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Opinion
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Role of Anthracycline Induce Cardiac Toxicity in Breast Cancer Patients

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Opinion

Breast cancer is the most common cancer among women counting about 125 per 100000 per year new case in United States and about 1.4 million new cases worldwide [1,2]. In the last twenty years early diagnosis, neoadjuvant and adjuvant systemic treatment that targeted to specific molecular targets have significantly reduced the mortality from breast cancer. However ,the increase in survival has allowed to observe the cardiotoxic effects of anticancer therapy and increase mortality from cardiovascular causes, resulting in a large literature where experts try to identify the correct management of critical problem.

Cardiotoxicity is potential complication of anticancer therapy; anthracycline therapy is a mainstay for breast cancer and other cancer, but it associated with rate of incidence heart failure more than five time the average, is a well-known problem in the treatment of cancer and limits the therapeutic use of this group of effective antineoplastic agents. Anthracycline are group of antibiotics that made remarkable advances in treatment of a wide variety of solid organ tumors and hematology malignancies, including leukemia, lymphoma, breast cancer, lung cancer, multiple myeloma, sarcoma [4,5]. Among all anthracycline, doxorubicin is the most commonly used anticancer drug [6-9]. Doxorubicin-induced cardiotoxicity may range from asymptomatic electrocardiographic (ECG) changes to decompensated cardiomyopathy which is characterized by decreased left ventricular ejection fraction (LVEF) [4].

The most common clinical presentation of cardiotoxicity is a dilatation-hypokinetic cardiomyopathy leading to heart failure [10]. The development of cardiotoxicity, even if asymptomatic, not only adversely affects the cardiac prognosis of the patient, but significantly limits the therapeutic possibilities in oncology when an additional anticancer treatment becomes necessary for recovery/relapse of cancer disease [11]. Anthracycline cardiac toxicity is presented by structural cardiomyocyte alteration and cell death (type 1 cardiotoxicity), it is generally not reversible and mediated at least in part by reactive oxygen spices (ROS) generated in iron -dependent chemical reactions. ROS led to the peroxidation of myocyte membranes and calcium influx into the intracellular space, which can ultimately lead to permanent myocyte damage [7,8].

The cardiotoxicity from anthracyclines can be acute, early or late. According to literature, there are many defections for cardiotoxicity based on change in LVEF. based on the previous findings, the American and European Society of Echocardiography Expert consensus defined cardiotoxicity related to cancer therapeutic as reduction of LVEF>10% to a value below 53% [9].

Pre-treatment assessment and cardiovascular risk factors evaluation, although no guidelines are available, it is common opinion that the first strategy to reduce and prevent chemotherapy -induced cardiotoxicity is an accurate analysis of pre-existing cardiovascular risk factors or subclinical cardiovascular damage and an assessment of the optimal type and cumulative dose of therapy [12].

According to literature, the main risk factors associated with anthracycline-induced cardiotoxicity are [12-15]:

i. Cumulative dose, the risk of anthracycline-induced cardiotoxicity is dose-dependent and increase with cumulative dose, for example, doxorubicin is associated with an incidence of congestive heart failure from 3% to5% with cumulative dose of 400mg/m2, from 7%to 26% at 55mg/m2, and from 18%to48 at 700mg/m2.the risk of inducing cardiac abnormalities including

the development of heart failure even years after treatment as result of anthracycline therapy has been recognized for a long time.

ii. Female sex

iii. Age >65years old

iv. Renal failure

v. Concomitant or previous radiation therapy involving the heart.

vi. Concomitant chemotherapy with alkylating or antimicrotubular agents or immune-and targeted therapies.

vii. Per-existing conditions such as cardiac disease associating increased wall stress arterial hypertension.

viii. Genetic factors

Risk assessment prior than treatment beginning should always include a clinical history collection, physical examination and measurement of vital signs. In the last decades, several studies have been conducted to detect the possible role of conventional congestive heart failure therapy in prevention and treatment of left ventricular dysfunction in cancer patients., Cardinale et al suggested than enalapril, an angiotensin -converting -enzyme (ACE) inhibitor could prevent late cardiotoxicity. Moreover, several studies suggested as possible protective role of beta-blockers in prevention of anthracycline-induced cardiotoxicity [3].

For patients per-exposed to anthracyclines and experienced asymptomatic sequalae, the international clinical guidelines suggest the inclusion of liposomal formulation to reduce the cardiac toxicity. In fact, the use of liposomal delivery is thought to have the same value as dexrazoxane in terms of being as cardio-protectant [9].

An adequate preliminary stratification of cardiotoxicity risk and early identification and treatment of subclinical cardiac damage may allow oncologists to avoid withdrawal of chemotherapy and cardiologists to improve the patient's prognosis avoiding irreversible cardiovascular dysfunction.

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