Rahul Kurapati et al., J Clin Exp Cardiolog 2015, 6:4
http://dx.doi.org/10.4172/2155-9880.S1.024

OMICS International
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5th International Conference on
Clinical & Experimental Cardiology
April 27-29, 2015 Philadelphia, USA

Catastrophic anti-phospholipid syndrome causing non-ST elevation myocardial infarction

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The hypercoagulable state from antiphospholipid syndrome (APS) is well known to increase the risk of thrombosis. Very rarely patients with APS can have widespread thrombotic disease with multiorgan failure, also called "catastrophic APS". Vascular disease often leads to this multiorgan damage, which can include the heart rarely due to coronary artery involvement. We present a patient with multiorgan failure including a non-ST elevation myocardial infarction (NSTEMI).

A 45-year-old female with a previous history of a stroke and deep vein thrombosis admitted with acute renal failure, an elevated troponin and acute development of gangrene in her distal digits of both her feet and hands. Given her previous history and presenting symptoms, there was a clinical suspicion for antiphospholipid antibody. Upon further investigation, lupus anticoagulant was found to be positive. Patient was immediately started on a five-day treatment with plasmapheresis, which lead to normalization of the coagulation tests. The patient's troponin eventually peaked and normalized. Transthoracic echocardiogram showed normal systolic function and no wall motion abnormalities. Therefore, there was no warrant for further invasive evaluation with coronary angiography.

Common cardiac diseases in APS are valvular disorders such as valvular thickening, mitral valve nodules and nonbacterial thickening. APS has also been associated with intracardiac thrombi, pericardial effusion, cardiomyopathy, premature restenosis of vein grafts for coronary bypass and peripheral vascular disease. The relationship between APS and increased incidence of ischemic heart disease has been controversial. It is known that APS makes one hypercoagulable predisposing to thrombus formation. As in the case described above, multiorgan thrombosis took place. In retrospective, we can conclude that the NSTEMI probably was due to a coronary artery thrombus formed by the hypercoagulable state incited by the antiphospholipid antibodies. Due to the multiorgan failure, prompt therapeutic plasma exchange was used to clear these inciting antibodies with a favorable outcome. We present this case as the risk of thrombosis in the cardiac vasculature can be reduced with early diagnosis of APS and initiation of appropriate therapy in conjunction to traditional lifestyle modifications.

Biography

Rahul Kurapati has completed his MD from Dr. B. R. Ambedkar Medical College and is currently a PGY-1Internal Medicine resident at Baton Rouge General, an affiliate of Tulane University School of Medicine, in Baton Rouge, Louisiana. Has a future goal to pursue a cardiology fellowship.

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