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Acute Infarct of Myocardium without Coronary Arterial Obstructive Disease in a Patient with Colon Cancer: A Case Report

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ABSTRACT

We report a 40-year-old female with colon cancer, in its first session of treatment with capecitabine/oxaliplatin, who presented an event of acute coronary syndrome type myocardial acute infarction with ST inferior elevation in the first 30 days after initiation of adjuvant treatment, angiographically without lesions and echocardiographically presented impaired ventricular function. This case report illustrates myocardial damage induced by antimetabolites in an acute manner.

KEYWORDS: Colon cancer, Acute myocardial infarction, capecitabine/oxaliplatin, Antimetabolite therapy.

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INTRODUCTION

Fluoropyridines (5- Fluorouracil) and their oral pro-drug, capacitabine, are the main chemotherapeutic agents used in solid tumors of glandular and squamous origin, involving the gastrointestinal tract, are part of the standard treatment of advanced colo-rectal cancer, may cause cardiotoxicity manifested by chest pain secondary to vasospasm and even acute myocardial infarction, shock, Takotsubo syndrome, arrhythmias, cardiogenic shock, pericarditis and even sudden cardiac death (1,2). 5-FU is an analog of pyrimidine that inhibits thymidylate synthase (TS), an enzyme involved in DNA replication.

Timeline

Enero 3, 2023	Diagnosis of colon cancer
Enero 23, 2023	Sigmoid colon resection and colostomy.
Marzo 22, 2023	Initiation of chemotherapy.
Abril 20 2023	Acute coronary syndrome
Abril 28 2023	Hospital Discharge

CASE REPORT

The patient was 40 years old, with a history of colon cancer diagnosed on January 23, 2023, exploratory laparotomy was performed, with sigmoid colon resection and colostomy, adjuvant treatment began, on March 22, 2023, based on XELOX scheme (capecitabine/oxaliplatin). On April 20, 2023, he presented chest pain, oppressive, with pain duration of 30 minutes, radiated to left arm accompanied by adrenergic discharge, being classified as acute coronary syndrome type AMI, electrocardiographically with poor progression of the first vector in the anteroseptal face, negative t-wave from v2 to v6, positive ST segment difference in DII, DIII, AVF and V4-V6 **(figure I)** biochemically with troponin I 115 ng/mL, BNP 1370 pg/mL, so he was thrombolized upon admission to the emergency room in a 2nd level unit, with 8 hours of ischemia, with tenecteplase 60 mg, without meeting indirect reperfusion criteria, is sent to our unit where rescue PCI was performed, documenting coronary arteries without obstructive lesions **(figure II)**, normal flow, left ventricle with anterolateral acinesia apical and inferior hypokinesia, normal basal portions, echocardiography documented LVEF (left ventricular ejection fraction) of 35% and lower wall hypokinesia in its three segments and basal and middle anteroseptal. He was with cardiogenic shock that required management with inotropic and vasopressors, presenting

improvement until he managed to hospital discharge to continue with cardiac rehabilitation and management of oncological pathology.

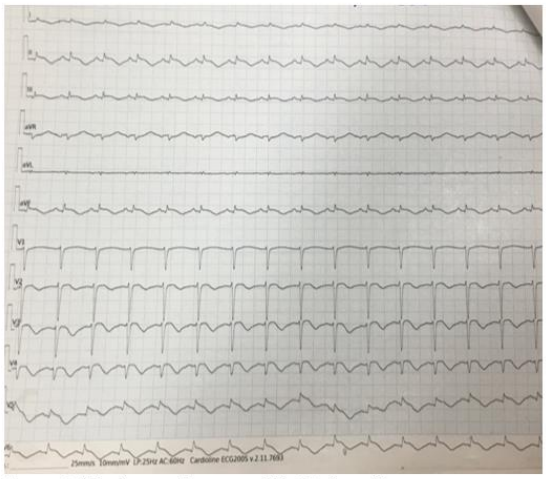

Figure I. Electrocardiogram with ST elevation.

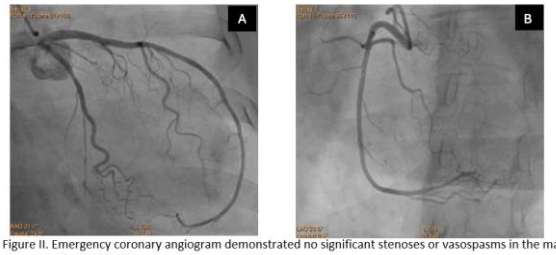

Figure II. Emergency coronary angiogram demonstrated no significant stenoses or vasospasms in the major coronary arteries. A, left coronary artery; B, right coronary artery.

DISCUSSION

Anti-metabolites such as capecitabine can cause cardiotoxicity such as ischemia and myocardial infarction and arrhythmias, the mechanism of action is cardiomyocyte toxicity (myocarditis) and coronary spasm. However, the damage is associated with 72 hours of the first cycle. Capacitabine functions as S-phase antimetabolites and promotes genomic instability by inducing breaks in the double helix of DNA and a single helix of DNA, as well as by interfering with DNA synthesis, repair and elongation. Capacitabine is metabolized to 5-FU in a series of reactions involving cytidine deaminase enzymes and thymidine phosphorylase, which are expressed in tumor cells, thus targeting the cancerous tissue rather than the tissue that is normally divided. There are other theories related to 5-FU-induced toxicity, such as endothelial damage and thrombus formation, the theory of oxidative stress, vasospasm, in which it can be observed that there is no single mechanism of cardiotoxicity, so it could be considered multifactorial (3). The incidence of fluoropyrimidine-associated cardiotoxicity is estimated at 7% to 18% of exposed patients (4). The addition of oxaliplatin to capacitabine improves disease-free survival in patients with stage III colon cancer. In cancer patients, it is important to identify cardiovascular risk factors, evaluate primary and secondary prevention, to avoid chemotherapy-related complications (5). In a substudy of BleeMACS it was observed that at one year, mortality at one year from cardiovascular disease is 11.7% up to 50%, the risk of reinfarction and bleeding is up to 8.3%, Hazard ratio of 2.1 and 2.5, $P < 0.001$) the presence of cancer is an independent predictor of cardiovascular disease, so treatment should be temporarily discontinued, consider alternative oncological treatments, and an urgent multidisciplinary approach is indicated to personalize management, taking into account the state of the cancer, its prognosis and the patient's preferences for invasive management. Prophylactic treatment may be initiated in patients without coronary obstruction, as in this case, with prolonged-acting nitrates and calcium channel blockers (6.7) .These types of patients who underwent percutaneous coronary interventionism are considered high-risk (8).

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