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3



Serial troponin measurement during anthracycline-containing chemotherapy may predict early cardiotoxicity

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Despite well-established cardiotoxicity profiles, anthracyclines are widely used in haematological malignancies. Despite the well known side effects, cardioprotection is not yet common practice. The European Society of Cardiology recommends a thorough assessment of baseline cardiac risk factors and using serial cardiac biomarkers to identify those at risk. Recent studies show that echocardiography and clinical features of heart failure are late findings of cardiotoxicity and fail to capture early changes. They also emphasise that patients started on cardioprotective medications on the basis of rising cardiac biomarkers have better long-term outcomes.

AIM

Our aim is to quantify early cardiotoxicity occurrence in patients receiving anthracycline-containing chemotherapy regimens. Detection of both clinical and subclinical cardiotoxicity using different parameters is necessary to identify at risk patients. We also wanted to observe the correlation between early cardiotoxicity and presence of cardiac risk factors prior to starting chemotherapy. From a total of 65 patients who received ABVD or RCHOP therapy, 83% had significant troponin rise.

Data showed progressive troponin rise in our cohort of patients with each cycle of chemotherapy; however, echocardiogram failed to detect any changes.

79% of the patients who had no cardiac risk factors and were under the age of 65 had significant troponin rise.

Pre existing cardiac risk factors did not seem to play a significant role in the degree of troponin rise.





Data demonstrated that majority of our patient cohort had a significant overall troponin rise. Whilst average troponin rose from each cycle, there was no correlation between the degree of troponin rise and presence of cardiac risk factors.

METHOD

This prospective study focused on patients receiving ABVD or R-CHOP chemotherapy from November 2016 to December 2018. Serial troponin measurements were taken at baseline and prior to each cycle. Echocardiography measurements were performed at baseline, at the end of the treatment and one-year post chemotherapy. Significant troponin rise was defined as a 50% rise in troponin from baseline. If this occurred patients were started on ramipril as cardioprotection. Patients' cardiac risk factors were analysed for correlation with troponin rise. Echocardiogram reports were used to detect any decline in left ventricular function.

CONCLUSIONS

The majority of patients in our study exhibited significant troponin rise regardless of baseline cardiac risk factors. Conversely, echocardiography didn't show an overall trend of cardiac compromise despite the significant troponin rises. This finding is in keeping with early myocyte damage detected by biomarkers before progression to overt cardiac dysfunction. Although limited by a small population size, our study supports starting an ACE inhibitor in patients receiving anthracycline regimens as a primary prevention strategy. Data collection is ongoing to determine the impact of early intervention on long term cardiac outcomes.

ACKNOWLEDGEMENT

REFERENCES

- Cardinale D, Sandri MT, Colombo A, et al. Prognostic value of troponin I in cardiac risk stratification of cancer patients undergoing high-dose chemotherapy. Circulation. 2004;109(22):2749–54.

- Zardavas et al. 'Role of Troponins I and T and N-Terminal Prohormone of Brain Natriuretic Peptide in Monitoring Cardiac Safety of Patients With Early-Stage Human Epidermal Growth Factor Receptor 2-Positive Breast Cancer Receiving Trastuzumab: A Herceptin Adjuvant Study Cardiac Marker Substudy.' Journal of Clinical Oncology. 2017 Mar 10;35(8):878-884. doi: 10.1200/JCO.2015.65.7916.

- Cardinale et al. 'Prevention of high dose chemotherapy induced cardiotoxicity in high risk patients by ACE inhibition' Circulation 2006; 114:2474-81. ESC 2016 Guidelines

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