American Journal of Case Reports

e-ISSN 1941-5923
© Am J Case Rep, 2026; 27: e955231
DOI: 10.12659/AJCR.955231

Received: 2026.08.28
Accepted: 2026.10.27
Available online: 2026.11.05
Published: 2026.XX.XX

Suspected Drug-Associated Coronary Vasospasm With Transient ST-Segment Elevation During Multi-Agent Chemotherapy in a Patient With Gastric Cancer: A Case Report

Authors' Contribution:
Study Design A
Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
Literature Search F
Funds Collection G

ABCDE Michael Mohseni
BCEF Richard Courtney
BCDEF Saundra A. Jackson
ADEF Charles Graham

Department of Emergency Medicine, Mayo Clinic, Jacksonville, FL, USA

Corresponding Author: Michael Mohseni, Mayo Clinic, 4500 San Pablo Rd, Jacksonville, FL 32224, USA
Phone: (678) 517-2144, Fax: (904) 956-0034, e-mail: mohseni.michael@mayo.edu
Financial support: None declared
Conflict of interest: None declared

APPROVED GALLEY PROOF

Patient: Male, 71-year-old
Final Diagnosis: Vasospasm • vasospastic angina
Symptoms: Chest pain
Clinical Procedure: —
Specialty: Cardiology
Objective: Unusual clinical course
Background: Fluorouracil (5-FU) can induce coronary vasospasm that mimics ST-segment elevation myocardial infarction. During multi-agent chemotherapy, attribution to a single agent is difficult. This case illustrates diagnostic uncertainty when transient ST-segment elevation resolves with vasodilators and coronary evaluation is deferred.
Case Report: A 71-year-old man with locally advanced gastric adenocarcinoma presented with intermittent chest pain 2 days after starting cycle 1 of 5-FU, oxaliplatin, docetaxel, and durvalumab. The initial electrocardiogram showed mild lateral ST-segment depression. Approximately 2 hours later, severe chest pain developed with ST-segment elevation in V2-V6 and II, III, and aVF. Pain and ST-segment elevation resolved within approximately 45 minutes of intravenous diltiazem and nitroglycerin, and planned cardiac catheterization was not performed. High-sensitivity troponin values were 27, 44, and 55 ng/L on Day 0 and 16 ng/L on Day 5; no intervening measurements were obtained, and the peak was not captured. Echocardiography showed an ejection fraction of 57% without wall motion abnormalities. Computed tomography pulmonary angiography excluded pulmonary embolism. Coronary angiography, coronary computed tomography angiography, stress testing, and cardiac magnetic resonance imaging were not performed. No chest pain recurred during approximately 1 week of follow-up on isosorbide mononitrate and amlodipine.
Conclusions: Transient ST-segment elevation during multi-agent chemotherapy can reflect drug-associated coronary vasospasm. However, in the present case, obstructive coronary disease, thrombotic acute coronary syndrome, and immune checkpoint inhibitor myocarditis were not excluded, and no single agent could be implicated. Resolution with vasodilators did not establish the diagnosis. Deferring coronary evaluation was a case-specific decision, not a recommended approach.
Keywords: Cardio-Oncology • Cardiotoxicity • Coronary Vasospasm • Fluorouracil • Myocardial Infarction • Myocardial Ischemia • Antineoplastic Combined Chemotherapy Protocols
Full-text PDF: https://www.amjcaserep.com/abstract/index/idArt/955231

3629 1 3 24

Publisher's note: All claims expressed in this article are solely those of the authors and do not necessarily represent those of their affiliated organizations, or those of the publisher, the editors and the reviewers. Any product that may be evaluated in this article, or claim that may be made by its manufacturer, is not guaranteed or endorsed by the publisher

This work is licensed under Creative Commons Attribution-NonCommercial-NoDerivatives 4.0 International (CC BY-NC-ND 4.0)

Indexed in: [PMC] [PubMed] [Emerging Sources Citation Index (ESCI)] [Web of Science by Clarivate]

APPROVED GALLEY PROOF

Introduction

Fluorouracil (5-FU) is one of the most widely used chemotherapy agents for solid tumors [1,2]. While its therapeutic value is clear, the reported incidence of symptomatic cardiotoxicity with this drug varies widely: 0% to 20% with 5-FU and 3% to 35% with capecitabine in a systematic review, with larger studies suggesting 1.2% to 4.3% for 5-FU [3]. The risk appears to be higher with continuous infusions rather than bolus administration, with most cardiac events occurring during the first or second cycle [3,4]. The clinical spectrum ranges from asymptomatic electrocardiographic changes to angina, myocardial infarction, myopericarditis, heart failure, arrhythmia, and sudden cardiac death [4,5]. Coronary vasospasm is the most well-characterized mechanism, attributed to endothelial dysfunction with decreased nitric oxide release and elevated endothelin-1 levels [5-7].

Patients can present with ST-segment elevations or depressions mimicking acute coronary syndrome (ACS) despite normal coronaries on cardiac catheterization [5], creating a diagnostic dilemma in the emergency department (ED). Discontinuing the offending agent and initiating coronary vasodilators, along with nitrates and calcium channel blockers, remains the mainstay of treatment in the acute setting [8]. The dilemma is compounded in multi-agent regimens, in which oxaliplatin, taxanes, and immune checkpoint inhibitors have each been linked to coronary vasospasm, myocardial ischemia, or myocarditis [9-11], so that an ischemic event soon after treatment initiation may not be attributable to a single drug. Distinguishing suspected vasospasm from obstructive coronary disease with plaque rupture, myocardial infarction with non-obstructive coronary arteries, and myocardial injury such as myocarditis ordinarily requires further testing, each modality of which answers a different question and has limits. Coronary angiography defines coronary anatomy and can document spasm, but it is invasive; coronary computed tomography angiography (CTA) can exclude obstructive disease but cannot demonstrate vasospasm; stress testing can provoke ischemia but does not define anatomy; and cardiac magnetic resonance imaging (MRI) can identify myocarditis or infarction but does not assess the coronary arteries [12,13].

We report a case of a patient with gastric cancer who developed transient multi-territory ST-segment elevation 2 days after starting a regimen of 5-FU, oxaliplatin, docetaxel, and durvalumab, which resolved with vasodilator therapy and was not followed by coronary evaluation. The aim of this report is to illustrate the diagnostic uncertainty of attributing transient ST-segment elevation to chemotherapy without coronary evaluation, and the interpretive limits of a favorable response to vasodilator therapy.

Case Report

A 71-year-old man presented to the ED with chief concern of intermittent chest pain. He reported that the symptoms began the morning of the ED visit and worsened upon ambulation to his oncology appointment. He was recently diagnosed with locally advanced gastric carcinoma and was started on a continuous chemotherapy infusion 2 days prior (Day -2; Day 0 is the day of ED presentation). This regimen included 5-FU 5000 mg, oxaliplatin 180 mg, docetaxel 100 mg, and durvalumab 1500 mg, which corresponds in its components to FLOT (5-FU, leucovorin, oxaliplatin, docetaxel) plus durvalumab, but in our patient's case leucovorin was not administered in this first cycle of his chemotherapy. His chemotherapy infusion was scheduled for 46 hours, and he had presented to the clinic setting to have the infusion discontinued when he raised the concern of chest discomfort and was directed to the ED. Rash, pruritus, bronchospasm, or hypotension were not reported. He endorsed radiation of his chest pain to the left arm with associated shortness of breath and diaphoresis. Interestingly, he reported that the chest pain would occur sporadically and that between episodes he was asymptomatic. There was 1 reported episode of emesis, but he denied any fevers, chills, diarrhea, or other recent infectious history. The patient also did not report any history of bowel obstruction or pulmonary embolism. His medical history was significant, however, for chronic obstructive pulmonary disease (COPD), peptic ulcer disease, and tobacco abuse history. No prior cardiac history was documented; however, the patient did have a 46 pack-year smoking history with cessation 8 years earlier. The patient's previous lipid profile and hemoglobin A1c in the outpatient setting were normal. Family history of coronary artery disease and serum magnesium were not available in the records reviewed. A pre-treatment baseline ECG from 3 weeks prior showed normal sinus rhythm without ST-T changes. Oral medications pertinent to this emergency presentation included famotidine 20 mg twice daily, ondansetron 4 mg as needed, prochlorperazine 10 mg as needed, and oxycodone 5 mg as needed. He also used budesonide-formoterol 160-4.5 mcg/actuation inhaler 2 puffs 2 times daily. He denied any anticoagulant use. Surgical history was remarkable for remote umbilical hernia repair and exploratory laparotomy for his recent gastric carcinoma diagnosis.

Upon arrival to the ED, the patient's vital signs revealed a heart rate of 84, blood pressure of 133/82 mm Hg, temperature of 36.7 °C, respiratory rate of 16, and oxygen saturation of 96% on room air. Physical examination was initially reassuring without significant abnormalities, and the patient denied any ongoing chest discomfort. Breath sounds were normal, and cardiovascular examination revealed normal rate and regular rhythm. No lower extremity edema or calf asymmetry was noted. His initial ECG showed mild ST-segment depression

This work is licensed under Creative Commons Attribution-NonCommercial-NoDerivatives 4.0 International (CC BY-NC-ND 4.0)

Indexed in: [PMC] [PubMed] [Emerging Sources Citation Index (ESCI)] [Web of Science by Clarivate]

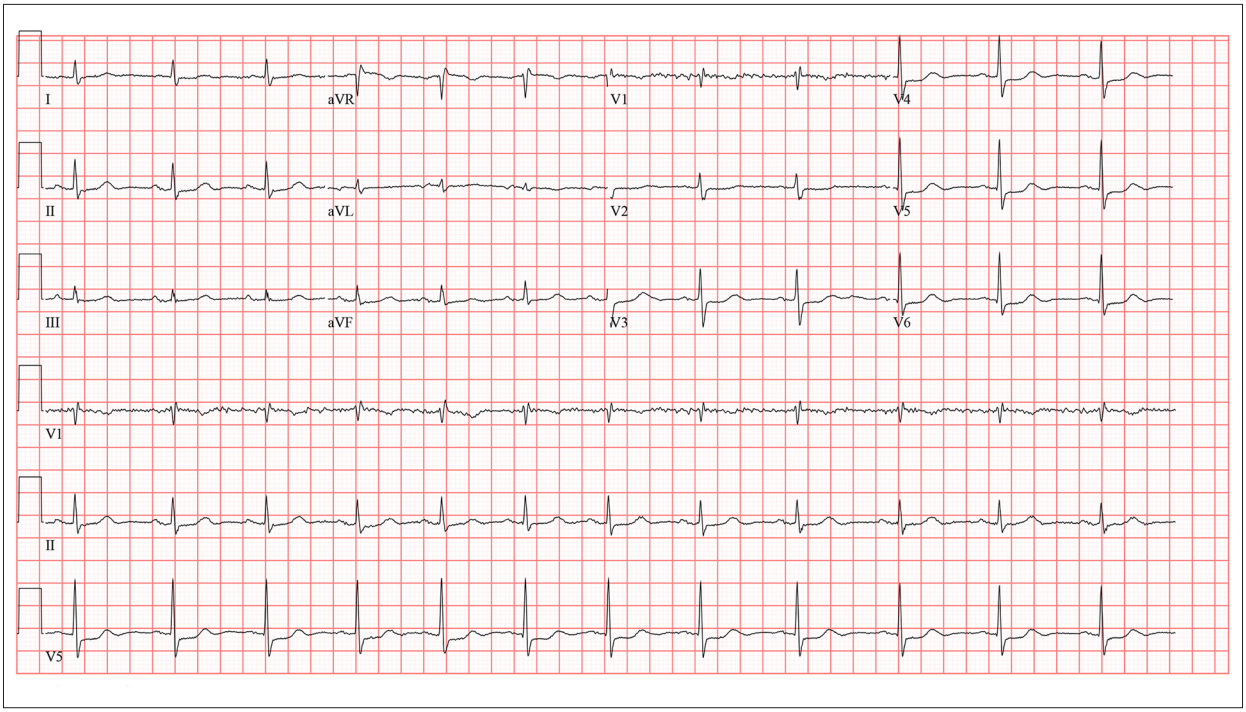


Figure 1. Patient's initial ECG on arrival to the ED while asymptomatic. Non-specific ST-segment depression noted in the lateral leads (V4-V6); documented heart rate on arrival 84 bpm. ST-segment depression was not quantified in the clinical documentation.

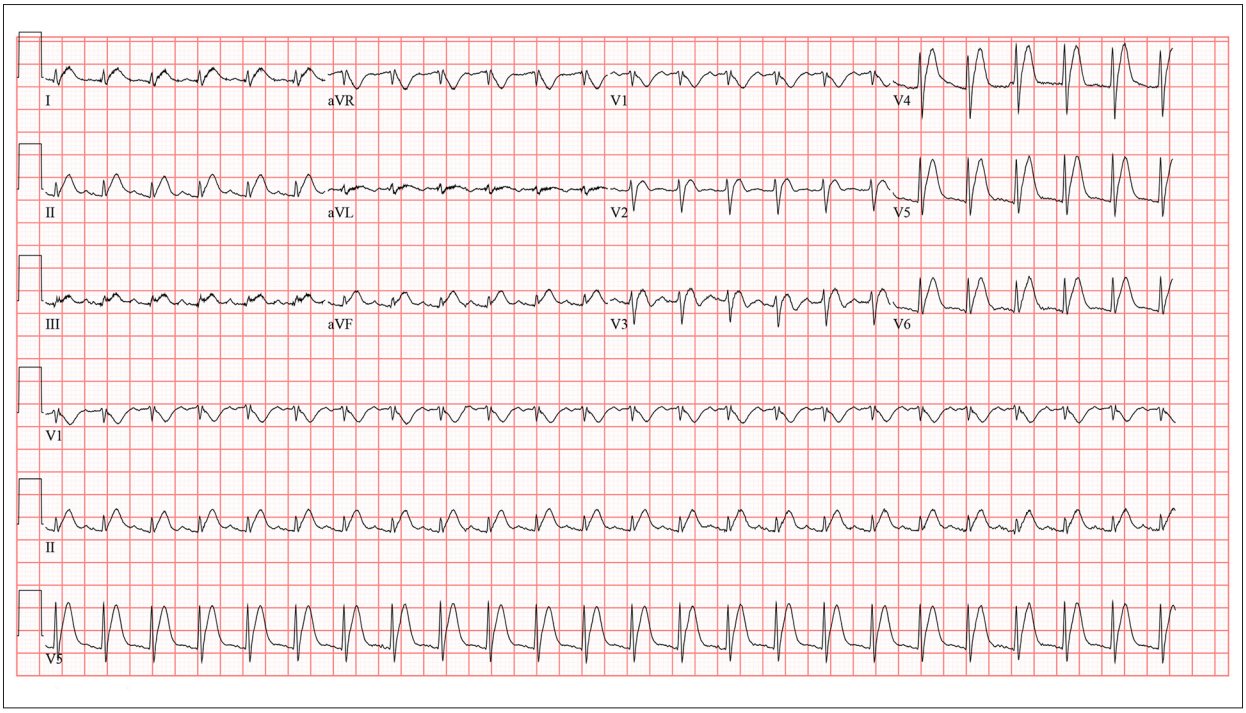


Figure 2. Symptomatic ECG obtained in the ED during the episode of severe chest pain, at a documented heart rate in the 130s to 140s, showing ST-segment elevation in leads V2-V6 and II, III, and aVF. The magnitude of ST deviation (mm) and the presence or absence of reciprocal changes was not reported. This ECG tracing documents lead distribution only; it does not establish the involved coronary territory or the mechanism.

APPROVED GALLEY PROOF

This work is licensed under Creative Commons Attribution-NonCommercial-NoDerivatives 4.0 International (CC BY-NC-ND 4.0)

Indexed in: [PMC] [PubMed] [Emerging Sources Citation Index (ESCI)] [Web of Science by Clarivate]

APPROVED GALLEY PROOF

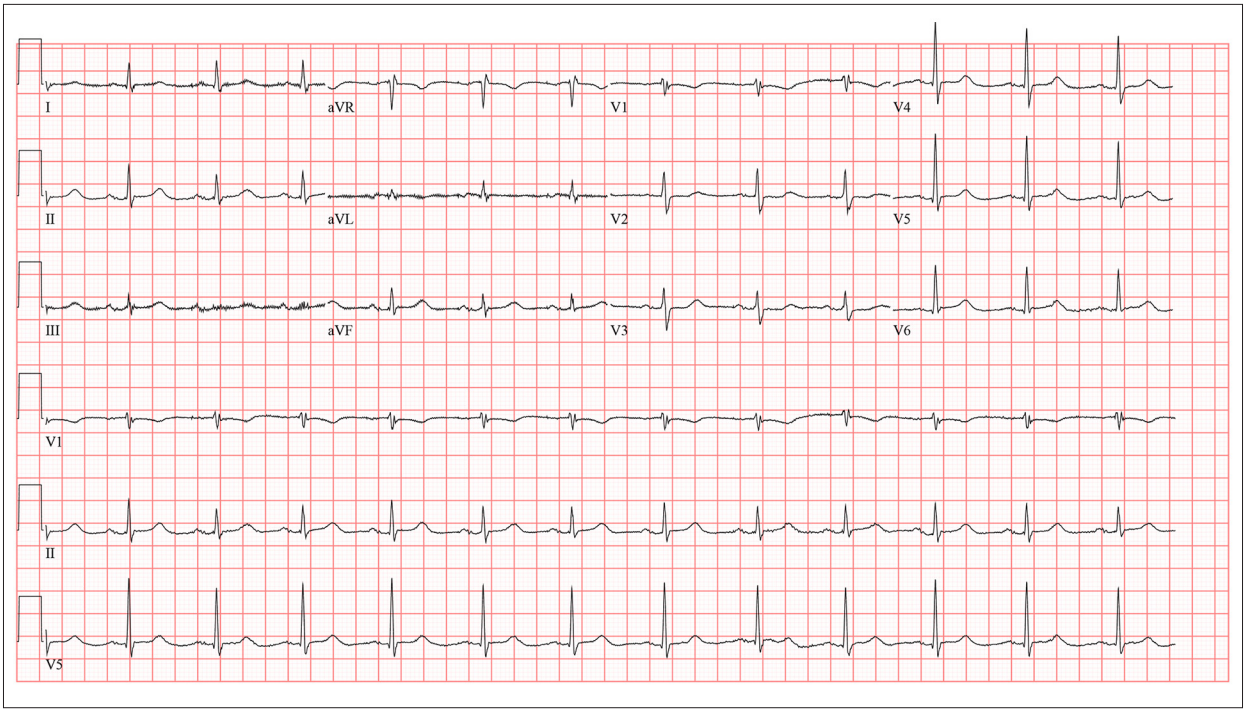


Figure 3. ECG obtained after initiation of diltiazem and nitroglycerin drips (approximately 45 minutes after infusions began). ST-segment abnormalities have resolved completely.

in the lateral leads (V4-V6; not quantified) (**Figure 1**). Initial laboratory evaluation revealed normal white blood cell count of 8.7×10^9/L, normal hemoglobin of 14.9 g/dL, normal sodium of 138 mmol/L, normal potassium of 4.0 mmol/L, normal creatinine of 0.90 mg/dL, elevated N-terminal pro B-type natriuretic peptide of 843 pg/mL, and high-sensitivity troponin of 27 ng/L drawn at 16: 10 on the day of presentation (99th percentile upper reference limit ≤ 15 ng/L; assay platform not recorded). Chest X-ray was interpreted as hyperinflated lungs without acute process, consistent with his history of COPD.

Approximately 2 hours into his ED course, the patient developed an episode of severe chest pain and became ill-appearing with diaphoresis. He became tachycardic with a pulse in the 130s to 140s. A repeat ECG (**Figure 2**) showed ST-segment elevation in leads V2-V6 and II, III, and aVF, as well as a heart rate in the 130s to 140s. The magnitude of ST deviation (in mm) and the presence of reciprocal changes were not reported. Rhythm designation was sinus tachycardia. No particular coronary territory was assigned to the ST-segment elevation. He was given aspirin 324 mg orally and nitroglycerin 0.4 mg sublingually. Given the constellation of findings on physical examination and serial ECGs, Cardiology was consulted.

The Cardiology team recommended initiation of diltiazem drip starting at 5mg/h intravenously as well as nitroglycerin drip starting at 5 mcg/min with titration to pain relief. These measures were initiated in concert with anticipation of proceeding for cardiac catheterization. Approximately 45 minutes into his infusions, however, he reported complete pain relief; a repeat ECG demonstrated resolution of the ST-segment abnormalities (**Figure 3**). The Cardiology team regarded these findings as suggestive of coronary vasospasm and elected not to proceed with cardiac catheterization; their documented reasoning, and how it relates to guideline recommendations, is addressed in the Discussion section below. The Cardiology team admitted the patient for serial troponin testing, telemetry monitoring for arrhythmias, and ongoing infusion of diltiazem and nitroglycerin.

The clinical timeline is outlined in **Table 1**. Hospital day numbering corresponds to calendar day numbering (hospital day 1 is Day 1; the day of ED arrival is Day 0). On hospital day 1, the patient underwent transthoracic echocardiography. This revealed normal left ventricular chamber size with an estimated ejection fraction of 57%. Grade 1/3 left ventricular diastolic dysfunction was reported, but there was normal right ventricular chamber size and function. No pericardial effusion, intracardiac mass, or thrombus was noted. No regional wall motion abnormalities were identified. Regarding high-sensitivity troponin laboratory testing, values rose from 27 ng/L at 16: 10 on the day of presentation to 44 ng/L at 18: 34, approximately 30 minutes after the episode of severe chest pain with ST-segment elevation, and to 55 ng/L at 22: 29. No troponin measurements were obtained between 22: 29 on Day 0 and Day 5, when a value obtained at outpatient follow-up (07: 57) was 16 ng/L. The peak concentration, its timing, and the time

This work is licensed under Creative Commons Attribution-NonCommercial-NoDerivatives 4.0 International (CC BY-NC-ND 4.0)

Indexed in: [PMC] [PubMed] [Emerging Sources Citation Index (ESCI)] [Web of Science by Clarivate]

Table 1. Clinical timeline of chemotherapy exposure, symptoms, electrocardiographic findings, treatment, biomarkers, imaging, and follow-up.

Time point	Event
Day -2, exposure details	Continuous infusion chemotherapy initiated for locally advanced gastric adenocarcinoma. Regimen: 5-FU, oxaliplatin, docetaxel, and durvalumab (components consistent with FLOT plus durvalumab); leucovorin: not given. Treatment intent (neoadjuvant versus other): not documented. Cycle number: one. Doses: 5-FU 5000 mg, oxaliplatin 180 mg, docetaxel 100 mg, and durvalumab 1500 mg. Chemotherapy continuous infusion with 46-hour duration (with plan to discontinue at oncology appointment 2 days later)
Day 0, morning	Onset of intermittent chest pain, worse on ambulation to a scheduled oncology appointment when chemotherapy infusion was discontinued
Day 0, ED arrival	Asymptomatic on arrival. HR 84 bpm, BP 133/82 mm Hg, RR 16, SpO_2 96% on room air, temperature 36.7 °C. Examination unremarkable. ECG: mild ST-segment depression in lateral leads V4-V6 (**Figure 1**); depression not quantified; baseline ECG from 3 weeks prior showed normal sinus rhythm without ST-T changes. Chest radiograph: hyperinflation without acute process
Day 0, 16: 10	High-sensitivity troponin 27 ng/L (99th percentile upper reference limit ≤ 15 ng/L; assay platform not recorded). NT-proBNP 843 pg/mL. Creatinine 0.90 mg/dL, hemoglobin 14.9 g/dL, WBC 8.7 × 10^9/L
Day 0, ≈ 2 h into ED course	Severe chest pain with diaphoresis and ill appearance; HR 130s-140s. ECG: ST-segment elevation in leads V2-V6 and II, III, aVF (**Figure 2**); ST deviation in mm and reciprocal changes not recorded. Aspirin 324 mg orally and nitroglycerin 0.4 mg sublingually administered. Cardiology consulted
Day 0, following consultation	Intravenous diltiazem 5 mg/h and intravenous nitroglycerin 5 mcg/min initiated, titrated to pain relief, in anticipation of cardiac catheterization
Day 0, ≈ 45 min after infusions start	Complete resolution of chest pain; ECG demonstrated full resolution of ST-segment abnormalities (**Figure 3**). Cardiac catheterization aborted (see Discussion). Admitted for serial troponins, telemetry, and continued infusions
Day 0, 18: 34	High-sensitivity troponin 44 ng/L
Day 0, 22: 29	High-sensitivity troponin 55 ng/L
Day 0, 22: 29 to Day 5	No troponin measurements were reported; the peak was not captured
Hospital day 1 (Day 1)	Transthoracic echocardiography: normal left ventricular chamber size, ejection fraction 57%, grade 1/3 diastolic dysfunction, normal right ventricular size and function, no regional wall motion abnormalities, no pericardial effusion, mass, or thrombus. CT pulmonary angiography negative for pulmonary embolism; emphysematous changes only
Hospital day 2 (Day 2)	Mild bradycardia (50s bpm)—diltiazem discontinued. Nitroglycerin weaned and discontinued. Isosorbide mononitrate 30 mg extended-release daily started. Discharged
Day 5, 07: 57	High-sensitivity troponin 16 ng/L at outpatient laboratory follow-up
≈ 1 week after discharge (≈ Day 9)	Cardio-Oncology clinic: no recurrent chest discomfort. Amlodipine 5 mg daily added for suspected drug-associated coronary vasospasm. Isosorbide mononitrate continued with plan to titrate toward 90 mg before chemotherapy resumption (planned without 5-FU; subsequent regimen not documented). No further 5-FU planned. No additional cardiac investigation pursued
Not performed or not documented	Coronary angiography, coronary CT angiography, stress testing, and cardiac MRI: not performed. Formal causality assessment: not performed. Rash, pruritus, bronchospasm, hypotension: not reported. Family history and serum magnesium: not available

Day 0 denotes the day of Emergency Department presentation. Day -2 denotes the day of chemotherapy initiation. Hospital day n corresponds to Day n. ED, Emergency Department; ECG — electrocardiogram; HR, heart rate; BP, blood pressure; RR, respiratory rate; NT-proBNP, N-terminal pro B-type natriuretic peptide; WBC, white blood cell count; CT, computed tomography; 5-FU, fluorouracil; FLOT, fluorouracil, leucovorin, oxaliplatin, docetaxel; MRI, magnetic resonance imaging; HbA1c, hemoglobin A1c.

APPROVED GALLEY PROOF

This work is licensed under Creative Commons Attribution-NonCommercial-NoDerivatives 4.0 International (CC BY-NC-ND 4.0)

Indexed in: [PMC] [PubMed] [Emerging Sources Citation Index (ESCI)] [Web of Science by Clarivate]

APPROVED GALLEY PROOF

course of the decline are therefore unknown. Pulmonary embolism was also ruled out with computed tomography chest angiogram of the pulmonary arteries with intravenous contrast; no other abnormalities were seen on this imaging other than known emphysematous changes. The full sequence of chemotherapy exposure, symptom onset, serial electrocardiographic findings, vasodilator treatment, troponin measurements, echocardiography, computed tomography pulmonary angiography, discharge, and follow-up again is summarized in **Table 1**.

On hospital day 2, the patient developed mild bradycardia with a pulse rate in the 50s; thus, diltiazem drip was discontinued. Furthermore, given lack of recurrent chest pain, the nitroglycerin drip was weaned and discontinued. He was started on isosorbide mononitrate 30 mg extended-release tablet once daily by mouth. He was deemed stable for discharge from the hospital with close follow-up in the outpatient setting with the Cardio-Oncology team. The only new cardiac medication at discharge was isosorbide mononitrate 30 mg extended-release once daily. In the clinic setting approximately 1 week later (approximately Day 9), the patient was doing well without recurrent chest discomfort. In coordination with his Oncology team, no further administration of 5-FU was planned at that time. He was additionally started on low-dose amlodipine 5 mg daily by mouth for suspected drug-associated coronary vasospasm. He was continued on isosorbide mononitrate 30 mg extended-release tablet once daily with goals to titrate upward, targeting 90 mg before resumption of chemotherapy, which was planned without 5-FU; the specific subsequent regimen was not documented in the records available to us. No additional investigative cardiac work-up was pursued in the outpatient setting. Follow-up was limited to this visit.

Discussion

Fluoropyrimidines, most often recognized as 5-FU and its oral counterpart capecitabine, are commonly used chemotherapy agents in the treatment of gastrointestinal malignancies, as was the case with our patient's presentation. Cardiotoxicity has been defined as symptoms of chest pain, dyspnea, or palpitations, in conjunction with ECG changes, hemodynamic abnormalities, documented decreased ejection fraction, or, less commonly, elevated cardiac biomarkers, which have been found in a small percentage (12%) of patients [8,14]. The vast majority of these complications have developed during continuous infusion of 5-FU versus bolus dosing and within the first 12 to 72 hours of treatment initiation [8,14]. Interestingly, an existing diagnosis of coronary artery disease did not seem to factor into the incidence of adverse events [8].

There have been several proposed mechanisms of the exact pathophysiology behind these events, the most widely accepted being that of coronary vasospasm, with some attention also given to the medication's negative effects on both smooth muscle and endothelial cells [8,14]. The mainstay of treatment focuses on immediate discontinuation of the fluoropyrimidine and vasodilation with the use of nitroglycerin and calcium channel blocker infusions [14,15]. Uridine triacetate, approved by the U.S. Food and Drug Administration in 2015, is indicated for the emergency treatment of fluorouracil or capecitabine overdose and for early-onset, severe or life-threatening fluoropyrimidine toxicity presenting within 96 hours of administration; it is not an approved therapy for coronary vasospasm and is distinct from, rather than a substitute for, nitrate and calcium channel blocker therapy in the management of vasospastic ischemia [14]. Levosimendan, a calcium sensitizer, has separately been described as adjunctive therapy in fluoropyrimidine-associated cardiotoxicity in which myocardial dysfunction predominates [16]. As in the case with our patient, ECG changes resolved, as did his discomfort while on infusions of nitroglycerin and diltiazem, which were both initiated in the ED. This was clinically consistent with possible drug-associated coronary vasospasm but is not specific for it.

Attribution in this case is limited by multi-agent exposure. The regimen administered (5-FU, oxaliplatin, docetaxel, and durvalumab) corresponds in its components to FLOT plus durvalumab, a perioperative regimen for resectable gastric and gastroesophageal junction adenocarcinoma that the U.S. Food and Drug Administration approved in November 2025 on the basis of the MATTERHORN trial [17,18]. Emergency clinicians are therefore likely to encounter patients receiving a fluoropyrimidine, a platinum agent, a taxane, and an immune checkpoint inhibitor concurrently. In the FLOT schedule, 5-FU is given as a 24-hour or 46-hour infusion [19]; the infusion duration of 46 hours used in our patient yields an interval of approximately 2 days between treatment initiation and symptom onset.

Several features are compatible with a fluoropyrimidine contribution: onset within the first days of treatment, when most fluoropyrimidine-associated events occur [8,14], and clinical response to a nitrate and a calcium channel blocker. None of these features is specific. Oxaliplatin has been associated with coronary vasospasm [9]; docetaxel with rare ischemic events, with proposed mechanisms that include vasospasm, endothelial dysfunction, hypersensitivity, and thrombosis [10]; and immune checkpoint inhibitors with myocarditis [11]. Hypersensitivity-type reactions to oxaliplatin or docetaxel are typically described as occurring during or soon after infusion [9], and immune checkpoint inhibitor myocarditis is generally reported weeks to months after treatment initiation [11]; the onset of symptoms about 2 days after initiation is therefore less typical of these patterns. However, these considerations do not exclude any of the agents. Rash, pruritus, bronchospasm, and hypotension were not reported (breath sounds

This work is licensed under Creative Commons Attribution-NonCommercial-NoDerivatives 4.0 International (CC BY-NC-ND 4.0)

Indexed in: [PMC] [PubMed] [Emerging Sources Citation Index (ESCI)] [Web of Science by Clarivate]

were normal and blood pressure was 133/82 mm Hg on arrival; blood pressure during the severe episode of chest pain was similar). Because these features were not systematically documented, their absence cannot be taken as evidence against a hypersensitivity contribution. No formal causality assessment (eg, the Naranjo algorithm [20] or the WHO-UMC system) was performed. Given the concurrent exposures, the absent coronary evaluation, and the lack of agent-specific dechallenge or rechallenge information, we would expect any such assessment to support no more than a possible association. The attribution of this event to drug-associated vasospasm, and to 5-FU in particular, therefore remains unconfirmed.

The Cardiology team's decision not to proceed with catheterization was a case-specific judgment made under diagnostic uncertainty. As documented, it was based on complete resolution of both chest pain and ST-segment elevation within approximately 45 minutes of initiating intravenous vasodilator therapy, the multi-territory rather than single-territory distribution of the ST-segment elevation, the absence of hemodynamic instability or ventricular arrhythmia, and the close temporal relationship to a chemotherapy regimen that included 5-FU begun 2 days earlier. These considerations were weighed against features that would ordinarily favor invasive evaluation, including the patient's age, tobacco use history, active malignancy with its attendant prothrombotic risk, and a high-sensitivity troponin value above the upper reference limit on presentation. The response to vasodilator therapy was treated as supportive of coronary vasospasm rather than diagnostic of it, and plaque rupture with transient thrombosis was not excluded by that response alone.

This approach departed from the pathway recommended by authoritative guidance. Current ACS guidelines direct patients with ST-segment elevation and ischemic symptoms toward emergency coronary angiography with reperfusion when indicated [21,22]; the ACC/AHA guideline is focused primarily on type 1 myocardial infarction [21]. The 2022 European Society of Cardiology cardio-oncology guideline recommends an invasive strategy for patients with cancer who have ST-segment elevation myocardial infarction or high-risk non-ST-segment elevation ACS and a life expectancy of at least 6 months (our patient's life expectancy was not documented) and advises withholding a suspected causative anticancer agent during the acute phase, with multidisciplinary discussion before any retreatment [12]. To our knowledge, neither document endorses deferring coronary evaluation because ST-segment elevation resolves with vasodilator therapy, and the management observed here is not presented as a generally supported conservative-management model. The patient's hemodynamic stability, rapid resolution, and recently initiated multi-agent regimen explain the team's decision but do not validate it. Guideline-concordant alternatives would have included coronary angiography, which can also document spasm, or, after stabilization, coronary CTA, stress testing, or cardiac MRI [12,14].

The diagnostic limitations of this case are consolidated here. Our patient did not undergo further investigation with cardiac catheterization, and obstructive coronary artery disease was not anatomically excluded per se. Given the absence of objective testing such as coronary angiography, coronary computed tomography angiography, or stress testing, occult coronary disease or other causes of myocardial injury could not be excluded in our patient. Several alternatives warrant explicit consideration in this presentation. Obstructive coronary artery disease with plaque rupture and transient thrombosis, followed by spontaneous or pharmacologically-assisted lysis, could produce a similar pattern of transient ST-segment elevation with troponin release, and remains the principal unexcluded diagnosis. A myocardial infarction with non-obstructive coronary arteries (MINOCA)-like presentation is likewise compatible with the observed findings, and coronary vasospasm is itself among the recognized mechanisms of MINOCA, although MINOCA by definition requires angiographic documentation of non-obstructive coronary arteries, which was not obtained [23]. Cancer-associated hypercoagulability may have provided an additional substrate for transient coronary thrombosis in this patient. Myocarditis was considered less likely given the absence of a preceding febrile or infectious prodrome, the abrupt onset and complete resolution of ST-segment elevation within approximately 45 minutes of vasodilator therapy, and the low-level troponin values that were measured; however, immune checkpoint inhibitor-associated myocarditis merits consideration in any patient receiving durvalumab [11], and this diagnosis was not excluded, as neither cardiac magnetic resonance imaging nor endomyocardial biopsy was obtained. Takotsubo syndrome was not supported by the echocardiographic findings, which demonstrated preserved ejection fraction without regional wall motion abnormality; however, echocardiography was obtained on hospital day 1, after symptom resolution, and cannot exclude transient dysfunction at the time of the event. None of these possibilities was formally excluded, and each is presented here as an unexcluded alternative rather than an established finding. Additionally, his response to vasodilators was supportive, but not diagnostic, of coronary vasospasm: nitrate responsiveness is one component of, but does not by itself establish, the diagnosis of vasospastic angina [13], and neither the multi-territory distribution of ST-segment elevation nor the observed troponin course establishes a vasospastic mechanism, proves reversible ischemia, or indicates a low-risk process. The ECG evidence is also limited: ST deviation was not measured in millimeters, reciprocal changes were not reported, and the tracing in **Figure 2** was obtained at a heart rate in the 130s to 140s, so we cannot state whether published ECG criteria for vasospastic angina (ST-segment elevation of at least 0.1 mV in 2

APPROVED GALLEY PROOF

This work is licensed under Creative Commons Attribution-NonCommercial-NoDerivatives 4.0 International (CC BY-NC-ND 4.0)

Indexed in: [PMC] [PubMed] [Emerging Sources Citation Index (ESCI)] [Web of Science by Clarivate]

APPROVED GALLEY PROOF

contiguous leads) were met [13]. Troponin sampling was similarly limited: no measurements are reported between 22: 29 on Day 0 and Day 5, the peak was not captured, and a rising pattern above the 99th percentile in the setting of ischemic symptoms and transient ST-segment elevation does not allow myocardial infarction to be excluded [23]. Furthermore, no formal causality assessment was performed. Dissimilar to our patient's course of care, the literature has suggested low-risk patients be screened with coronary computed tomography angiography, and exercise stress testing occurs following symptom resolution [14]. No such post-resolution testing was performed in this patient, and no additional cardiac investigation was pursued in the outpatient setting. This omission limits the exclusion of underlying coronary disease and reduces the certainty with which the presentation can be attributed to drug-associated coronary vasospasm. In addition, follow-up in this report extends only to approximately 1 week after discharge. That interval is insufficient to establish freedom from recurrent symptoms, recurrent ischemia, or later cardiac events, and the reported outcome should be interpreted as applying only to the observed period.

Compared with previously reported cases of fluoropyrimidine-associated coronary vasospasm, the present case is consistent in its timing, occurring within the first several days of starting a regimen that included 5-FU, and in the prompt resolution of both symptoms and ST-segment elevation with nitrate and calcium channel blocker therapy. It differs in that coronary angiography was not performed, and the course was uncomplicated by arrhythmia, in contrast to the report of Hasnie et al, in which 5-FU-associated vasospasm (in a patient who received FOLFOX, a multi-agent regimen) precipitated ST-segment elevation myocardial infarction and polymorphic ventricular tachycardia in a younger patient [15]. Ultimately, it was decided that 5-FU therapy would be discontinued and that an alternative chemotherapy regimen be used for the patient in our case presentation (the specific regimen was not available to us). There have been reports of continued use of 5-FU following cardiotoxicity in those patients where alternative therapies were not deemed viable to the survivability of the cancer in question [14]. In these instances, a rechallenge or further use of 5-FU despite adverse events linked to cardiotoxicity, was attempted with successful outcomes [24]. Research and case reports have pointed to pre-treatment with calcium channel blockers and long-acting nitrates prior to chemotherapy, with change from an infusion protocol delivery of 5-FU to bolus chemotherapy [15]. Other recommendations included the use of sublingual nitroglycerin and short-acting calcium channel blockers during infusion and the use of post-treatment calcium channel blockade [14]. These observations from the literature are provided for context and are not management recommendations derived from this single unconfirmed case.

Conclusions

This case illustrates a diagnostic dilemma. Transient ST-segment elevation arose during multi-agent chemotherapy that included 5-FU, oxaliplatin, docetaxel, and durvalumab, resolved with nitrate and calcium channel blocker therapy, and was attributed by the treating team to possible drug-associated coronary vasospasm. Because coronary evaluation was not performed, the troponin peak was not captured, and no formal causality assessment was done, the diagnosis remains unconfirmed; obstructive coronary disease, thrombotic ACS, and immune checkpoint inhibitor myocarditis were not excluded; and no individual agent can be implicated. For emergency medicine clinicians, the lesson is that resolution of ST-segment elevation with vasodilators does not by itself establish a benign mechanism, and that early cardiology and cardio-oncology involvement is needed to decide how the diagnosis can be established safely. The deferral of invasive coronary evaluation in this patient was a case-specific decision under uncertainty and should not be taken as a recommended management approach.

Institution Where Work Was Done

Mayo Clinic, Jacksonville, FL, USA.

Patient Consent

Appropriate patient consent/permission was obtained.

Declaration of Figures' Authenticity

All figures submitted have been created by the authors who confirm that the images are original with no duplication and have not been previously published in whole or in part.

References:

1. Grem JL. 5-Fluorouracil: Forty-plus and still ticking. A review of its preclinical and clinical development. Invest New Drugs. 2000;18(4):299-313
2. Sara JD, Kaur J, Khodadadi R, et al. 5-fluorouracil and cardiotoxicity: A review. Ther Adv Med Oncol. 2018;10:1758835918780140
3. Polk A, Vaage-Nilsen M, Vistisen K, Nielsen DL. Cardiotoxicity in cancer patients treated with 5-fluorouracil or capecitabine: A systematic review of incidence, manifestations and predisposing factors. Cancer Treat Rev. 2013;39(8):974-84
4. Layoun ME, Wickramasinghe CD, Peralta MV, Yang EH. Fluoropyrimidine-induced cardiotoxicity: Manifestations, mechanisms, and management. Curr Oncol Rep. 2016;18(6):35
5. More LA, Lane S, Asnani A. 5-FU cardiotoxicity: Vasospasm, myocarditis, and sudden death. Curr Cardiol Rep. 2021;23(3):17

This work is licensed under Creative Commons Attribution-NonCommercial-NoDerivatives 4.0 International (CC BY-NC-ND 4.0)

Indexed in: [PMC] [PubMed] [Emerging Sources Citation Index (ESCI)] [Web of Science by Clarivate]

6. Mosseri M, Fingert HJ, Varticovski L, et al. In vitro evidence that myocardial ischemia resulting from 5-fluorouracil chemotherapy is due to protein kinase C-mediated vasoconstriction of vascular smooth muscle. Cancer Res. 1993;53(13):3028-33
7. Thyss A, Gaspard MH, Marsault R, et al. Very high endothelin plasma levels in patients with 5-FU cardiotoxicity. Ann Oncol. 1992;3(1):88
8. Saif MW, Shah MM, Shah AR. Fluoropyrimidine-associated cardiotoxicity: Revisited. Expert Opin Drug Saf. 2009;8(2):191-202
9. Samol J, Waterston A. Oxaliplatin-induced coronary artery spasm: First report of an important side-effect. BMJ Case Rep. 2009;2009:bcr06.2008.0334
10. Tong F, Fan Z, Huang H, et al. A patient with triple-negative breast cancer developed acute myocardial infarction after docetaxel chemotherapy: Case report and literature review. Front Cardiovasc Med. 2026;13:1791074
11. Mahmood SS, Fradley MG, Cohen JV, et al. Myocarditis in patients treated with immune checkpoint inhibitors. J Am Coll Cardiol. 2018;71(16):1755-64
12. Lyon AR, Lopez-Fernandez T, Couch LS, et al. 2022 ESC Guidelines on cardio-oncology developed in collaboration with the European Hematology Association (EHA), the European Society for Therapeutic Radiology and Oncology (ESTRO) and the International Cardio-Oncology Society (IC-OS). Eur Heart J. 2022;43(41):4229-361
13. Beltrame JF, Crea F, Kaski JC, et al. International standardization of diagnostic criteria for vasospastic angina. Eur Heart J. 2017;38(33):2565-68
14. Kanduri J, More LA, Godishala A, Asnani A. Fluoropyrimidine-associated cardiotoxicity. Cardiol Clinic. 2019;37:399-405
15. Hasnie A, Neace Z, Ellison K, et al. Case Report: Coronary vasospasm precipitating STEMI and polymorphic ventricular tachycardia—A case of cardiotoxicity from 5-FU based chemotherapy in a 41-year-old woman. Front Cardiovasc Med. 2025;12:1594338
16. Wu-Chin C, Zuniga-Orlich C, Salas-Segura J, et al. Acute cardiotoxicity induced by 5-fluorouracil successfully treated with levosimendan: A case report. Cureus. 2025;17(8):e89996
17. Janjigian YY, Al-Batran SE, Wainberg ZA, et al. Perioperative durvalumab in gastric and gastroesophageal junction cancer. N Engl J Med. 2025;393(3):217-30
18. U.S. Food and Drug Administration. FDA approves durvalumab for resectable gastric or gastroesophageal junction adenocarcinoma. November 25, 2025. https://www.fda.gov/drugs/resources-information-approved-drugs/fda-approves-durvalumab-resectable-gastric-or-gastroesophageal-junction-adenocarcinoma
19. Al-Batran SE, Homann N, Pauligk C, et al. Perioperative chemotherapy with fluorouracil plus leucovorin, oxaliplatin, and docetaxel versus fluorouracil or capecitabine plus cisplatin and epirubicin for locally advanced, resectable gastric or gastro-oesophageal junction adenocarcinoma (FLOT4): A randomised, phase 2/3 trial. Lancet. 2019;393(10184):1948-57
20. Naranjo CA, Busto U, Sellers EM, et al. A method for estimating the probability of adverse drug reactions. Clin Pharmacol Ther. 1981;30(2):239-45
21. Rao SV, O'Donoghue ML, Ruel M, et al. 2025 ACC/AHA/ACEP/NAEMSP/SCAI guideline for the management of patients with acute coronary syndromes: A report of the American College of Cardiology/American Heart Association Joint Committee on Clinical Practice Guidelines. Circulation. 2025;151(13):e771-862
22. Byrne RA, Rossello X, Coughlan JJ, et al. 2023 ESC Guidelines for the management of acute coronary syndromes. Eur Heart J. 2023;44(38):3720-826
23. Thygesen K, Alpert JS, Jaffe AS, et al. Fourth universal definition of myocardial infarction (2018). Circulation. 2018;138(20):e618-51
24. Classen SC, Ky B, O'Quinn R, et al. Fluoropyrimidine-induced cardiac toxicity: Challenging the current paradigm. J Gastrointest Oncol. 2017;8:970-79

APPROVED GALLEY PROOF

This work is licensed under Creative Commons Attribution-NonCommercial-NoDerivatives 4.0 International (CC BY-NC-ND 4.0)

Indexed in: [PMC] [PubMed] [Emerging Sources Citation Index (ESCI)] [Web of Science by Clarivate]