Chemotherapy-mediated Coronary Artery Vasospasm

Wallace, C; Subramanian, S
Medical College of Wisconsin, Milwaukee, WI

INTRODUCTION
Chemotherapeutic drug induced coronary vasospasm is a rare event, but with the increasing use of new line chemotherapeutic drugs, awareness of this side effect is essential both for prevention and management of coronary vasospasm.

CASE
58 year old male who was recently diagnosed rectal adenocarcinoma and started on chemotherapy (capecitabine) presented to the clinic with complaints of chest pain 1 week after being started on chemotherapy. Chest pain was left sided, present both at rest and on exertion, radiating to left arm and had occasionally woken him from sleep. He did not have dyspnea, syncope or palpitations, or previous cardiac symptoms. Past history was negative for hypertension, hyperlipidemia and diabetes. No family history of premature CAD, no smoking, alcohol, or drug use. During initial office visit, patient’s vitals were stable, physical examination, and labs were unremarkable. Based on his symptoms which were suggestive of angina, an exercise treadmill myocardial perfusion study was ordered to assess for obstructive CAD.

COURSE
Patient exercised for 5 minutes and 10 seconds, and achieved 7 METS. During the exercise phase, he had no EKG changes concerning for ischemia. During recovery, EKG showed diffuse 3 mm ST elevation in leads II, III, aVF, V2-V6. Patient started having chest pain during recovery and he was given sublingual nitrates which resolved the chest pain and ST elevation. Patient was transferred for emergent cardiac catheterization. Left heart catheterization showed mild atherosclerosis in left main, LAD, RCA and LCX. There was a discrete 90% stenosis in the proximal portion of the ramus intermedius third of the vessel segment which was a small caliber vessel. LVEF calculated by contrast ventriculography was 42%. The coronary anatomy did not correlate with the diffuse ST changes seen during stress test and low LVEF. Based on the patient’s recent chemotherapy and lack of correlation between EKG changes and coronary anatomy, the etiology of the chest pain and EKG changes are due to vasospasm secondary to capecitabine.

DISCUSSION
Capecitabine is a flupiramidine antimetabolite which is increasing used as a chemotherapeutic agent for treatment of metastatic colorectal cancer and breast cancer. Cardiotoxicity has been reported in literature, but has been lower than its active metabolite fluorouracil (FU). Patient can present with classic symptoms of angina or myocardial infarction. Patients with preexisting coronary disease have a higher incidence of cardiotoxicity. Management includes discontinuing the capecitabine, though trials with coronary vasodilators have been noted in the literature. Chemotherapeutic drug induced coronary vasospasm is a rare adverse event, but with the increasing use of new line chemotherapeutic drugs and their potential effects on coronary vasculature, the knowledge and awareness of the spectrum of capecitabine toxicity may prevent adverse patient outcomes.

TAKEN HOME POINTS
• Importance of recognizing drug induced coronary vasospasm in chemotherapy drugs
• Mechanism, clinical presentation, and management of drug induced coronary vasospasm

REFERENCES
Yuliya B. Goldsmith, Nancy Roistacher, and Michael S. Baum Capecitabine-Induced Coronary Vasospasm JCO August 1, 2008:3802-3804
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Figure 1. EKG during recovery