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## Images in Cardiology

# 5-Fluorouracil–Induced Coronary Vasospasm

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A 64-year-old woman with newly diagnosed anal squamous cell carcinoma presented to the emergency department with sudden-onset chest pain experienced while receiving chemotherapy and radiation. She had no medical history. She had no cardiac risk factors and was taking no regular medications.

On presentation, her blood pressure was 155/111 mm Hg, heart rate was 78 beats/min, respiratory rate was 20 breaths/min, and oxygen saturation was 100% on room air. Cardiac examination results were unremarkable. Serial troponin T levels were 34 and 45 ng/L (normal < 14 ng/L). Electrolytes, including potassium, were within normal limits.

Electrocardiography showed hyperacute T waves in the anterolateral leads followed by marked ST elevation in leads I, II, and V2-V6 (Fig. 1). A 15-lead electrocardiogram immediately thereafter had normalized. She received 160 mg of aspirin, 600 mg of clopidogrel, and 4000 units of unfractionated heparin. A code was activated within the emergency department at 7:25 PM, and, per institutional standards for aggressive management of ST-elevation myocardial infarction, she was referred for immediate cardiac catheterization. She underwent coronary angiography at 8:40 PM; this did not demonstrate evidence of plaque rupture, atherosclerosis, or vasculopathy.

However, throughout the night, she continued to have intermittent episodes of chest pain that increased in intensity by morning (Fig. 2). Upon further examination, she was found to be wearing a pharmacotherapy pump providing 5-fluorouracil (5-FU) through continuous subcutaneous infusion. The pump, concealed in a well-disguised pack, had not been noted during the previous night's emergency management. The infusion was stopped, and a nitroglycerin patch was provided, relieving her chest pain almost immediately. A diagnosis of likely 5-FU–induced coronary vasospasm was made.

Cardiotoxicity is a well-known and serious consequence of 5-FU therapy that can mimic acute coronary syndrome.[1] There are multiple proposed mechanisms for 5-FU–induced cardiotoxicity, including coronary vasospasm causing myocardial ischemia. The estimated frequency of symptomatic 5-FU cardiotoxicity is reported as 1.2% to 4.3%,[2] but this risk is increased with continuous infusions compared with bolus therapy.[3] Recognition, symptomatic management, and consideration of alternative chemotherapy agents in such cases require dialogue between cardiologists and oncologists for optimal care of the patient. This case highlights the importance of recognizing the cardiotoxic effects of certain chemotherapeutic agents in the acute setting and treating symptomatically once the clinical diagnosis is made.

## Disclosures

The authors have no conflicts of interest to disclose.

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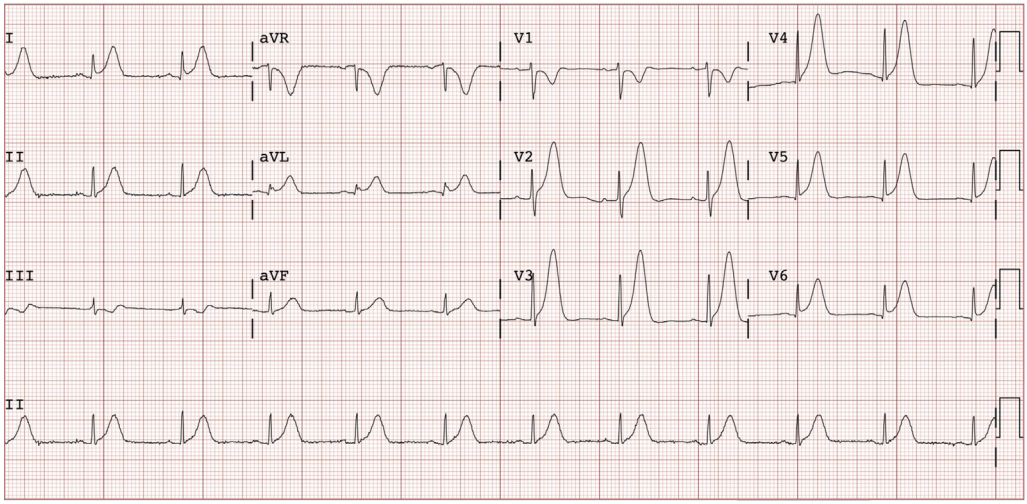


**Figure 1.** Progression of hyperacute T waves with anterolateral ST-elevation (V2-V6, I, II, aVL). Code ST-segment myocardial infarction activated.

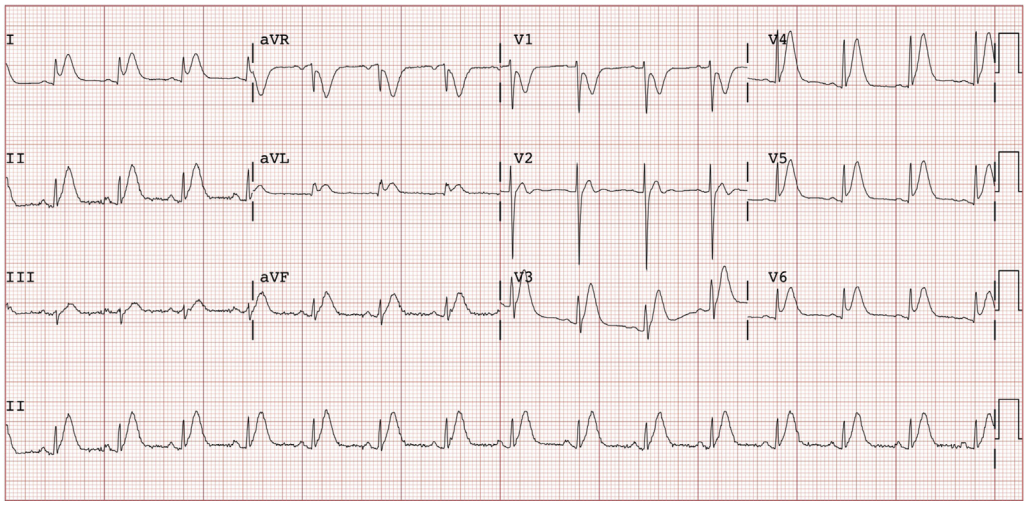


**Figure 2.** Recurrence of chest pain with accompanying anterolateral ST-elevation (V3-V6, I, II, aVL).