A STRANGE CASE OF CARDIOTOXICITY FROM ANTIBLASTIC TREATMENT: A HIGH PRICE TO PAY

S. Samperi, C. Laura, P. Trambaiolo, A. D’Ambrosi, M. Mustilli, S. Romano, and A. Granatelli
Università degli Studi De L’Aquila - Ospedale Sandro Pertini, Roma

Introduction: Cardiovascular diseases and cancer represent the two main public health problems in the west. Targeted oncological therapies extend the average life expectancy of patients, but also cause acute or chronic cardiovascular toxicity.

Case Report: A 58-year-old woman without cardiovascular risk factors comes to the Emergency Department for oppressive chest pain, associated with vomiting and tachycardia. Two days before admission, adjuvant chemotherapy treatment with 5-fluorouracil (5-FU) and oxaliplatin had been administered to the patient for a rectal cancer detected the previous year. At blood tests D-Dimer was negative, the troponin was 0.44 ng/ml [0.034 ng/ml], NTproBNP 8820 pg/ml [<125 pg/ml]. The electrocardiogram showed a mild but widespread elevation in the anterior lead and the transthoracic echocardiography (TTE) revealed a reduction in
Ejection fraction (EF 20%) with global hypokinesia and akinesia of the apex extending to the middle segments. At the angiography the coronary arteries were free from significant stenoses. Hospitalization in cardiac intensive care unit was complicated by episodes of torsades de pointes de genrating into ventricular fibrillation (VF), treated with 3 DC-shocks; the patient was intubated and mechanically ventilated for two days. During the hospitalization, heart failure and antiarrhythmic therapy was started, with improvement in the patient’s ventricular systolic function and symptoms. The patient rejected cardiac magnetic resonance imaging (cardiac MRI). On discharge, after ten days, TTE revealed an EF of 40%, filling pressures reduced, NTproBNP and troponins values normalised.

Discussion: 5-FU is the second most cardiotoxic oncology drug, after anthracyclines. Patients who start therapy with fluoropyrimidines may present chest pain associated with findings suggestive of coronary occlusion, as well as biochemical evidence of myocardial injury in the absence of disease on coronary angiography. The pathogenetic mechanism is probably related both to coronary vasospasm and a direct myocardial toxicity of the drug. However severe left ventricular dysfunction remains a behavior not described in the literature with this type of antiblastics. Cardiac MRI would have been helpful in clarifying the etiology of the toxicity.