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The breast cancer, early disease: toxicity from therapy with epirubicin regimens - cardiac assessment and risk evaluation (BETTER-CARE) study: CMR with early gadolinium relative enhancement, but not high-sensitivity troponin T, predicts the risk of chronic anthracycline cardiotoxicity

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Background

A growing number of cancer patients are at risk from chronic anthracycline cardiotoxicity (cAC) as a result of improving cancer prognosis. Susceptibility is cumulative dose-related, but also idiosyncratic. At present there is no ideal test to identify those at risk: endomyocardial biopsy is inappropriate for routine monitoring; while serial measurement of LV ejection fraction (LVEF) only identifies cardiotoxicity after significant damage has been incurred. We hypothesised that risk of cAC could be determined from a combination of baseline factors and assessment of the response to the 1st anthracycline dose. Here we report the associations between cAC and myocardial insult after cycle 1: assessed by CMR and high-sensitivity troponin T (hsTnT).

Methods

Women due to receive anthracycline-based chemotherapy for early breast cancer were recruited to the BETTER-CARE study. Those with known cardiovascular disease were excluded. CMR was performed before chemotherapy and at follow-up (>1 year after the final anthracycline cycle, and >3 months after Trastuzumab). A subgroup was studied at baseline and on day-3 after the 1st cycle of anthracycline with CMR (early gadolinium relative enhancement, EGRE) and measurement of hsTnT (Roche Diagnostics). A 2nd subgroup had hsTnT measured after the final anthracycline cycle (before radiotherapy or trastuzumab). LVEF was measured by a single operator (PK) and EGRE by a 2nd, independent operator (GS). Chronic AC (cAC) was defined as a fall in absolute LVEF≥5% at follow-up.

Results

55/60 subjects assessed on day-3 completed follow-up (median 19 months); 51/55 had paired EGRE and 49/55 hsTnT data. 20% were in the cAC group (N=11). 58 subjects had hsTnT measurements after completing anthracycline treatment (median 21 days). No patients developed a rise in hsTnT after cycle 1. However, following the final cycle (median 4), 78% were hsTnT positive (≥0.004 μg/l). Post-treatment hsTnT was a poor discriminator of cAC (area under ROC curve of 0.51, p=0.95). The EGRE response was heterogeneous: median increase 11.6%, mean 23.8% (p<0.001). EGRE increased significantly more in those in the cAC group (p=0.02); area under ROC curve of 0.75 (p=0.01).

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Conclusions

This study shows that myocardial injury (elevated hsTnT) occurs in the majority of women treated with low-dose anthracyclines. However, hsTnT cannot discriminate between those who develop contractile dysfunction and those who do not. Myocardial inflammation can be detected after the 1st dose of anthracycline using CMR-EGRE, before hsTnT rises. Furthermore, the magnitude of EGRE response is greater in those who later develop contractile dysfunction due to cAC. Development of this, or related techniques, may lead to a superior means of monitoring the cardiotoxic effects of chemotherapy, both in clinical practice and interventional trials.

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