

Vasospastic coronary event following a single dose of amoxicillin in a patient with normal coronary arteries: Kounis syndrome and the myocardial infarction with normal coronary arteries conundrum

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In the interesting report of *Pradhan*, *Christ and Trapper* (1) published in the *Journal of Cardiovascular Diagnosis and Therapy*, a 22-year-old man developed acute retrosternal pain and chest tightness with ischemic changes in inferolateral wall (ST elevation =0.2 mV) and positive troponin following a single dose of amoxicillin administered for acute tonsillitis treatment. Urgent coronary angiography demonstrated normal coronary arteries. The initial diagnosis of acute ST elevation myocardial infarction (STEMI) was attributed to coronary artery spasm, whereas acute myocarditis considered as a differential diagnosis.

Indeed, myocardial infarction with normal coronary arteries (MINOCA) constitutes a diagnostic and therapeutic conundrum with various etiologies and pathogenetic mechanisms. Several causes of MINOCA have been incriminated such as coronary artery spasm, myocarditis, Takotsubo syndrome, coronary artery dissection, coronary embolism, arrhythmias, mild plaque disruption, hypercoagulable status, type 2 myocardial infarction, amyloid light-chain (AL) amyloidosis, psycho-emotional disorders and clinically unrecognized Kounis syndrome (2).

The Kounis hypersensitivity-associated type I variant refers to patients with normal or nearly normal coronary arteries without predisposing factors for coronary artery disease representing the most common type (72.6%) of Kounis syndrome. In this variant, the acute release of inflammatory mediators may induce either coronary artery spasm without increased myocardial enzymes or coronary artery spasm progressing to acute myocardial infarction with raised troponin levels. The ensuing acute coronary syndrome could lead to major adverse cardiac events such as cardiogenic shock (2.3%), cardiac arrest (6.3%), death 5 (2.9%) due to ventricular fibrillation, anterior or inferior ST elevation myocardial infarction (3).

In a recent report (4), 9,092 consecutive unique patients out of 199,163 myocardial infarction admissions, presented with MINOCA. Interestingly, 2,147 of MINOCA patients experienced a new major adverse cardiovascular event and 1,254 patients (14%) died during the mean followup of 4.5 years. The electrocardiographic changes of MINOCA patients revealed ST-segment elevation (16.4%), ST-segment depression (16.0%) and T wave abnormalities (13.0%). In another recent report (5) a 75-year-old female with history of non-ischemic dilated cardiomyopathy of unknown etiology, presented with witnessed out of the hospital cardiac arrest (OHCA). Increased creatine-kinase and high sensitivity troponin along with the presence of ST-segment elevation supported the diagnosis of acute myocardial infarction, but coronary angiography demonstrated patent, free of stenosis, with no signs of peripheral thrombus embolization coronary arteries, findings compatible with MINOCA. The authors

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expressed their great concern to report the incidence of MINOCA in OHCA patients, as well as the relevant patient characteristics related to this entity, as such data have not been previously published. Furthermore, STsegment elevation with MINOCA has been reported in 3% to 4% of cases of myocardial infarction complicated with out of hospital cardiac arrest and this could be attributed to coronary artery spasm progressing to acute myocardial infarction (6).

All published data support that MINOCA is neither rare nor a benign syndrome. It is anticipated that even more causes of MINOCA would have been added in the near future.

Therefore, in respect to the authors' concern regarding the appropriate diagnosis of this 22-year-old man with STEMI and normal coronary arteries, we suggest a detailed and thorough clinical examination together with diagnostic investigations to identify the causal underlying mechanisms in every MINOCA patient.

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Footnote

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