admission) and chronic tobacco chewing for 15 years. The family history was not contributory.

Examination

On admission, the patient was conscious, oriented, and afebrile. Pulse was 66 beats per minute and regular, blood pressure 150/90 mmHg, respiratory rate 18 per minute, and SpO_2 97% on room air. Pallor, icterus, raised jugular venous pressure, clubbing, cyanosis, lymphadenopathy, and pedal edema were absent. The single breath count was 28. Whole blood clotting time (WBCT) was prolonged at more than 20 minutes. WBCT was performed as the bedside 20-minute whole blood clotting test (2 mL of venous blood in a clean, dry glass tube, left undisturbed for 20 minutes; failure to clot was taken as abnormal). Cardiovascular examination revealed normal first and second heart sounds without murmurs. Respiratory, abdominal, and neurological examinations, including cranial nerves, motor power (5/5 in all four limbs), sensory modalities, deep tendon reflexes (2+ and symmetrical), and plantar responses (flexor bilaterally), were unremarkable.

Observations during the symptomatic episode

During the episode, the pulse was 110 beats per minute, blood pressure was 98/74 mmHg, respiratory rate was 27 per minute, and SpO_2 was 92%. Features of hypersensitivity such as flushing, wheezing and hypotension were present.

Investigations

Serial WBCT remained prolonged (>20 minutes) on three consecutive occasions, meeting criteria for ASV administration. Hematological and biochemical parameters at presentation (day 0) and on day 3 are shown in Table *1*. The peripheral smear was normocytic and normochromic, with white blood cell counts within normal limits and adequate platelets; malarial parasites were not seen. Liver function tests at presentation showed total bilirubin 0.80 mg/dL (direct 0.10, indirect 0.70), aspartate aminotransferase (SGOT) 106 U/L, alanine aminotransferase (SGPT) 116 U/L, alkaline phosphatase 82 U/L, albumin 3.8 g/dL, and globulin 3.3 g/dL. Cardiac markers are shown in Table *2* and metabolic and coagulation parameters in Table *3*. Urine microscopy showed bacteria (3), white blood cells 6/high-power field (hpf), and red blood cells 5/hpf, with blood >200 and negative bilirubin, protein, ketones, and nitrites. HIV-1 and HIV-2, hepatitis B surface antigen, and hepatitis C virus serology were negative. Blood group was B Rh-positive. Hematological and biochemical parameters at presentation and on Day 3 have been presented in Table *1* below; cardiac markers with reference ranges have been presented in Table *2*; and metabolic and coagulation parameters have been presented in Table *3*.

Parameter	Day 0 (presentation)	Day 3	Reference range
Hemoglobin (g/dL)	14.8	12.9	13.0-17.0
Total leucocyte count (/µL)	5,000	6,000	4,000-11,000
Hematocrit (%)	48.2	43.1	40-50
Platelets (/µL)	189,000	21,200	150,000-450,000
Mean corpuscular volume (fL)	91	65.1	80-100
Urea (mg/dL)	15	23	15-40
Creatinine (mg/dL)	0.62	0.96	0.6-1.2
Sodium (mmol/L)	136	137	135-145
Potassium (mmol/L)	3.9	3.9	3.5-5.0

TABLE 1: Hematological and biochemical parameters at presentation and on Day 3

Parameter	Patient value	Reference range
Troponin-T (ng/ml)	47.97	Assay-dependent; e.g., <14 ng/L (high-sensitivity cTnT, 99th percentile)
CK-MB (ng/mL)	7.14	<5 ng/mL (laboratory-specific)

TABLE 2: Cardiac markers with reference ranges

CK-MB: Creatine kinase-MB; cTnT: Cardiac troponin T

Parameter	Patient value	Reference range
HbA1c (%)	6.8	<5.7 (non-diabetic); <7.0 (usual treatment target in diabetes)
Total cholesterol (mg/dL)	203	<200 (desirable)
Triglycerides (mg/dL)	141	<150
HDL cholesterol (mg/dL)	43	>40 (men)
LDL cholesterol (mg/dL)	131	<100 (optimal); 100-129 (near optimal)
VLDL cholesterol (mg/dL)	28.2	Feb-30
TSH (µIU/mL)	1.09	0.4-4.0
PT (s)/INR	13.7 / 1.05	PT 11-13.5 s; INR 0.8-1.2
aPTT (s)	39.4	25-35

TABLE 3: Metabolic and coagulation parameters

HbA1c: Glycated hemoglobin; HDL: High-density lipoprotein; LDL: Low-density lipoprotein; VLDL: Very-low-density lipoprotein; TSH: Thyroid-stimulating hormone; PT: Prothrombin time; INR: International normalized ratio; aPTT: Activated partial thromboplastin time

Electrocardiography

The admission electrocardiogram (ECG), recorded before ASV administration, showed sinus rhythm with T-wave inversion in V1 and augmented vector left (aVL) and poor R-wave progression, without significant ST-T changes. During the symptomatic episode, which began around 20 minutes after ASV administration, an ECG (Figure *1*) demonstrated ST-segment elevation in leads I, aVL, and V2-V3 with reciprocal ST depression

in leads II, III, and augmented vector foot (aVF). The changes reverted within 10 minutes of treatment; this temporal association does not by itself establish the mechanism.

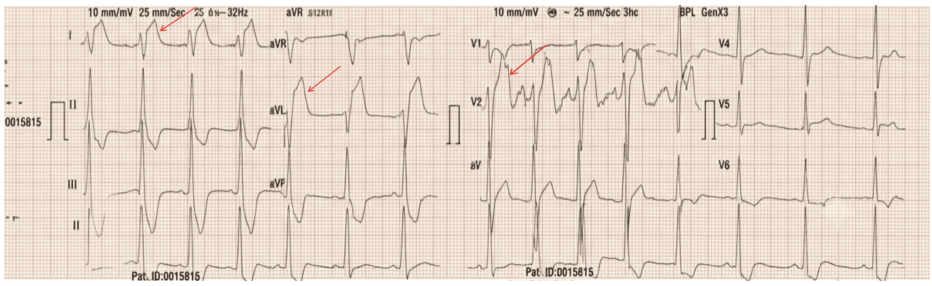


FIGURE 1: ECG recorded during the symptomatic episode after ASV administration, showing ST-segment elevation in leads I, aVL, and V2–V3 with reciprocal ST depression in leads II, III, and aVF. Red arrows indicate the ST-segment elevation in leads I, aVL, and V2.

ECG: Electrocardiogram; ASV: Anti-snake venom; aVL: Augmented vector left; aVF: Augmented vector foot

Echocardiography and coronary angiography

Two-dimensional echocardiography showed normal chamber dimensions and normal cardiac valves, with a hypokinetic infero-posterior wall and an ejection fraction of 45-50%. Whether this wall motion abnormality was acute or chronic could not be established, because no previous echocardiogram was available for comparison and serial echocardiography was not performed. The impression was hypertensive heart disease. The original two-dimensional echocardiographic images could not be retrieved for inclusion in this report. In view of the ST-segment elevation and troponin rise, coronary angiography was performed after hemodynamic and coagulation stabilization. It showed normal epicardial coronary arteries without any obstructive atherosclerotic lesion (Figures *2A*, *2B*); no intracoronary provocation testing or intravascular imaging was performed, so transient vasospasm or minor plaque disruption cannot be excluded.

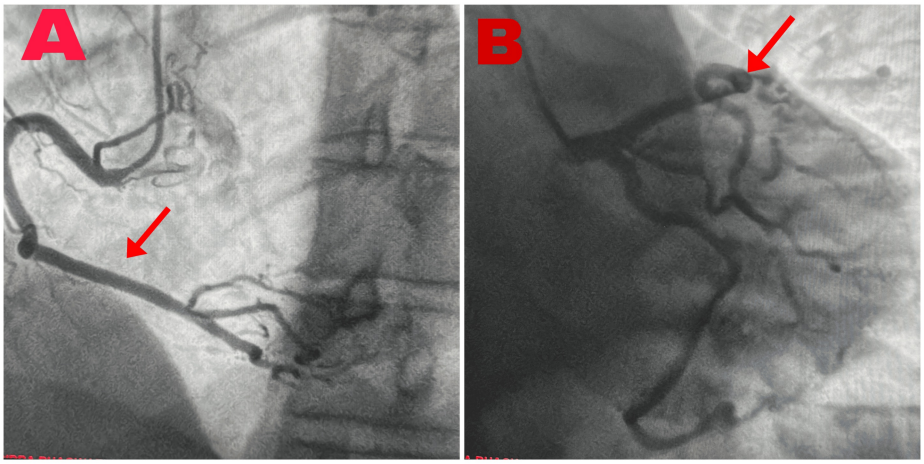


FIGURE 2: Coronary angiographic images demonstrating patent right coronary and left circumflex arteries without significant obstructive lesions

(A) RCA angiogram showing a patent RCA without significant obstructive lesions (red arrow); (B) LCx coronary angiogram demonstrating a patent LCx artery without significant obstructive lesions (red arrow). Overall, the angiographic findings demonstrate the absence of significant obstructive epicardial coronary artery disease.

RCA: Right coronary artery; LCx: Left circumflex

Differential diagnosis

The differential diagnosis for transient ST-segment elevation with a troponin rise in this setting included: (1)

primary atherosclerotic acute coronary syndrome, rendered unlikely by normal coronary angiography; (2) direct venom-induced myocardial injury, described with several snake species and capable of producing ECG changes and biomarker elevation independent of an allergic mechanism , which cannot be excluded here and may coexist with a hypersensitivity mechanism; (3) Takotsubo (stress) cardiomyopathy, which can be triggered by acute physical or emotional stress including envenomation but typically shows apical ballooning on echocardiography rather than the infero-posterior hypokinesia seen here , although serial imaging was not available to exclude it; (4) myocarditis, considered less likely given the rapid, complete reversal of ECG changes within minutes , though cardiac MRI was not performed; and (5) Kounis syndrome, considered a possible explanation on the basis of the temporal relationship to ASV administration, the rapid resolution after anti-allergic therapy, and the absence of obstructive coronary disease, but documented systemic features of hypersensitivity or mast-cell markers as mentioned previously.

Treatment

The patient received ASV (10 vials diluted in normal saline, administered intravenously over one hour) on account of a serial WBCT exceeding 20 minutes on three occasions. During the symptomatic episode with ST-segment elevation, he was treated with intramuscular adrenaline (0.5 mg, administered into the right anterolateral thigh), intravenous hydrocortisone (200 mg), and slow intravenous pheniramine (30 mg), with resolution of ECG changes within ten minutes. ASV infusion was stopped during the episode. Aspirin was initially withheld in view of the persistently deranged WBCT. On cardiology consultation, a combination tablet of aspirin 75 mg and atorvastatin was advised once he was stable, and coronary angiography was arranged after stabilization. Supportive treatment included ceftriaxone, a multivitamin infusion, a proton-pump inhibitor, rosuvastatin, metoprolol, telmisartan, and metformin, reflecting his background hypertension and diabetes mellitus (glycated hemoglobin (HbA1c) 6.8%).

Outcome and follow-up

The patient improved symptomatically over the course of admission. At the time of assessment for discharge, he was afebrile with a blood pressure of 140/90 mmHg, pulse of 60 beats per minute, respiratory rate of 22 per minute, and SpO_2 of 98% on room air, with all peripheral pulses palpable and no pallor, icterus, pedal oedema, raised jugular venous pressure, cyanosis, clubbing, or lymphadenopathy. Further evaluation of the incidental bladder lesion (CT urography and cystoscopy) was not completed during this admission: the patient and relatives were counseled regarding the need for a longer hospital stay for this purpose but declined, and the patient was discharged against medical advice with advice for urological follow-up, along with ongoing cardiac medications (rosuvastatin, metoprolol, and telmisartan) and metformin.

Discussion

This case describes transient, reversible ST-segment elevation with a troponin rise that began shortly after ASV administration in a patient with hypertension and type 2 diabetes mellitus but no known coronary artery disease. Kounis syndrome is one possible explanation among several, and the diagnosis remains suspected rather than proven. The findings that support it are the temporal relationship to ASV administration, the reciprocal ST changes, the troponin rise with angiographically normal epicardial coronary arteries, and the rapid reversal of the ECG changes. Essential evidence is missing, however: systemic features of hypersensitivity were documented, serum tryptase or other mast-cell markers were not measured, and troponin was not measured serially. Rapid resolution after treatment is compatible with, but does not prove, a hypersensitivity mechanism, because spontaneous resolution of vasospasm cannot be excluded. Type I Kounis syndrome, in which hypersensitivity produces coronary vasospasm without underlying atherosclerotic coronary artery disease, as distinct from type II, in which plaque erosion or rupture is precipitated in pre-existing coronary disease [2,9], is therefore a provisional clinical interpretation in this patient.

Both snake venom and ASV (an equine- or ovine-derived antiserum) are recognized, if uncommon, triggers of Kounis syndrome [5-7], and distinguishing between the two as the causative allergen is often not possible on clinical grounds alone, as reflected in the treating team's documentation of both ASV and snake venom as possible triggers in this patient. Reported precedents include Kounis syndrome with cardiogenic shock after a cobra bite [6]; Kounis syndrome with ST-segment elevation and normal troponin after a hump-nosed pit viper bite, managed supportively without antivenom [5]; and Kounis syndrome after ASV administration in a patient with a neurotoxic snake bite [7]. Together, these isolated reports suggest that the association, although rare, spans elapid and viper bites and can occur with envenomation alone or after antivenom therapy. They do not establish causality, and direct venom-induced myocardial injury [4] and Takotsubo syndrome remain alternative explanations that cannot be excluded in the present case.

The elevated troponin-T and CK-MB in this patient, despite normal coronary arteries, are compatible with transient myocardial injury from coronary vasospasm rather than a fixed atherosclerotic culprit lesion, although they are also seen with direct venom toxicity. Because the diagnosis rests on the combination of clinical features, laboratory tests, ECG, echocardiography, and angiography [10], confirming normal epicardial coronary anatomy was an important step: it excluded an atherothrombotic process and supported a primarily allergic, vasospastic mechanism without proving it. The infero-posterior hypokinesia does not

correspond to the territory of the ST-segment elevation (leads I, aVL, and V2-V3), and the admission ECG already showed T-wave inversion in V1 and aVL with poor R-wave progression; these findings may reflect pre-existing hypertensive myocardial disease and are a further reason for caution. The prognosis of type I Kounis syndrome is reported to be generally favorable once the triggering allergen is withdrawn and the reaction is treated [9,11].

From a management perspective, this case illustrates the published cautions regarding beta-blockers and morphine-class analgesics in suspected Kounis syndrome, as each may theoretically potentiate mast-cell degranulation or unopposed vasoconstriction [2,8,11]. Adrenaline remains first-line treatment for anaphylaxis even when cardiac involvement is suspected and should be given with continuous cardiac monitoring; because adrenaline can itself provoke coronary vasospasm, the ECG should be interpreted with the timing of its administration in mind. Antiplatelet therapy was appropriately deferred in this patient because of the coagulopathy associated with the snake bite itself, illustrating the additional complexity of managing Kounis syndrome when it coexists with venom-induced consumptive coagulopathy.

Limitation

This is a single case, and the association between ASV or venom and the cardiac event is based on temporal association alone. The snake was not identified; ASV and venom could not be separated as triggers; features of systemic hypersensitivity and mast-cell markers were not documented; troponin was measured once; the chronicity of the infero-posterior hypokinesia could not be determined and the echocardiographic images were unavailable; and angiography was performed after the episode without provocation testing. These limitations preclude any causal inference.

Conclusions

This case describes transient ST-segment elevation with a troponin rise and normal epicardial coronary arteries after a suspected viper bite and ASV administration, for which Kounis syndrome is a possible but unproven explanation. Clinicians should consider it, alongside direct venom-induced myocardial injury and other causes, in snake-bite patients who develop chest pain or ECG changes during or after ASV administration, and should document allergic features, timing of drugs, and serial ECGs and biomarkers carefully. Adrenaline should still be given for genuine anaphylaxis, with close cardiac monitoring, and beta-blockers and morphine should be used with caution. A causal relationship between ASV and the cardiac event cannot be established from a single case.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

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