Catch 22: To Anticoagulate or Not to Anticoagulate

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Introduction

Coronary artery ectasia (CAE) is diffuse dilatation exceeding more than 1/3 the coronary artery length and diameter exceeding the adjacent reference segments by 1.5 times. CAE is found incidentally on diagnostic coronary angiography with incidences of 1.4 to 4.9% and is an uncommon cause of acute coronary syndrome (ACS). The most common etiology is atherosclerosis, but could be congenital or secondary to sequelae or vasculitic coronary disorders. These ectatic segments may serve as a nidus for thrombus formation with possibility of distal embolization. Thus, patients with CAE have worse outcomes than the general population. Due to the stagnant non-laminar flow associated with these segments, anticoagulation has been recommended for larger caliber aneurysms on the basis of an increased risk of thrombosis and myocardial infarction that is not related to plaque rupture. However, the benefits of anticoagulation must outweigh the risk of bleeding.

Case description

78 y/o Ecuadorian female with history of hypertension, dyslipidemia and prior inferior MI treated initially with balloon angioplasty in South America followed by RCA stent in 2014 presented with worsening typical chest pain. Radiating to her left arm and South America followed by RCA stent in 2014 presented with childhood illness suggestive of Kawasaki disease. Admits to social aspirin, atenolol, hydralazine and atorvastatin. No history of febrile and prior inferior MI treated initially with balloon angioplasty in association with exercise for the last 6 months. Medications included aspirin, atenolol, hydralazine and atorvastatin. No history of febrile childhood illness suggestive of Kawasaki disease. Admits to social tobacco use. Denied alcohol or illicit drug use.

On initial assessment, patient was in no acute distress. Vital signs were stable. On exam, regular heart sounds, no jugular venous distention or lower extremity edema was noted. Peak troponin I elevated at 2.9 ng/mL. EKG showed a right bundle branch block with nonspecific ST segment and T wave changes. Q waves were noted in the inferior leads. Echocardiogram revealed normal LV function, with an ejection fraction of 60% with no valvular disease. Coronary angiography revealed 90% stenosis of the posterior descending artery (PDA), with dilation and severe ectasia affecting multiple major vessels (LMCA, LAD, LCx, RCA). A drug eluting stent was deployed to relieve the stenosis in the PDA.

Discussion

Treatment options for CAE include risk-factor modification, dual anti-platelet therapy (DAPT) and antithrombotic therapy. Anticoagulation therapy to prevent coronary thrombus formation has been a controversial topic because of limited randomized trials demonstrating its benefit in CAE. The therapeutic decision should be focused on how to reduce the heavy thrombus burden to improve coronary flow. If the patient’s response to standard DAPT is inadequate, then warfarin or novel oral anticoagulation (NOACs) may provide an alternative in combination with DAPT. In this case of Type I coronary artery ectasia the patient was deemed not to be high risk for recurrent thrombosis and stenosis. She was placed on aspirin and clopidogrel. The treatment of CAE is a vexing clinical question and the decision to anticoagulate must be made on an individual basis based on thrombosis VS bleeding risk until further studies are done to evaluate the clinical efficacy of this strategy.

1. Distinguish coronary ectasia in the realm of aneurysmal coronary disease
2. Discuss how the management of coronary ectasia often poses a significant challenge

References