

Kounis Syndrome: A Rare Mediator of Acute Coronary Syndrome

Amogh M. Joshi DO

Lehigh Valley Health Network, Amogh.Joshi@lvhn.org

Chun Siu

Lehigh Valley Health Network, chun.siu@lvhn.org

Zeel Patel DO

Lehigh Valley Health Network, zeel.patel@lvhn.org

Mirza Ali

Lehigh Valley Health Network, mirza.ali@lvhn.org

Amy Lam DO

Lehigh Valley Health Network, amy.lam@lvhn.org

See next page for additional authors

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Authors

Amogh M. Joshi DO, Chun Siu, Zeel Patel DO, Mirza Ali, Amy Lam DO, Tarick Sheikh, Sagar Vadhar DO, Akhil Kher MD, and Apurva Vyas MD

Kounis Syndrome: A Rare Mediator of Acute Coronary Syndrome

Amogh M. Joshi, DO,¹ Chun Siu, DO,² Zeel J. Patel, DO,² Mirza Ali, MD,² Amy Lam, DO,² Tarick Sheikh, MD,¹ Sagar Vadhar, DO,¹ Akhil Kher, MD,¹ Apurva Vijay Vyas, MD¹

¹Department of Cardiology, ²Department of Medicine, Lehigh Valley Health Network, Allentown, PA

Background

Kounis Syndrome (KS) is a rare mediator of ACS precipitated by mast cell and platelet activation in the setting of hypersensitivity reactions. The pathophysiology is thought to be mediated by the induction of inflammatory cytokines, after an allergic insult that potentiates coronary vasospasm, plaque rupture or in-stent thrombosis. Clinical suspicion should be high in patients that present with ACS and a history of allergic insult. Below, we describe the case of a patient with anaphylaxis and rapid progression to STEMI.

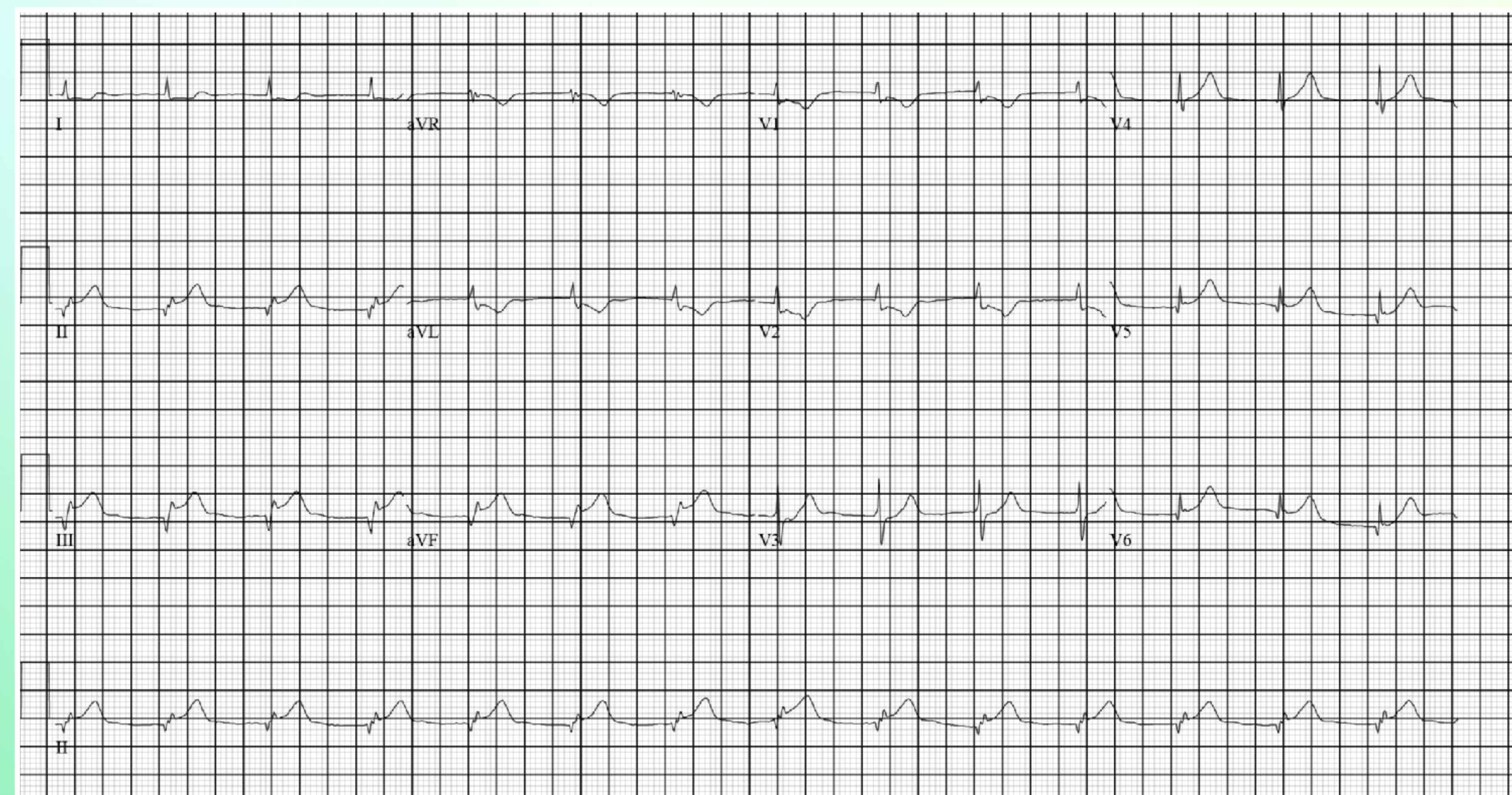


IMAGE 1: Inferior ST Elevation MI

Given the confluence of two distinct life-threatening conditions associated with Kounis Syndrome, clinicians must remain cognizant of this unique and rare disease presentation.

Case

A 59 year old man with a history of CAD status post RCA stent and COPD presented with progressively worsening chest pain, dyspnea, and hives after a bee sting. Electrocardiogram demonstrated an inferior STEMI. The patient received IM epinephrine and was loaded with aspirin, clopidogrel and heparin and taken for emergent coronary angiography.

Decision-making

Cardiac catheterization demonstrated an in-stent thrombosis in the mid to distal portion of a large, dominant RCA. Percutaneous intervention with a drug-eluting stent was performed. The patient was continued on aspirin, clopidogrel and diphenhydramine with progressive clinical improvement and subsequent discharge in stable condition.

Conclusion

Kounis Syndrome can be subdivided into three categories. Type I and II are seen in patients without CAD and asymptomatic CAD respectively. While Type III is seen with hypersensitivity reactions leading to in-stent thrombosis. Percutaneous coronary intervention and initiation of anti-thrombotic therapy should be pursued as appropriate, and intravenous anti-histamines should be initiated for management of the allergic response. Beta-blocker therapy, while appropriate in MI, is contraindicated in KS due to the risk of unopposed alpha-adrenergic activity with concurrent epinephrine administration. Intramuscular epinephrine is preferred to intravenous administration due to risk of ischemia and vasospasm. Given the confluence of two distinct life-threatening conditions, clinicians must remain cognizant of this severe overlap syndrome.