Imaging and Histopathologic Evidence of Cardiotoxicity after Chemotherapy


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Letter to Editor

Cardiotoxicity is a well-established complication of anticancer therapy. The increased risk of cardiovascular disease and cardiovascular disease-related death in cancer survivors is multifactorial, including cardiotoxic effects of chemotherapy, comorbidities and harmful lifestyle habits. Cardiovascular adverse effects can range from coronary heart disease, pericardial disease, valvular disease, myocarditis, thromboembolism, and arrhythmias; the risk of cardiotoxicity varies according to the type and dose of chemotherapy. Heart failure incidence rates associated with the commonly-prescribed chemotherapy agents include 0.14% to 48% for anthracyclines. For high-dose cyclophosphamides the risk ranges from 7% to 28%, for trastuzumab is 1% and for tyrosine kinase inhibitors is 8% to 12.5%.

The management of patients with cancer under chemotherapy must be multidisciplinary in order to cure them but with a lower risk of cardiotoxicity. (Figure 1). Female 65-years-old, with history of colorectal cancer (stage IIIB) since September 2015, who received chemotherapy with capecitabine and oxaliplatin with remission in October 2017. She presented recurrence in December 2017, so she started with oxaliplatin and 5-fluoracil (5-FU). Minutes after the 5-FU infusion she began with typical angina at rest. The initial ECG (Figure 1A) showed elevation of the J point of 0.5 mm with hyperacute T waves in all precordial leads. The ECG performed 4 hrs later (Figure 1B) with elevation of the ST segment in V2-V3 of 0.5 mm. She underwent primary percutaneous coronary intervention, but no angiographically significant coronary lesions were found, except for vasospasm in the left anterior descending coronary artery (Figure 1C). The cardiotoxic effect of some chemotherapeutic agents that cause myocardial ischemia is due to coronary vasospasm, endothelial injury and acute thrombosis, as well as changes in lipid metabolism due to premature atherosclerosis.

(Figure 2). Female 65-years-old with cervical cancer in 1996 and intestinal Hodgkin lymphoma in 2012, treated surgically and 6 cycles of CHOP, in remission. She had 2-year history of heart failure. On physical examination, tachycardia, pulse parus et tardus and aortic, mitral and tricuspid valvular murmurs were found. ECG showed atrial fibrillation and left ventricular hypertrophy and cardiomegaly in chest X-ray (Figure 2A). No coronary artery lesions were detected (Figure 2B).
Two and three-dimensional echocardiography revealed severe double mitral lesion (Figure 2C1 to 2, Clip 1), moderate double aortic lesion (Figure 2C1, Clip 2) and moderate tricuspid regurgitation (Figure 2C-3). She underwent double valvular replacement and 3 days later died due to uncontrolled bleeding. Histopathological findings demonstrated extensive plurivalvular thickening, fibrosis and dystrophic calcification (Figure 2D-1 to 3). Neither neovascularity nor Aschoff bodies were found, which are specific for rheumatic carditis. We consider that chemotherapy was responsible for the plurivalvular affection in this patient.