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Intravenous Contrast-Induced Kounis Syndrome Presenting as Unstable Monomorphic Ventricular Tachycardia: A Rare Case Report

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KEYWORDS

Kounis syndrome; allergic myocardial infarction; contrast media; ventricular tachycardia

ABSTRACT:

Kounis syndrome (KS) is defined as the occurrence of acute coronary syndromes in the setting of hypersensitivity reactions mediated by mast-cell activation. Various triggers have been implicated, including iodinated contrast agents. We report a 65-year-old woman who developed hypotension and unstable monomorphic ventricular tachycardia approximately 30 minutes after administration of iodinated contrast for computed tomography of the abdomen. Cardiac biomarkers were elevated, and echocardiography demonstrated regional wall motion abnormalities with reduced ejection fraction. Coronary angiography revealed non-obstructive coronary artery disease. The temporal relationship between contrast exposure and myocardial injury supported a diagnosis of contrast-induced Kounis syndrome. This case highlights a rare and life-threatening presentation of KS with malignant ventricular arrhythmia and underscores the importance of recognizing allergic triggers of acute coronary syndromes in emergency settings.

Introduction

Kounis syndrome (KS) describes the occurrence of acute coronary syndrome in the context of an allergic or hypersensitivity reaction. First described by Kounis and Zavras in 1991 as allergic angina, the condition represents a complex interaction between immunologic activation and coronary pathophysiology.(1) Activation of mast cells during hypersensitivity reactions leads to the release of vasoactive and inflammatory mediators such as histamine, leukotrienes, platelet-activating factor, and cytokines, which can result in coronary vasospasm, plaque rupture, or stent thrombosis.(2) Three clinical variants have been described. Type I occurs in patients with normal coronary arteries and is associated with coronary vasospasm. Type II occurs in patients with pre-existing atherosclerotic disease in whom inflammatory mediators precipitate plaque rupture or thrombosis. Type III involves hypersensitivity-mediated stent thrombosis.(2) Numerous triggers have been implicated, including antibiotics, nonsteroidal anti-inflammatory drugs, foods, insect stings, and iodinated contrast agents.(3-6) Although chest pain is the most common presentation, severe ventricular arrhythmias such as ventricular tachycardia (VT) and ventricular fibrillation have been infrequently reported.(7-9) This case was reported from the Department of Emergency Medicine, Sri Ramachandra Institute of Higher Education and Research, Chennai, Tamil Nadu, India.

Case Report

A 65-year-old woman with a history of hypertension presented to the emergency department with fever, right upper abdominal pain, and non-bilious vomiting for three days. She had no prior history of coronary artery disease and no known allergies. On arrival, she was febrile (99.9°F) but hemodynamically stable with a blood pressure of 140/80 mmHg, heart rate of 94 bpm, and oxygen saturation of 99% on room air. Initial electrocardiography showed normal sinus rhythm without ischemic changes (Figure 1). Venous blood gas analysis demonstrated mild lactic acidosis (lactate 3.03 mmol/L). Ultrasonography and contrast-enhanced computed tomography of the abdomen demonstrated

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findings suggestive of acalculous cholecystitis. Approximately 30 minutes after contrast administration, the patient developed chills and profuse sweating and was treated with intravenous pheniramine (45 mg) and hydrocortisone (200 mg) for a suspected allergic reaction. Shortly thereafter, she developed chest discomfort, dyspnea, and hypotension with a heart rate of 160 bpm, blood pressure of 80/60 mmHg, and oxygen saturation of 89% on room air. A 12-lead electrocardiogram revealed sustained monomorphic ventricular tachycardia (Figure 2).

Synchronized cardioversion was performed (50 J, 50 J, and 100 J) for monomorphic ventricular tachycardia with pulse, but ventricular tachycardia persisted. Intravenous amiodarone was initiated (150 mg bolus followed by infusion at 1 mg/min), along with magnesium sulfate (2 g). Due to ongoing hemodynamic instability, the patient was intubated and vasopressor support with norepinephrine (0.3 mcg/kg/min) was initiated. The patient subsequently reverted to sinus rhythm after 6 hours of amiodarone infusion. Initial troponin-I was 0.10 ng/mL prior to cardioversion and increased to 3.96 ng/mL after 6 hours, with elevated CK-MB and BNP levels. Transthoracic echocardiography revealed regional wall motion abnormalities over mid and basal septum, apical septum, LV apex, apical lateral, mid and apical inferior, apical anterior with an ejection fraction of 36%. Patient was given tablet Aspirin 325mg, Clopidogrel 300mg and Atorvastatin 80mg via nasogastric tube along with Inj. Heparin 5000 IU intravenously. Coronary angiography demonstrated non-obstructive coronary artery disease, with approximately 50% ostial right coronary artery stenosis (Figure 3). The patient improved with supportive management and was discharged on hospital day five. The temporal relationship between iodinated contrast exposure and myocardial injury in the absence of obstructive coronary disease supported a diagnosis of contrast-induced Type II Kounis syndrome.

Discussion

This case is clinically important because it captures several defining but easily missed features of Kounis syndrome: a clear temporal relationship to iodinated contrast exposure, rapid progression from suspected hypersensitivity to acute coronary injury, and an atypical electrical presentation with sustained monomorphic ventricular tachycardia rather than the more familiar ST-segment elevation pattern.(10, 11) Kounis syndrome remains an underrecognized form of allergic acute coronary syndrome, and in the recent systematic review by Cahuapaza-Gutierrez et al. including 214 reported patients, chest pain was the most frequent symptom (66.35%), dyspnea occurred in 34.11%, hypertension was the commonest comorbidity (33.64%), and overall mortality was 7.47%, underscoring the need for prompt recognition in emergency settings.(12)

The pathophysiologic sequence in the present patient is highly compatible with mediator-driven coronary injury. Kounis syndrome is understood as mast-cell and platelet activation during an allergic or hypersensitivity reaction, with release of histamine, platelet-activating factor, leukotrienes, neutral proteases, cytokines, and chemokines that can induce coronary vasospasm, promote platelet aggregation, and destabilize atherosclerotic plaque. Thwe et al. emphasized that these mediators may trigger either coronary spasm or acute thrombosis,(10) while Kounis in Clinical Therapeutics described histamine, tryptase, chymase, cathepsin-D, arachidonic acid products, and platelet-activating factor as central effectors in this coronary hypersensitivity disorder.(13) In our patient, the immediate allergic symptoms after contrast exposure, followed within minutes by chest discomfort, hypotension, biomarker rise, regional wall motion abnormalities, and angiographic evidence of underlying coronary disease, fit this mechanism closely.(14) The trigger in this report—iodinated contrast—is uncommon but well recognized. The 2024 ACR Manual on Contrast Media states that allergic-like reactions to modern iodinated contrast media are uncommon, with an aggregate incidence of about 0.6% and severe reactions around 0.04%.(15) Against that background of low absolute risk, Wang et al. reviewed 26 published cases of contrast-induced Kounis syndrome and found that 12 were related to iodine-containing contrast media, the median age was 60 years, onset was usually within 30 minutes of administration, and 24 of 26 patients recovered completely, although 2 died.(16) The onset in our patient at approximately 30 minutes after contrast exposure therefore follows the most typical time window described for contrast-triggered Kounis syndrome.

Classification of the present event as Type II Kounis syndrome is also well supported. Type II disease occurs

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in patients with pre-existing but often clinically silent atheromatous coronary disease, in whom the allergic inflammatory surge precipitates vasospasm together with plaque erosion or rupture, producing myocardial infarction. Nasrollahi et al. similarly stressed that Kounis syndrome should be separated from uncomplicated anaphylaxis because it may reflect plaque-related coronary injury requiring urgent cardiac evaluation,(14) and Thwe et al. noted that Type II cases should initially be treated according to acute coronary syndrome protocols, with concurrent therapy for the allergic reaction.(10) The approximately 50% ostial right coronary artery stenosis in our patient indicates underlying atherosclerotic substrate, making a Type II designation more appropriate than Type I vasospastic disease in angiographically normal coronaries.

A particularly notable feature of this case is the electrical phenotype. In the contrast-induced series by Wang et al., ST-segment elevation was the predominant electrocardiographic pattern, whereas malignant ventricular arrhythmias were far less commonly described.(16) Campo et al. reported a Kounis-Zavras syndrome case complicated by malignant ventricular arrhythmias and cardiogenic shock,(7) and Lameiras et al. documented sustained monomorphic ventricular tachycardia with pulse in another Kounis presentation, showing that ventricular tachyarrhythmia is possible but distinctly unusual.(17) Our case therefore broadens the recognized spectrum of contrast-induced Kounis syndrome by showing that sustained monomorphic VT may be the dominant early manifestation of allergic coronary injury, even when the initial ECG before contrast exposure is normal.

The marked troponin rise from 0.10 ng/mL to 3.96 ng/mL, elevation of CK-MB and BNP, extensive regional wall motion abnormalities, and left ventricular ejection fraction of 36% indicate that this was not a purely electrophysiologic event but a true ischemic myocardial insult. Cahuapaza-Gutierrez et al. found that elevated troponin I was common in reported cases,(12) while Wang et al. observed that echocardiography in contrast-induced cases ranged from normal studies to abnormalities consistent with ischemia, and angiography could show vasospasm, occlusion, stenosis, or even normal vessels.(16) In that context, our patient appears to represent a more severe hemodynamic phenotype, with transient but substantial left ventricular dysfunction accompanying the arrhythmic presentation. This case also highlights an important diagnostic pitfall: Kounis syndrome may evolve without prominent cutaneous manifestations. The patient had chills and profuse sweating with hypotension and respiratory compromise, but no rash or urticaria were documented. Adachi et al. specifically reported Kounis syndrome caused by anaphylaxis without skin manifestations after cefazolin,(18) and Forzese et al. noted that absence of cutaneous findings should not exclude the diagnosis.(19) This is relevant in emergency medicine because hypotension after contrast exposure may be attributed to vasovagal reaction, sepsis, or isolated anaphylaxis; however, the combination of allergic timing, chest symptoms, biomarker elevation, ventricular tachycardia, and echocardiographic ischemia should immediately raise suspicion for Kounis syndrome.(14)

Management in this patient was appropriate and reflects the therapeutic tension inherent to Kounis syndrome. Standard resuscitation for unstable monomorphic VT includes synchronized cardioversion and antiarrhythmic support; the AHA adult tachycardia algorithm lists amiodarone 150 mg over 10 minutes followed by 1 mg/min infusion as a recommended infusion strategy for wide-QRS tachycardia. At the same time, Thwe et al. emphasized that epinephrine, while first-line in classic anaphylaxis, can aggravate ischemia and coronary spasm in Kounis syndrome, and beta-blockers may worsen vasospasm through unopposed alpha-adrenergic activity.(10) The present case therefore illustrates the need to balance arrhythmia control, hemodynamic stabilization, allergic treatment, and acute coronary syndrome therapy rather than managing the episode as isolated anaphylaxis alone. The decision to proceed with aspirin, clopidogrel, atorvastatin, and heparin, followed by coronary angiography, was also justified by the suspected Type II phenotype. Nasrollahi et al. explicitly noted that percutaneous coronary evaluation should not be delayed in Kounis syndrome because coronary vasospasm, plaque rupture, or thrombosis may coexist, and worse outcomes have been described when PCI is deferred.(14) In the broader inpatient analysis by Desai et al., Kounis syndrome was associated with significantly greater mortality than allergic reactions without Kounis syndrome, with an adjusted odds ratio of 9.74, reinforcing the need for aggressive early cardiac assessment once myocardial ischemia is suspected.(11)

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Another useful lesson from this case is the role of ancillary biomarkers. Serum tryptase was not reported, but this marker can strengthen the allergic mechanism when measured during the acute phase. Forlani et al. recommended anaphylactic biomarkers such as tryptase during acute coronary syndromes when Kounis syndrome is suspected,(20) while Pastorello et al. showed in 65 ACS patients that serum tryptase measured during index admission correlated with major adverse cardiovascular events at 2 years.(21) Although diagnosis in the present case is sufficiently supported by timing, clinical course, ECG, biomarkers, echocardiography, and angiography, early measurement of tryptase in future similar presentations may improve diagnostic confidence and help distinguish allergic ACS from conventional plaque-mediated infarction or isolated anaphylactic shock.(22)

Conclusion

This case highlights contrast-induced Type II Kounis syndrome as a rare but important cause of acute coronary syndrome presenting with sustained monomorphic ventricular tachycardia and hemodynamic instability. The close temporal relationship with iodinated contrast exposure, rise in cardiac biomarkers, transient left ventricular dysfunction, and non-obstructive coronary artery disease supported the diagnosis. Early recognition and prompt multidisciplinary management were crucial for a favorable outcome. This report emphasizes the need to consider Kounis syndrome in patients who develop allergic manifestations with concurrent cardiac symptoms or arrhythmias after contrast administration.

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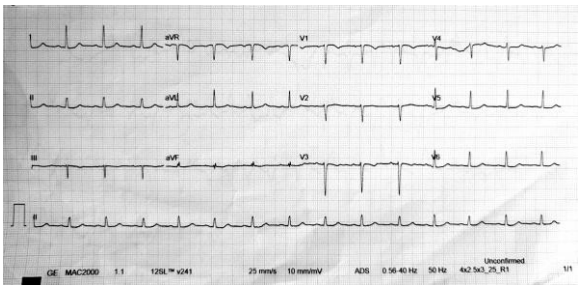

Figure 1: Baseline electrocardiogram demonstrating normal sinus rhythm with no ischemic changes at the time of presentation to ED

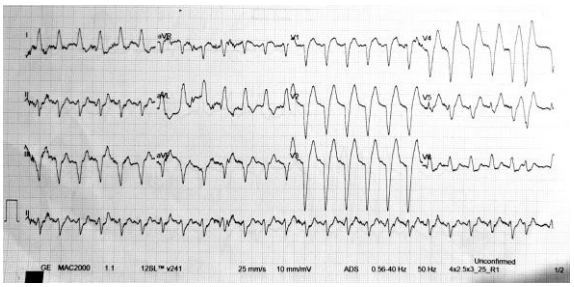

Figure 2: Electrocardiogram demonstrating sustained monomorphic ventricular tachycardia approximately 30 minutes after administration of intravenous iodinated contrast

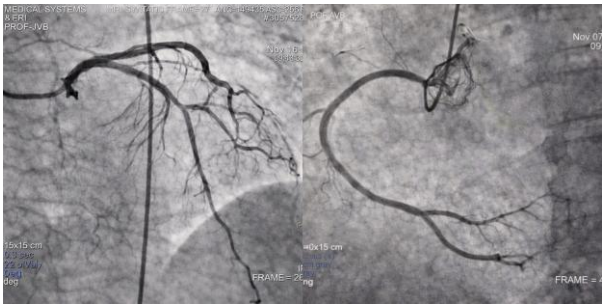

Figure 3: Coronary angiography demonstrating non-obstructive coronary artery disease