Cardiotoxicity-induced by antitumoral agents: prevention and treatment.

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Cardiotoxicity is a common complication of many antitumoral agents that may compromise clinical effectiveness of anticancer therapy, independently of the oncologic prognosis, and that may impact on the patient's survival and quality of life. Cardiotoxicity is a growing problem in the setting of clinical oncology, given the increasing number of long-term cancer survivors, the tendency to use more and more aggressive chemotherapy schedules (and consequently more effective), the continuous introduction in clinical practice of new antitumor agents with possible cardiotoxic properties, and combined treatments with interactive harmful effects on the heart.

Probably, cardiotoxicity is a unique and continuous phenomenon starting with myocardial cell injury, followed by progressive left ventricular ejection fraction (LVEF) decline that, if disregarded and not treated progressively leads to overt heart failure (HF). The main strategy for minimizing cardiotoxicity is early detection of high-risk patients and prompt prophylactic treatment. According to the current standard for monitoring cardiac function, cardiotoxicity is usually detected only when a functional impairment has already occurred, precluding any chance of its prevention. At present, we can detect cardiotoxicity at a preclinical phase, very much before the occurrence of HF symptoms, and before LVEF drops by measurement of cardiospecific biochemical markers or by Doppler myocardial and deformation imaging. The role of troponins in identifying subclinical cardiotoxicity and treatment with angiotensin-converting enzyme inhibitors, in order to prevent LVEF reduction is an effective strategy emerged in the last fifteen years. If cardiac dysfunction has already occurred, partial or complete LVEF recovery may still be achieved if cardiac dysfunction is detected early after the end of chemotherapy and HF treatment is promptly initiated.