

Optimizing anticoagulants and antiplatelets in high risk patients

Eric Williams, MD

Story from the front lines:

An elderly man with PMH significant for extensive CAD (including CABG), HFrEF/ICM, a-fib/flutter, CKD presented with shortness of breath, chest pain, volume overload, generally feeling poor. In 2016, he presented to an OSH with chest pain, and was found to have an NSTEMI, with coronary angiography demonstrating 100% occlusion of LIMA to LAD (and no patent saphenous grafts), CTO of his native LAD, as well as 100% occlusion of the PDA. During that hospitalization, was considered for repeat CABG, but family was unwilling to proceed, so he was referred to a CTO clinic for possible intervention on the LAD and discharged on apixaban for paroxysmal AF. In 6/2017, he underwent elective coronary angiography for intervention on RCA, LAD, or both. At that time, he was found to have a significant 70% stenosis of the proximal RCA that was treated with a DES. He was loaded with clopidogrel, and discharged on DAPT without any anticoagulation despite his history of atrial fibrillation and risk of thrombotic events (CHA2DS2-VASc of 4). Per chart review, he had not been on any A/C since discharge in June 2017. During this hospitalization in 4/2018, he was found to be in a fib with RVR. Repeat TTE demonstrated a “large laminated left ventricular apical thrombus.” CHA2DS2-VASc score is calculated to be at least 4 (age, CHF, HTN, and vascular disease).

Teachable Moment:

As more and more information is gathered, it is becoming increasingly clear that triple oral antithrombotic therapy (TOAT) is not of significant benefit in reducing ischemic events, while increasing the risk of bleeding in patients with atrial fibrillation and recent stent. We now have multiple randomized trials to guide our TOAT vs dual therapy decision. The WOEST (What is the Optimal Antiplatelet and Anticoagulant Therapy in Patients with Oral Anticoagulation and Stenting)¹, PIONEER AF-PCI trial (Open-Label, Randomized, Controlled, Multicenter Study Exploring Two Treatment Strategies of Rivaroxaban and a Dose-Adjusted Oral Vitamin K Antagonist Treatment Strategy in Subjects with Atrial Fibrillation who Undergo Percutaneous Coronary Intervention)², and RE-DUAL PCI trial (Randomized Evaluation of Dual Antithrombotic Therapy with Dabigatran versus Triple Therapy with Warfarin in Patients with Nonvalvular Atrial Fibrillation Undergoing Percutaneous Coronary Intervention)³ all significantly demonstrate elevated risk of bleeding with TOAT (although choice of anticoagulant is different among the three trials) while not significantly reducing thrombotic events (MI, stroke, systemic embolism, unplanned revascularization, stent thrombosis). A recent meta-analysis by Piccini and Jones⁴ found that the odds of major bleeding with dual therapy was half that of TOAT (OR, 0.49; 95% CI, 0.34 to 0.72; P<0.001). They also examined the risk of thrombotic events. Although there is heterogeneity with each trial using a different oral anticoagulant (warfarin, rivaroxaban, and dabigatran, respectively), the meta-analysis suggests that the risk of thrombotic cardiovascular events is not higher with dual therapy as compared to TOAT (OR, 0.80; 95% CI, 0.58 to 1.09; P=0.16).

In our patient's unique circumstance, TOAT and dual therapy was abandoned for DAPT after he received a stent. It is known that DAPT is not an appropriate choice for thrombosis prevention in a fib alone. With additional information coming out on dual

therapy vs TOAT, clinicians should feel comfortable dropping DAPT after PCI in patients with atrial fibrillation for dual therapy with an oral anticoagulant and P2Y12 inhibitor.

References:

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