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Case Report

Transient ST Elevation following Anaphylactic Shock: A Case Report of The Potential Kounis Syndrome.

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Abstract

Background: Anaphylactic shock rarely can induce allergic-induced acute coronary syndrome known as Kounis Syndrome. It involves the release of inflammatory cytokines through mast cell activation, which leads to coronary artery vasospasm and ST elevation presentation on electrocardiography (ECG).

Case Illustration: A 45-year-old woman with no known past medical history presented with weakness all over her body, dizziness, pain in her left hand, and a history of fainting immediately after being stung by small wasps. She was in hypotension with wheezing and weak peripheral pulses. Her laboratory examination showed leukocytosis, thrombocytosis, and high levels of blood sugar and triglycerides. Initial twelve-lead ECG demonstrated ST-segment elevation on the inferior leads (II, III, and aVF) and reciprocal ST-depression on the lateral lead. Diagnosis of anaphylactic shock caused by an insect bite was made, with a potential of becoming Kounis Syndrome. Treatment for anaphylactic shock was initiated with fluid resuscitation, intramuscular epinephrine, intravenous methylprednisolone, and ranitidine. A repeat ECG one hour post-intervention showed resolution of ST-segment elevations. The patient was discharged in stable condition two days later.

Discussion: Kounis Syndrome consists of three main types, including Type I Kounis Syndrome. Its treatment is mostly in the form of aborting the anaphylactic reaction until the symptoms resolved. Based on this case, the patient was a young non-smoker Asian woman with a low risk (<1% of 10-year risk) of fatal cardiovascular disease (CVD) in populations with high CVD risk. Clinically, the patient did not show any vascular thrombotic symptoms. In addition, administration of adrenaline, corticosteroid, and antihistamine relieved the patient's symptoms, thus, this case can be hypothesized as a potential Type I Kounis Syndrome. Emergency coronary angiography or echocardiography has to be done to clarify the diagnosis of this allergic-induced acute coronary syndrome.

Conclusion: Transient ST elevation could happen in some rare cases following an anaphylactic shock. The swift recognition, accurate diagnosis, and prompt treatment are important for optimal outcomes in the probability of Kounis Syndrome.

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Keywords: *anaphylactic shock, Kounis syndrome, ST elevation*.

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Introduction

Acute coronary syndrome following mast cell activation due to hypersensitivity, allergic, or anaphylactoid reactions was first described by Kounis and Zavras in 1991 and has been named "Kounis Syndrome" (KS) after its discoverer. The release of inflammatory cytokines through mast cell activation, which leads to coronary artery vasospasm and/or atheromatous plaque rupture, was the mechanism of KS.[1] Various presentations of this allergic angina have been documented following a variety of external exposures.[2] In this case report, we present transient allergic-induced ST-elevation in the patient without underlying coronary artery disease.

Case Presentation

A 45-year-old woman with no known past medical history presented with weakness all over her body, dizziness, pain in her left hand, and a history of fainting immediately after being stung by four small wasps 30 minutes prior to hospital admission. She also felt minimal discomfort on her chest a day before. Her initial vital signs were: blood pressure of 80/50 mmHg, pulse of 130 beats per minute, respiration rate of 28 breaths per minute, temperature of 36.7^{O}C, and oxygen saturation of 95% on room air. The examination revealed wheezing and weak peripheral pulses. Her laboratory examination showed leukocytosis (15.6µ10^{3}/µL), thrombocytosis (416µ10^{3}/µL), a slightly high level of random blood sugar (204 mg/dL), increased level of serum transaminase, and mild hypokalemia (3.3 mmol/L). The initial twelve-lead ECG demonstrated ST-segment elevation in the inferior leads (II, III, and aVF) and reciprocal ST-depression in the lateral leads (Figure 1). A diagnosis of anaphylactic shock caused by an insect bite was made. However, the ST elevation could not be concluded as acute ST elevation myocardial infarction because typical chest pain was absent, and initial myocardial enzyme levels (Troponin I) remained within the normal range. Treatment for anaphylactic shock was initiated with fluid resuscitation (1000 mL normal saline), 0.5 cc intramuscular epinephrine, 1 cc intravenous methylprednisolone (125 mg/mL), and 2 cc intravenous ranitidine (25 mg/mL), resulting in an improvement in the patient's symptoms. The patient's blood pressure also improved to 110/70 mmHg. A repeat ECG showed resolution of ST segment elevations an hour later (Figure 2). The patient was then transferred to the general ward. Vital sign was good and serial ECG did not show any abnormal changes during treatment. She experienced mild weakness and reduced pain in his left hand on the first day of her hospital stay. The redness and swelling at the site of the sting also subsided. The patient discharged in stable condition two days later. Unfortunately, no post-discharge follow-up data available for this patient.

Discussion

Kounis Syndrome is a rare type of acute coronary syndrome because of coronary artery spasm with or without the erosion or rupture of atherosclerotic plaque due to inflammatory factors released during an allergic reaction.[1,3] The exact mechanism is still unclear, however, the release of inflammatory mediators including histamine, platelet-activating factor, leukotrienes, neutral protease, cytokines, and prostaglandins, after the direct activation of mast cells, is believed to be the main inducer of this allergic-related coronary vasospasm.[4] Certain foods, drugs (commonly non-steroidal anti-inflammatory drugs and antibiotics), contrast media, and environmental exposure may provoke Kounis Syndrome.[5] More than half of the affected patients are 40-70 years old, although this syndrome can happen at any age. The patient in our case was 45 years old, belonging to the most commonly affected age group.

Both allergic reaction symptoms (including erythematous rash, hives, wheezing, and even angioedema) and acute myocardial ischemia symptoms (including chest pain, dyspnea, and palpitations) occurring concurrently are the main clinical characteristics of Kounis Syndrome. The diagnosis of this disease is primarily based on clinical manifestations. Laboratory tests (eosinophils, immunoglobulin E, and cardiac enzymes), ECG, echocardiography, and coronary angiography can be performed to clarify the diagnosis. An essential part of the diagnosis is still a careful review of the patient's clinical history, including allergy and medication history. The presence of elevated Immunoglobulin E level in the diagnosis of Kounis Syndrome remains unclear, and a normal level of it cannot exclude the possibility of the diagnosis.[6] The

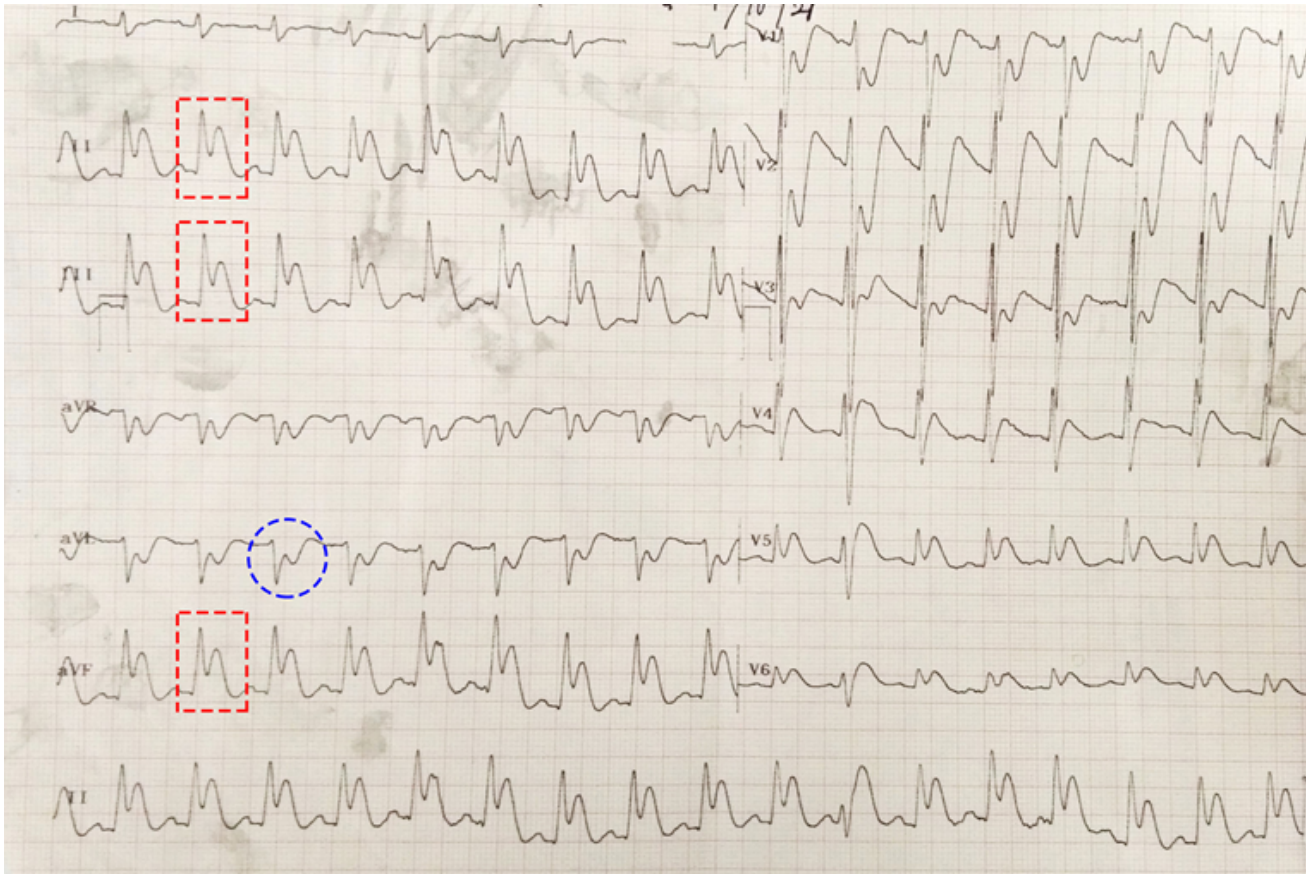

Figure 1. Initial electrocardiogram demonstrated ST-elevation in the inferior leads (red-dotted) and reciprocal ST-depression on the lateral leads (blue-dotted).

rise of cardiac enzymes in the disease implies that the allergic reaction decreases myocardial perfusion and eventually causes myocardial injury. The ECG features are commonly indistinguishable from those presented among patients with acute myocardial infarction or coronary heart disease.[7] Those ECG may vary from ST-T segment elevation or depression as the most common findings, to conduction defects, and even arrhythmias.[3] Therefore, ECG result alone could not be used to establish the diagnosis of KS. Echocardiography may indicate abnormal heart wall motion in the area supplied by the affected coronary artery. Further diagnostic tools such as coronary angiography or cardiac MRI are needed to definitively diagnose Kounis Syndrome and rule out other potential causes of ST-segment elevation. Both examinations may show the occurrence of coronary vasospasm or stenosis legibly.[8]

Kounis Syndrome consists of three main types according to clinical manifestation and coronary angiography result.[3] The most common type — Type I Kounis Syndrome — manifests as coronary artery vasospasm with or without cardiac biomarker elevation among patients without predisposing factors of coronary artery disease. This type differs from the second and third type, which present plaque erosion or thrombosis leading to myocardial infarction on patients with a history of cardiovascular disease.[3,8]

The treatment of Kounis Syndrome is still challenging and lacks consensus. The main goals are the treatment of anaphylaxis and myocardial revascularization. Removal of the allergens is the first thing to do, followed by antihistamines and corticosteroids administration to abort the allergic reaction.[9] Patients with anaphylactic shock also need appropriate fluid resuscitation. Vasodilators, including calcium channel blockers and nitrates, can help resolve the allergic vasospasm.

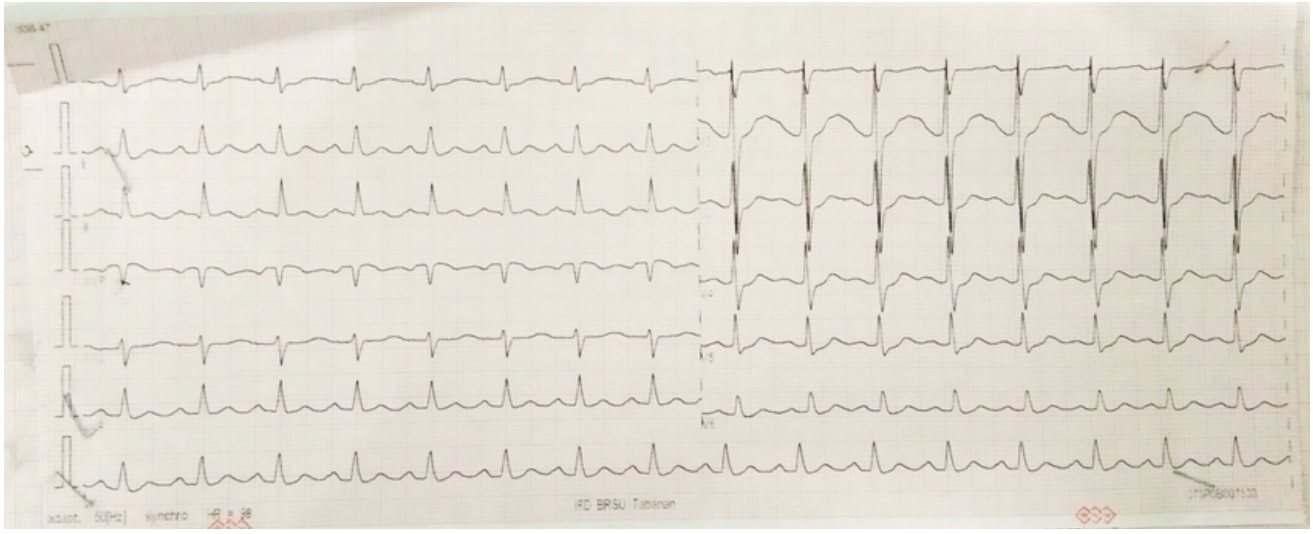

Figure 2. Repeated electrocardiogram showed resolution of inferior leads' ST-elevation.

However, the treatment of KS requires careful use of several common drugs. Epinephrine, which is used routinely in treating anaphylaxis, should be used with careful monitoring, as it may potentially worsen coronary vasospasm in KS.[10] The use of morphine, one important drug for treating acute chest pain, can be disadvantageous due to its histamine-releasing feature, which can exacerbate symptoms. Beta-blockers used in an acute exacerbation of KS may also induce coronary vasospasm due to unopposed alpha-adrenergic action.[10,11]

The patient in this case was a young Asian woman who was a non-smoker and had no history of dyslipidemia, hypertension, or type 2 diabetes mellitus. This was similar with the characteristics of patient from two Chinese reports.[8,12] Therefore, based on the Systematic Coronary Risk Estimation (SCORE) Chart, she was categorized as low risk (<1% of 10-year risk) for fatal cardiovascular disease (CVD) in populations with high CVD risk. Clinically, the patient did not show any vascular thrombotic symptoms. Some anaphylactic-related symptoms were the dominant finding, including generalized pruritic, rash, and breathlessness with/without chest discomfort.[12–14] In addition, the administration of adrenaline, corticosteroid and antihistamine relieved the patient's symptoms and normalized the ST elevation on the ECG. Some studies have shown that coronary vasospasm and allergic symptoms presented in the first type of Kounis Syndrome can be easily reversed through the administration of coronary vasodilators, steroids, and anti-histamine drugs, leading to a good prognosis with appropriate treatment.[12,15] However, due to this syndrome vagueness, some clinicians also treat it with the ACS (acute coronary syndrome) treatment algorithm, although no difference was found on the patient's outcome.[7,8,14] The patient and her family rejected any further diagnostic examinations, like echocardiography or coronary angiography, due to financial reasons. Thus, this case can be hypothesized as a potential Type I Kounis Syndrome. The comparison between previous Kounis Syndrome reports can be seen in Table 2.

Our patient also experienced hypokalemia, which is known can present various ECG abnormalities. Although this hypokalemia-related ECG changes mostly in the form of ST-segment depression, T-wave amplitude decrement, prominent U waves, and a ratio of U-wave to T wave >1.[16,17] Hypokalemia raises resting potential and subsequently accelerates depolarization, which raises the automaticity and excitability.[17] However, those phenomena mostly occur in severely low potassium state—i.e. 1.6 (3.5 - 5.1 mmol/L) in a case reported by Sethuraman et al.[17] and 1.8 mmol/L in a case reported by Petrov et al.16 It also gets trickier, because the improvement in ECG can be seen post potassium correction even before the serum potassium achieves normal level. The patient in one report had a complete resolution of ECG at a very low potassium level of 2.3 mmol/L (3.5 - 5.1 mmol/L).[16]

Table 1. Summary of the patient's clinical course.

	Hospital admission	1st Day	2nd Day (Discharged)
Specific symptom(s)	Weakness all over her body, dizziness, pain on her left hand	Slight weakness, pain on left hand was decreased	All symptoms were completely resolved
Related physical examination result(s)	Wheezing, weak peripheral pulses, redness, and swelling on the left hand	Redness and swelling on the sting site were slightly disappeared	No abnormalities found
Vital sign	Hypotension (BP: 80/50 mmHg) was found along with increased respiratory rate (28 breaths/minute)	The patient was fully awake, all vital signs were within normal limit	Vital signs were within normal limit
Laboratory examination result(s)	Laboratory examination result(s) Complete blood count RBC: 4.94 x 106/µL (4.60-6.20) HB: 14.1 g/dL (13.5-18.0) HCT: 44.0% (40-54) WBC: 15.64 x 103/µL (4.50-11.0) PLT: 416 x 103/µL (150-440) Blood chemistry test RBS: 204 g/dL (70-140) ALT: 32 U/L (11.0-34.0) AST: 42 U/L (5-34) BUN: 7.2 mg/dL (8.00-23.00) SC: 0.8 mg/dL (0.57-1.11) Na+: 139 mmol/L (136-145) K+: 3.3 mmol/L (3.50-5.10) Cl-: 105 mmol/L (94-110) Cardiac enzyme Troponin I: <0.10	Blood chemistry test RBS: 111 g/dL (70-140) Total cholesterol: 200 mg/dL (0-200) HDL: 40 mg/dL (40-60) LDL: 121 mg/dL (<100) TG: 194 mg/dL (<150)	N/A

BP = blood pressure; RBC = red blood cell; HB = hemoglobin; HCT = hematocrit; WBC = white blood cell; PLT = platelet; RBS = random blood sugar; ALT = alanine transaminase; AST = aspartate transaminase; BUN = blood urea nitrogen; SC = serum creatinine; HDL = high-density lipoprotein; LDL = low-density lipoprotein; TG = triglyceride; N/A = not available

Conclusion

Transient ST elevation can occur in some rare cases following an anaphylactic shock, which can result in Kounis Syndrome. Therefore, it is important to have a serial ECG examination in patients with both systemic anaphylaxis and angina-equivalent symptoms at the same time. It is also essential to have a specific consensus regarding a personalized approach to treatment, since medications for allergy could potentially exacerbate myocardial ischemia, and vice versa.

List of Abbreviations

ACS	Acute coronary syndrome
CVD	Cardiovascular disease
ECG	Electrocardiography
KS	Kounier syndrome
SCORE	Systematic coronary risk estimation

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Table 2. Comparison between previous Kounis Syndrome case reports and this study.

	This study	Basnet et al. 2023[13]	Ebrahimi et al. 2024[14]	Liao et al. 2024[12]	Pradhan et al. 2018[7]	Wu et al. 2020[8]
Patient's characteristic	Female, 45-year-old, Indonesian	Male, 48-year-old, Nepali	Male, 51-year-old, Iranian	Female, 47-year-old, Chinese	Male, 22-year-old, German	Female, 48-year-old, Chinese
History	Got stung by four small wasps.	Got stung by around 40–50 wasps.	Got diclofenac intramuscular injection 30 minutes before. History of penicillin allergy.	Got bitten by bug (Cryptopteran) two days before. No history of medical issues.	Administered with a single dosage of amoxicillin.	Intramuscularly injected with 10 mg anisodamine.
Main symptoms	Weakness all over her body, dizziness, pain in her left hand, and a history of fainting.	Pain all over his body, light-headedness,	Generalized pruritic, rash, breathing difficulty, central heaviness in chest.	Itchy and pain on skin, pressuring sensation in the chest, and a slight shortness of breath.	Acute retrosternal pain and chest tightness.	Chest pain with sweating 10 minutes after injection of anisodamine.
Significant physical examination result	Wheezing and weak peripheral pulses.	GCS 3 of 15, pulse was very feeble, blood pressure could not be recorded, and SpO2 was unrecordable.	BP of 160/95 mmHg, HR of 130 beats per minute, RR of 25 breaths per min, and SpO2 94% on room air.	BP of 144/88 mmHg. Patchy brown-red rashes on the neck.	Tender throat and white patches over the tonsils.	Low BP (82/50 mmHg), erythematous pruritic rash on the chest, abdomen and limbs.
Significant additional examination result	ST-segment elevation in the inferior leads and reciprocal ST-depression in the lateral leads. Increased level of serum transaminase (ALT 32 U/L, AST 42 U/L), a mild hypokalemia (3.3 mmol/L), and Troponin I <0.10	ST-elevation in the inferior leads, while ST depressions in leads I, aVL, V1, V2, and V3. AST level of 452 U/L and ALT level of 595 U/L	ST depression in leads I, II, III, AVL, AVF, and V5-V6. ST-segment elevation in the AVR lead. LVEF was 65% and no abnormality heart's motion. Troponin I was negative.	ST-segment elevation in lead V1–V4. Hs-troponin 2.54 ng/ml, Hs-CRP 46.7 mg/L, and procalcitonin of 0.11 ng/ml. LVEF 67%. Emergency coronary angiography showed no stenosis, LCX and RCA patency, without any plaque grade stenosis.	ST elevations in II, III, aVF, V5, and V6. Positive troponin (300 pg/dL), elevated creatinine kinase (315 IU/L). Normal functioning heart with preserved LVEF during echocardiography.	ST-segment elevation in the inferior leads with ST-segment depression in the lateral leads. Coronary angiography showed no significant abnormalities. Immunoglobulin E level was 365 IU/mL, while the peak troponin I level was 0.247 ng/mL.

Treatment	Fluid resuscitation of (1000 mL) normal saline), 0.5 cc intramuscular epinephrine, 1 cc intravenous methylprednisolone (125 mg/mL), and 2 cc of intravenous ranitidine (25 mg/mL).	Initial: CPR with adrenaline, hydrocortisone (200 mg IV), pheniramine maleate (45.5 mg IV), enoxaparin (60 mg SC), clopidogrel and aspirin loading-dose orally. Later: ventilator support, sodium bicarbonate, inotropes.	Aspirin (325 mg orally), clopidogrel (300 mg orally), atorvastatin (80 mg orally), nitroglycerin (10 mcg per minute IV), an unfractionated heparin (UFH) infusion (60 U/kg/h), and metoprolol (50 mg orally)	Intravenous drip of calcium gluconate injection, topical halometasone ointment, and oral loratadine.	Heparin bolus, single antiplatelet therapy of acetylsalicylic acid (100 mg).	Initial: Saline solution, promethazine (25 mg) and dexamethasone (10 mg). Later: 300 mg aspirin and 180 mg ticagrelor for emergent coronary angiography.
Resolution	A repeat ECG showed resolution of ST segment elevations an hour later. Vital sign was good and serial ECG did not show any abnormal changes during treatment. The patient discharged in stable condition two days later.	His condition gradually deteriorated and died on the same day	The patient's ECG gradually returned to normal 12 hours later. Serial troponin levels were normal. Symptoms were completely resolved the day after. Good clinical outcome was confirmed at a follow-up visit 6 months later.	The patient's symptoms improved, ECG and troponin re-examination were normal. No symptoms during the one-week-after-discharge follow-up.	The patient was discharged in a stable condition after 5 days. A telephonic follow-up after a week revealed good recovery of the patient.	ST-segment elevation had disappeared 49 minutes after the first ECG. The patient was discharged without complications. During the 9-month follow-up, the patient avoided anisodamine injection and remained free of chest pain.

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