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Kounis Syndrome: Case Report, Pathophysiology and Management

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Abstract **Case Report**

Kounis syndrome is an acute coronary syndrome occurring in a context of anaphylaxis, it still an underdiagnosed syndrome. The prognosis of Kounis syndrome remains better than a conventional acute coronary syndrome, we describe 3types of Kounis syndrome with different kind of management, however further management guidelines are still needed to avoid misdiagnose or make the prognosis worse. We report a case of 24 years old Kounis syndrome after a sting of bee, its management in our ED.

Keywords: Kounis syndrome – Acute coronary syndrome- sting of bee.

INTRODUCTION

The occurrence of an acute coronary syndrome (ACS) in elderly is a serious condition but not rare, however when it happens in adulthood, it represents a rare emergency, especially after a sting of a bee.

This entity was first described by Kounis as an acute coronary syndrome in an anaphylaxis context, but this syndrome remains underdiagnosed.

Many studies have shown increasing lifetime prevalence of allergy and anaphylaxis with an estimated lifetime risk of 0.02-2.0% [1]. As a result, more cases of Kounis syndrome are being reported in the literature.

In this report, we describe a case of Kounis syndrome (KS) presenting with ST elevation associated with urticaria.

CASE REPORT

A 24-year-old male patient, known to have no medical condition, non-smoker, presented with a one-hour history of diffuse pruritic rash and retrosternal chest pain radiating to the left shoulder, associated with vomiting. he denied being exposed to penicillin, new drugs, foods or environmental allergens but he reported a sting of a bee 75 min ago. On presentation, his vital signs were unstable (temperature 37.7C, blood pressure 80/41 mmHg, heart rate 95 bpm, respiratory rate 22 breaths per minute). Physical examination revealed a well-developed man with a circumscribed punctuated sting mark with mild local swelling in the right arm. He was diaphoretic and in pain (EVA 7/10).

Twelve lead ECG was taken and showed ST segment elevation in the anterior leads (Figure 1). His basic laboratory studies were unremarkable except for a baseline troponin T of 1.34 ng/mL (0 – 0.014) that increased to 1.47 ng/mL after 6 hours.

In the ED, 0.5 ml (1:1000) adrenalin IM, Hydrocortisone 200 mg IV and ranitidine were given immediately with resolution of the rash and pruritus.

He felt better and improved over next 30 minutes and her blood pressure picked up to 106/70 mmHg. Symptomatically he became better with reduced chest tightness, chest pain and breathlessness. However, the repeat ECG taken 30 minutes later showed the same changes as first ECG. He was kept under observation in ED and he didn't develop any new symptoms. Blood pressure remained stable. The ECGs taken that the sixth hours after the admission (Figure 2) were normal with disappearance of ST and T in the first ECG. The Troponin T level was done again 12 hours after stinging and was within normal range.

Medications indicated for acute coronary syndrome such as heparin, nitrates and statins were not given as ECG changes were transient and he felt free of chest pain. He was discharge from the hospital after 48 hours and was followed up in the out patients clinic.

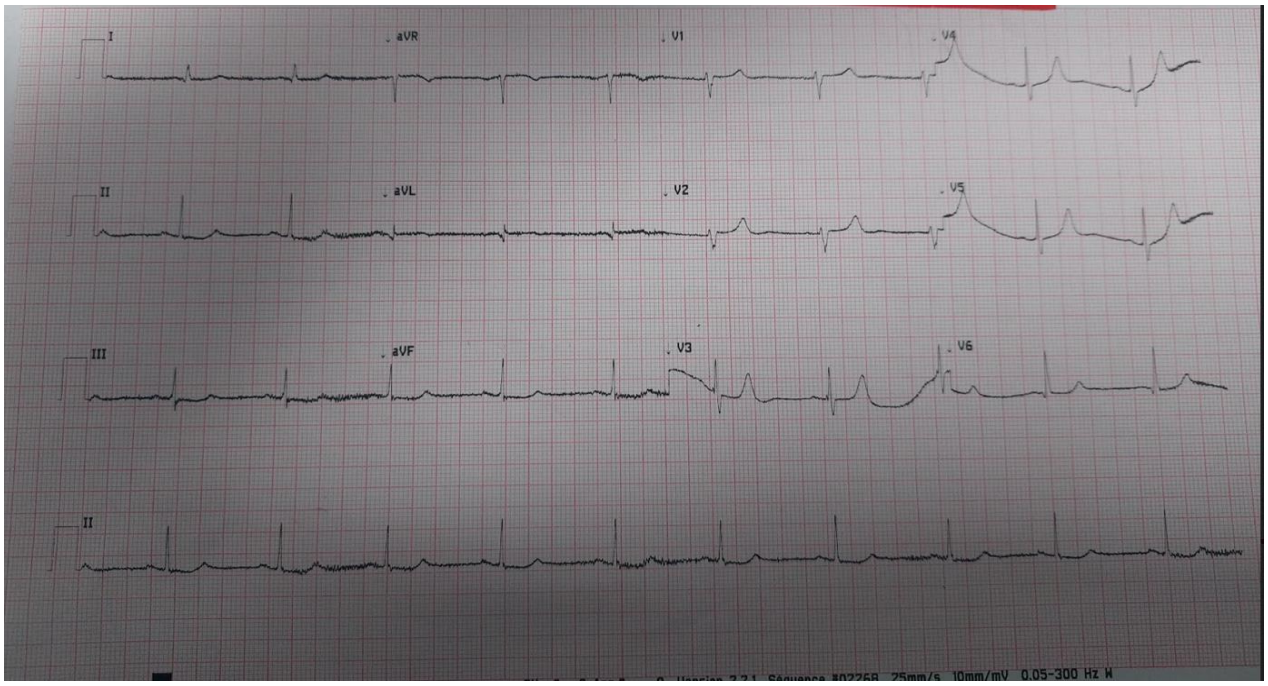

Figure 1: ECG revealing ST elevation in the anterior leads

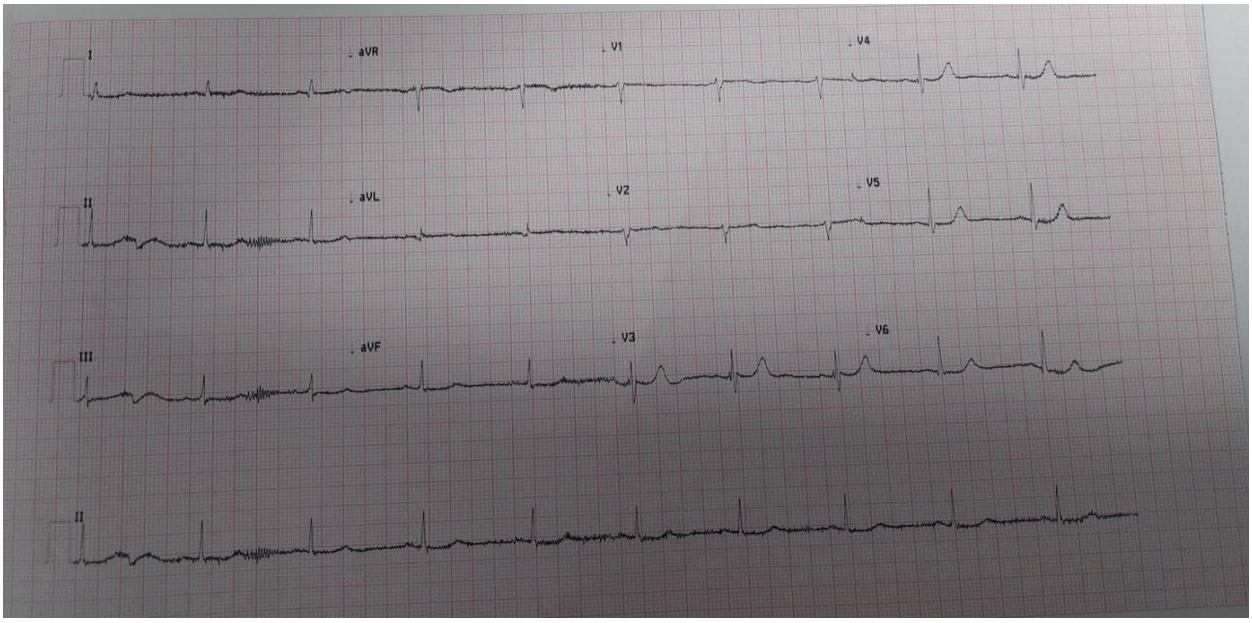

Figure 2: ECG revealing absence of ST elevation at the sixth hour

DISCUSSION

First described in the 1991 by Kounis and Zavas, the KS is defined by the occurrence of acute coronary syndromes in the setting of allergic or anaphylactic events.

It most commonly affects individuals between the ages of 40 and 70, with history of atopy, hypertension, smoking, diabetes or hyperlipidemia. KS should be suspected in patients with or without cardiovascular risk factors or pre-existing coronary artery disease (CAD) who present to the ED with chest pain or angina-equivalent symptoms accompanied by symptoms of systemic allergic reactions [2].

The pathophysiology of Kounis syndrome is known to be due to inflammatory mediators released from mast cell activation induced by allergic or hypersensitivity reaction. The reaction to Hymenoptera sting can be prolonged or severe in atopic individuals

and in some cases, anaphylaxis may ensue, with urticaria, circulatory collapse and bronchospasm with secretion of histamine, serotonin and leukotrienes.

Endogenous secretion of adrenaline and noradrenaline is stimulated by histamine and serotonin [3, 4]. All these substances can provoke myocardial ischemia either through profound hypotension or by increasing myocardial oxygen demand through direct ionotropic or chronotropic effect in the presence of a compromised myocardial blood supply.

Platelet aggregation is induced by serotonin and adrenaline. Adrenaline also accelerates thrombus formation in animals and in man possibly by increased factor v activity [5, 6] and shown in animals to release a thromboplastin-like substance from the walls of blood vessels. It causes both coronary vasodilatation and increased myocardial oxygen demand by direct ionotropic and chronotropic effect. Adrenaline has been used historically as a provocation test for angina pectoris and often used in the treatment of anaphylactic shock [6].

In the course of anaphylactic reaction, complements are activated with anaphylatoxin generation. The specific receptors for them are present on surface of cardiac mast cells [7]. The final step of this processes is mast cells degranulation, resulting in histamine, tryptase release, as well as in prostaglandin and leukotriene synthesis. The histamine concentration is elevated in arterial walls containing atheromatic changes [7]. Also, proteases (Tryptase, Chymase and Cathepsin-D) released from stimulated mast cells activate metalloproteinases (MMP: MMP-1, MMP-3 and MMP-9) which degrade connective tissue covering the atheromatous plaque. The plaque becomes vulnerable more prone to rupture [7]. The development of Kounis syndrome is a consequence of the above processes.

Three types have been described as follows:

- Type 1 describes patients without pre-existing coronary artery disease or risk factors who develop angina during an allergic reaction because of coronary artery spasm.
- Type 2 describes patients with a pre-existing, asymptomatic or symptomatic coronary artery disease in which an allergic reaction results in either coronary artery spasm or plaque erosion or rupture and myocardial infarction.
- Type 3 refers to allergic-induced coronary artery stent thrombosis in patients with pre-existing coronary artery disease (CAD) and stents.

Therapeutic management of KS is challenging as it aims for myocardial revascularization as well as treatment of the concomitant allergic reaction. Guidelines for the treatment of ACS are lacking management of KS, and most of the evidence on the efficacy of the treatment is based on individual case reports or case series.

Management of type I and II KS consists of treating the allergic reaction with H1 and H2 antihistamines with or without intravenous corticosteroids, as well as the ACS [8].

ACS management guidelines (antiplatelet and anticoagulant therapy, reperfusion guidelines) should be followed with special attention to some medications: Beta-blockers may precipitate coronary spasm due to unopposed alpha agonism, especially if used concomitantly with epinephrine [9]. Vasodilators (nitrates, calcium channel blockers) help resolve vasospasm, but may worsen hypotension in patients with vasodilation from anaphylaxis [8]. Fentanyl and its derivatives are preferred for pain control since other opiates may worsen histamine release [10].

For type III KS, cardiac catheterization with aspiration of the stent thrombus is recommended, with histologic examination of aspirated contents and staining for eosinophils and mast cells [8].

CONCLUSION

Kounis syndrome is an underdiagnosed syndrome, it presents a challenge to diagnose early in emergency and to give appropriate management. Its prognosis is better than a conventional ACS especially type I, however further management guidelines are still needed.

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Abbreviations

KS: Kounis syndrome
ACS: acute coronary syndrome
ECG: electrocardiogram
ED: emergency department
IM: intra muscular
IV: Intra venous
CAD: coronary artery disease

REFERENCES

1. Clemen, B., Nwosu, I., Chukwuka, N., Cordeiro, N. L., Ibeson, E., Gulati, A., ... & Weindorf, B. (2021). Recognizing Kounis Syndrome: A Report of Type 2 Kounis Syndrome and a Brief Review of Management. *Cureus*, *13*(11), e19712.
2. Rajha, E., Didi, A., Dakik, H., & Mufarrij, A. (2020). Acute ST Elevation myocardial infarction due to allergic reaction, Kounis syndrome. *The American Journal of Emergency Medicine*, *38*(2), 409-e5.
3. Ralapanawa, D. M. P. U. K., & Kularatne, S. A. M. (2014). A case of Kounis syndrome after a hornet

sting and literature review. *BMC Research Notes*, *7*(1), 1-5.
4. Levine, H. D. (1976). Acute myocardial infarction following wasp sting: report of two cases and critical survey of the literature. *American Heart Journal*, *91*(3), 365-374.
5. Milne, M. D. (1949). Unusual case of coronary thrombosis. *British Medical Journal*, *1*(4616), 1123.
6. O'brien, J. R. (1963). Some effects of adrenaline and anti-adrenaline compounds on platelets in vitro and in vivo. *Nature*, *200*(4908), 763-764.
7. Sinkiewicz, W., Sobański, P., & Bartuzi, Z. (2008). Allergic myocardial infarction. *Cardiology Journal*, *15*(3), 220-225.
8. Abdelghany, M., Subedi, R., Shah, S., & Kozman, H. (2017). Kounis syndrome: a review article on epidemiology, diagnostic findings, management and complications of allergic acute coronary syndrome. *International Journal of Cardiology*, *232*, 1-4.
9. Lieberman, P., & Simons, F. E. R. (2015). Anaphylaxis and cardiovascular disease: therapeutic dilemmas. *Clinical & Experimental Allergy*, *45*(8), 1288-1295.
10. Kounis, N. G. (2016). Kounis syndrome: an update on epidemiology, pathogenesis, diagnosis and therapeutic management. *Clinical Chemistry and Laboratory Medicine (CCLM)*, *54*(10), 1545-1559.