IOSR Journal of Dental and Medical Sciences (IOSR-JDMS)
e-ISSN: 2279-0853, p-ISSN: 2279-0861.Volume 22, Issue 2 Ser.6 (February. 2023), PP 49-51
www.iosrjournals.org

Kounis Syndrome – Vancomycin Induced Acute Coronary Syndrome.

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ABSTRACT:
We report a case of Kounis syndrome , an allergic reaction causing coronary artery vasospasm due to release of inflammatory cytokines through mast cell activation. A case of 41-year-old female diagnosed as disseminated tuberculosis with superadded MRSA infection, with no riskfactors for coronary artery disease developed coronary artery spasm, after 2 hours of initiation of intravenous vancomycin .ECG revealed significant ST-T changes, elevated cardiac enzymes. Drug reaction was suspected, Symptoms and ECG changes resolved after stopping vancomycin and administration of isosorbide mononitrate.

Date of Submission: 27-01-2023 Date of Acceptance: 09-02-2023

I. CASE:

A 41-year-old female presented with low grade fever and weight loss for 5 months. She was evaluated and diagnosed as disseminated TB and secondary bacterial infection.Sputum culture grew MRSA sensitive to Vancomycin, intravenous vancomycin 500mg was started after test dose. Two hours after infusion, patient developed retrosternal chest pain and breathlessness, typical of angina.

ECG showed ST-T changes in V1, V2, V3, V4 (FIGA) and cardiac marker, troponin I was elevated. Point of care 2DECHO revealed no regional wall motion abnormality and EF60%.

As patient did not have any cardiovascular risk factors or pasthistory of CAD, drugreaction was suspected, and the offending agent intravenous vancomycin was stopped promptly, and patient was treated with isosorbide mononitrate.

Her symptoms relieved completely, and serial ECG's showed resolution of changes (FIGB) and cardiac enzymes returned to baseline.

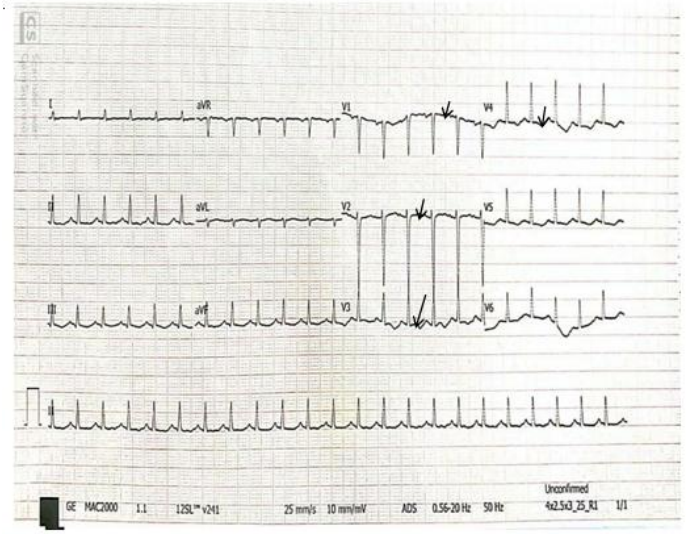

FIG A: Two hours after intravenousvancomycininjection.

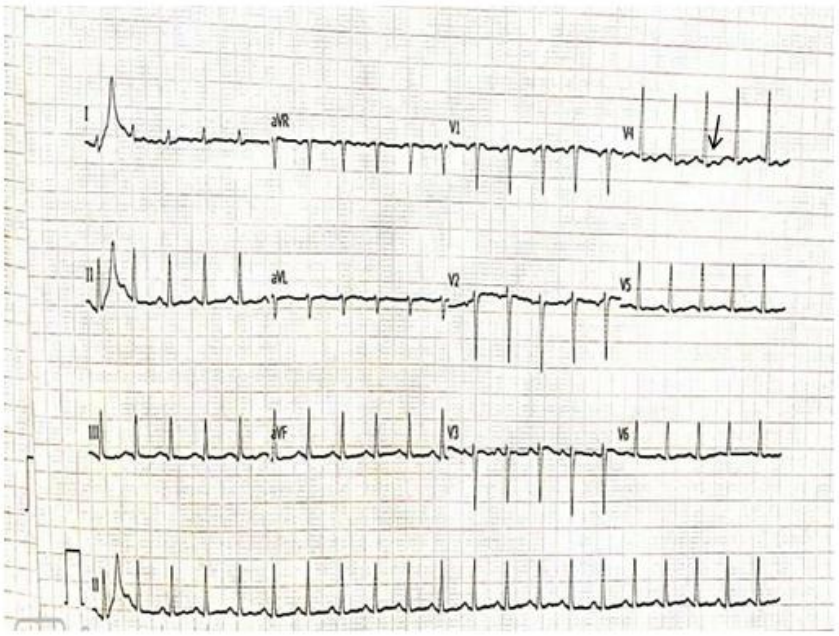

FIG B: After treating with isosorbidemononitrate.

II. DISCUSSION:

We report a case of drug induced vasospastic angina due to injection vancomycinin a 41 year female with no CAD and no atherosclerotic risk factors consistent with Kounis syndrome.The occurrence of acute coronary syndrome due to allergy or hypersensitivity reaction associated with mastcell activation is encountered in clinical practice. It is due to inflammatory mediators such as neutralproteases including tryptase, chymase, arachidonicacidproducts, histamine, plateletactivatingfactor and a variety of cytokines and chemokines released during activation process[1].

In the past, there have been a few Case reports which have shown occurance of Vancomycin induced Kounis syndrome.

Martinez et al has described a case of vancomycin inducedkounis syndrome in an 83-year-old female with previous aortic valve replacement and hyperlipidemia[2].

Leibee et al reported the case of 57-year-old male with peripheral vascular disease, known hypertensive, diabetic and smoker with normal angiography findings and with significantECG changes[3].

Luke R.Gagnon et al reported case of Kounis syndrome secondary to vancomy cinina 32 year old male with inguinal abscess caused by MRSA with normal angiography findings and with significant ECG changes.

Three variants of Kounis syndrome are recognised:
TypeI in which myocardial ischemia is induced through coronary vasospasm without evidence of pre-existing coronary artery diseaseas in this case.

Type II Kounis syndrome syndrome occurs when the hypersensitivity response triggers plaque rupture in keeping with atherosclerotic acute coronary syndrome.

Type III is specific to patients with previous coronaryartery stenting, where chronic allergen exposure and inflammation results in thrombosis of drug eluting stents[2].

Allergic drug reaction can present as:

1. Hypersensitivity-Skin allergy, anaphylactoid component-swelling of face, tongue, wheeze, and potentially very low blood pressure.

2. Cardiaccomponent present as ACS, bradycardia, tachycardia, hypotension, possible cardio respiratory arrest, and death.

Diagnosis is based on ECG findings showing ischemia and elevated cardiacmarkers. Coronary angiography helps to confirm diagnosis, furthermore cardiac MRI shows delayed contrast enhanced images with normal wash out in subendothelial lesion areain type1 variant.

No guidelines exist on treatment of Kounis syndrome except cessation of offending agent to reduce further cardiac injury and vasodilators such as nitroglycerin or calcium channel blockers.

In typeI/IIreaction vasodilators, corticosteroids and antihistamines can be considered.

In type III Kounis syndrome thrombus aspiration and placinga new stent is found to be beneficial[1].

III. CONCLUSION:

This case emphasizes the need for awareness among the physicians regarding the rare entity vancomycin induced Kounis syndrome.The timely recognition and prompt discontinuation of vancomycin will prevent further cardiac injury.

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Dr.Saikiran.D, et. al. "Kounis Syndrome – Vancomycin Induced Acute Coronary Syndrome." *IOSR Journal of Dental and Medical Sciences (IOSR-JDMS)*, 22(2), 2023, pp. 49-51.