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CASE REPORT

# Coronary ectasia in a man on breast cancer therapy presenting with acute coronary syndrome

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## ABSTRACT

Limited data exist on the association between breast cancer treatments and coronary artery disease anatomy, particularly in males. We describe an unusual case of diffuse coronary ectasia in a man with breast cancer presenting with acute coronary syndrome (ACS). A 66-year-old man with breast cancer on paclitaxel, tamoxifen, and carboplatin chemotherapy regimen, presents with new onset chest pain. Electrocardiogram reveals anterolateral ST-segment depressions and elevated troponin I level. Emergent angiography revealed grossly ectatic coronary arteries with a total thrombotic occlusion of the mid right coronary artery. Serial intracoronary aspiration thrombectomy revealed fragments of red thrombus. Intracoronary tenectaplase was ultimately administered to restore perfusion. The patient clinically improved and warfarin was added to his cardiac regimen. Laboratory work up for connective tissue disease was negative. Although paclitaxel has been implicated in coronary artery neointima and media proliferation in the setting of drug-eluting stents, we believe our case is the first to describe massive coronary ectasia with significant thrombus burden requiring complex coronary intervention and thrombolysis in the setting of breast cancer therapy in a man.

**Key words:** Acute coronary syndrome, breast cancer, coronary ectasia, paclitaxel, percutaneous coronary intervention

## LEARNING OBJECTIVE

Coronary ectasia can contribute to complicated presentation and management of ACS with regards to both primary percutaneous coronary intervention and anticoagulation needs in long-term management. This is a complicated case of a patient with both coronary ectasia and history of breast cancer and systemic adjuvant paclitaxel chemotherapy which may represent a coincidental finding given a paucity of evidence for increased coronary artery disease risk in other patients undergoing this therapy.

## INTRODUCTION

Breast cancer in males is a rare entity, for which the treatment consists of multidrug chemotherapies. Coronary artery disease remains the most common cause of death in the general population. While limited studies point to correlation between systemic chemotherapeutic use, cardiac ischemia, and thromboembolic disease, there is limited data to attribute coronary artery disease to breast cancer treatment in men and so may be coincidental to this already complex case. We report diffuse coronary ectasia and complicated intervention in a middle-age man with breast cancer presenting with acute coronary syndrome (ACS).

## CASE REPORT

A 66-year-old Caucasian male presents to the emergency department complaining of new onset chest pain and

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increasing left arm tingling for the past 12 h. He endorsed nausea without emesis and denied any associated shortness of breath. The patient has a history of hypertension and stage 2 invasive ductal carcinoma of the left breast. At the time of diagnosis, he was treated with mastectomy 14 months before presentation, followed by adjuvant chemotherapy for 18 weeks with paclitaxel Cremophor EL (Taxol), tamoxifen, and carboplatin. He is a nonsmoker and denied use of illicit drugs. There is no report of premature heart disease in his family. On initial cardiac examination, patient was hypertensive to 160/90 mmHg, heart rate 95 beats per minute, undisplaced point of maximal impulse, normal heart sounds without gallop or murmurs.

A12-lead electrocardiogram (EKG) revealed anterolateral ST-segment depression of 1.5 mm consistent with myocardial ischemia [Figure 1]. Limited bedside echocardiogram revealed a mildly reduced ejection fraction (40–45%) and possible new basal to mid inferior regional wall motion abnormality. Initial troponin I level was elevated to 7.17 ng/ml. Due to the above findings and patient's continued chest pain, the decision was made to proceed with emergent cardiac angiography.

Coronary angiography revealed a grossly ectatic left anterior descending artery with diffuse moderate coronary artery disease but without significant or discrete stenosis throughout its branches [Figure 2a]. The right coronary artery (RCA) displayed 70% proximal stenosis followed by an ectatic segment measuring 7.5 mm, followed by a 100% occlusion at the mid RCA with total cessation of contrast dye flow [Figure 2b].

Immediate intervention was initiated to restore blood flow to the RCA. Multiple initial attempts to pass through the thrombus with support wires were unsuccessful, with distal passage finally gained by Luge™ wire (Boston Scientific, Marlborough, MA, USA) [Figure 2c]. An Export® intracoronary aspiration thrombectomy catheter (Medtronic, Minneapolis, MN, USA) was then used serially to remove fragments of an organized red thrombus multiple times [Figure 2d]. However, only partial flow was restored, and despite the use in combination with intracoronary Integrilin® (eptifibatide) significant occlusion persisted. An ectatic distal RCA system was better visualized, with the widest diameter measuring 11 mm [Figure 2e]. Thereafter, a 3.5 mm × 20 mm Quantum Apex coronary dilation balloon (Boston Scientific) was taken from the posterior descending artery (PDA) to distal RCA with minimal restoration of anterograde flow. Finally, tenectaplase 2.5 mg was prepared and slowly administered in an intracoronary fashion through the Export® catheter selectively to the distal RCA with 50–70% resolution of thrombus and improved flow to the PDA [Figure 2f].

EKG at this point showed resolution of ST-segment depression and patient reported reduction of chest pain symptoms. As determined by the interventional cardiologist, given the complexity of intervention to this point it was felt that the patient was at high risk for potential coronary

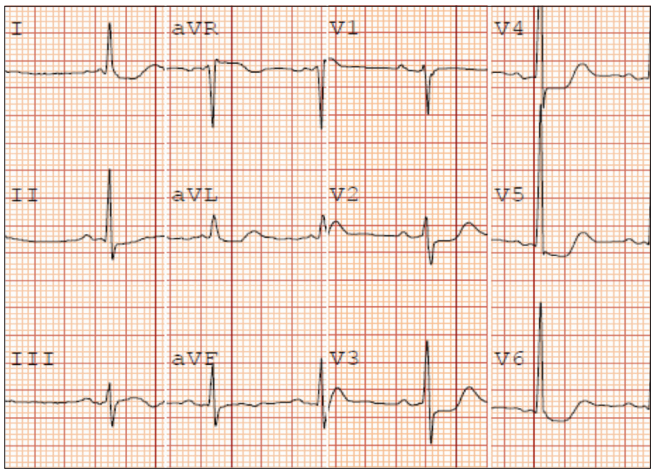


**Figure 1:** Presenting 12-lead electrocardiogram showing lateral ST segment depression suggestive of myocardial ischemia

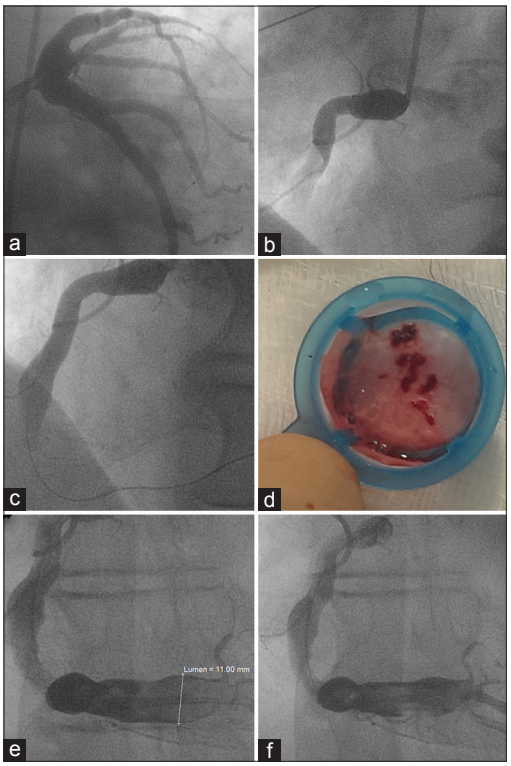


**Figure 2:** Left coronary angiogram with proximal ectasia (a), right coronary artery angiogram showing complete occlusion at the proximal segment (b), percutaneous intervention to right coronary artery with Luge™ wire (Boston Scientific, Marlborough, MA, USA) (c), aspirated thrombus burden (d), ectatic distal right coronary artery (e), final right coronary artery angiogram image postpercutaneous intervention. (f) full right coronary artery post intervention

perforation with limited potential benefit, and procedure was ended. Use of intravascular ultrasound was considered but ultimately not utilized due to the complexity and time of the intervention. The patient was closely monitored in the cardiac care unit, and discharged home 2 days later on optimal medical management, with the addition of warfarin. Of note, a serum immunofluorescence assay to evaluate for underlying connective tissue vasculitides, including antinuclear antibodies, anti-neutrophil cytoplasmic antibody (ANCA) (cytoplasmic-ANCA, perinuclear-ANCA), myeloperoxidase, and serine protease-3, was negative and a magnetic resonance imaging of the abdomen revealed normal aortic diameter.

## DISCUSSION

Coronary artery ectasia (CAE) is an uncommon finding represented by artery expansion 1.5 times the diameter of adjacent healthy vessel.[1] It is considered a variant of coronary artery disease and has been attributed to congenital malformation, vasculitides, and rarely posttraumatically during percutaneous intervention.[1,2] CAE appears to be correlated vascular disease, with considerably elevated rates of CAE reported among patients with aortic and pulmonary artery aneurysm.[3] Pathophysiology of CAE is not completely understood, but has been described to involve inflammatory infiltration of the intima and media of coronary vasculature, as well as reduction in proliferation of arterial smooth muscle cells, leading to progressive loss of organized structure and ensuing dilatation.[2,4] Matrix metalloproteinases are thought to play a considerable role in the breakdown of vessel connective tissue leading to dilation and progressive increase in wall stress.[1] Segmental dilatation results in reduced coronary flow rates, increased wall stress, and increased risk of thrombotic events.[4] Paclitaxel is a mitotic inhibitor that acts by stabilization of cellular microtubules' breakdown during cell division, and is approved for use in chemotherapy to treat lung, ovarian, and breast cancers.[5]

Paclitaxel eluding stents (PES) are used with good effect to reduce restenosis following percutaneous angioplasty in the setting of coronary artery disease but have also been implicated in increased rates of coronary artery aneurysm.[6] The etiology of stent-associated aneurysm is thought to be multifactorial involving a combination of mechanical vessel wall trauma, chemotherapeutic induced hypersensitivity vasculitis and subsequent weakening of vessel walls dilatation.[7] PESs have been shown to exhibit a dose dependent cytotoxic effect on coronary neointima and media, without consistent inflammatory involvement as well as a reduction in human arterial smooth muscle cell proliferation.[8]

Systemic paclitaxel dose relationship to coronary artery aneurysm or ectasia has not been examined widely upon review of current literature. Paclitaxel is poorly soluble in water and is combined with Cremophor EL (Taxol) for systemic chemotherapy to utilize albumin binding. As a result Taxol exhibits combined dose dependent and independent pharmacokinetics and therefore specific tissue effects may be more difficult to delineate.[9] Furthermore, review of literature shows that most studies into the cardiovascular side effects of Taxol chemotherapy for breast cancer examine these impacts only in female subjects. Cardiac complications including conduction disturbance, arrhythmia and cardiac ischemia have been implicated in female patients undergoing systemic Taxol therapy, partially attributable to the Cremophor EL component.[10,11]

Currently, no clear consensus exists regarding chronic anti-thrombotic management of CAE with or without ACS; however, low-dose warfarin was reported to be beneficial for thrombosis prevention within female breast cancer patients with CAE.[2,12,13] Clear recommendations likewise are lacking regarding screening for other vascular disease among patients with CAE.[3] Recent reports outline suggested management of CAE in ACS, however, the combination of breast cancer and use of chemotherapy in our male patient create a unique setting, for which acute or chronic therapeutic decisions remain challenging.

## CONCLUSION

CAE management represents a complex and under examined modality, especially in the uncommon presentation with ACS. Acute management in patients with CAE requires careful consideration of conservative and invasive approach and weighing of risk and benefit, collectively tailored into the individual patient's clinical scenario. Research is lacking regarding incidence of CAE in cancer populations treated with various anti-neoplastic therapies and the potential cause-effect relationship. While a few reports exist implicating increased cardiac ischemia and thromboembolic events in women undergoing chemotherapy, no clear elevated risk of CAD is shown and so the concomitant use of chemotherapy in this case may be coincidental. Early detection may prevent precipitation of ACS and/or thromboembolic events and better outcomes in these patients.

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### Conflicts of interest
There are no conflicts of interest.

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