

Cardiotoxicity: Signs and Symptoms

Evangeline Rose*

Department of Clinical Psychology, University of Calgary, Canada

Commentary

Cardiotoxicity is a case of dysfunction of cardiac electrophysiology or muscle injury. The heart becomes weak and ineffective in pumping and circulation. Cardiotoxicity may be caused by chemotherapy. Cardiac poisoning is a rare but potentially dangerous complication of large doses of chemotherapy and little is known about the incidence, severity and causes. High-dose cyclophosphamide-containing medications are often associated with cardiovascular toxicity and a reduction in decreased incidence over time. Dosage, regimens for use and simultaneous administration of other chemotherapeutic agents emerged as risk factors. Although cardiovascular toxicity is rarely associated with other cytotoxic drugs, the unexpected occurrence of severe cardiotoxicity is caused by low-dose conditioning drugs containing melphalan and fludarabine. The development of immune checkpoint inhibitors has revolutionized the treatment of stage cancer patients. But, serious side effects related to the immune system are often observed. Cardiac toxicity resulting from ICI treatment can range from elevated asymptomatic troponin-I to abnormal cardiac output and even to myocarditis. Although rare, myocarditis is a potentially fatal side effect of ICI treatment. The importance of high clinical suspicions, early diagnosis of myocarditis and early initiation of immunosuppressive therapy have been observed. The estimated number of heart tests for patients is limited, and patients with mild cardiac arrest may tolerate large doses of chemotherapy. Clinical examination, rest electrocardiography and dose adjustment in obese patients remains a pillar of prevention, with bidimensional echocardiography for patients with a history of anthracycline exposure. Strategies to reduce the long-term side effects of anthracycline administration in cardiac function are being investigated. Several widely used compounds of high doses of cyclophosphamide are not associated with an increased risk of cardiotoxicity than one high-dose cyclophosphamide agent. Cardiotoxicity is rarely reported in other cytotoxic drugs. In light of this

data, other strategies in addition to increasing dose limitations to reduce anthracycline cardiotoxic potential may be of interest. The effect of anthracycline exposure on cardiac function may be reduced by the use of cardioprotectors. Further decrease in the risk of cardiac toxicity may be achieved by pegylated liposomal anthracyclines, the pharmacokinetic which is characterized by a slow release of the drug to avoid high plasma concentrations. Pegylated liposomal doxorubicin showed similar efficacy with a much lower incidence of cardiovascular side effects compared to regular doxorubicin. Radiation exposure contributes to the risk of coronary heart disease. Therefore, previous radiation therapy in the left chest wall may represent a risk factor. In addition, radiation in the above areas appears to increase the cardiotoxicity of anthracyclines, although these two have different mechanisms of injury, patients with a history of both treatments potentially are at greater risk of chemotherapy-related cardiotoxicity. Adverse events related to the immune system are usually mild and usually improve with symptom management. Use of a Checkpoint inhibitor can lead to the development of adverse cardiotoxic events such as myocarditis, irregularities, cardiomyopathy and pericarditis. It is important for doctors to be aware of these adverse events because of the high risk of death. A number of basic cardiac function tests and regular monitoring confirms, especially for people who may be at high risk of developing significant clinical complications from immune-related cardiotoxicity, such as patients with pre-existing heart disease or autoimmune disease.

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Corresponding author: Evangeline Rose, Department of Clinical Psychology, University of Calgary, Canada; E-mail: evarose9498@uoc.ca

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