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Letter to the editor

Trastuzumab and QT dispersion

Ozgur Tanriverdi[1], Deniz Cetin[2]

[1] Department of Medical Oncology, Ministry of Health Mugla State Hospital, Mugla University Research and Practice Center, Mugla, Turkey

[2] Department of Internal Medicine, Adnan Menderes University Faculty of Medicine, Aydin, Turkey

The article entitled "The acute effect of trastuzumab infusion on ECG parameters in metastatic breast cancer patients" written by Yavas et al. and published in one of the 2007 issues of your journal was quite interesting [1]. We consider that this article is important and interesting as it studies the adverse effects profile of targeted molecules. Therefore, we also wanted to emphasise some points. Trastuzumab has recently been an indispensable therapeutic agent in adjuvant and metastatic treatment modalities for breast cancer patients with over-expressed her2 receptor. The prevalence of congestive heart failure manifested by decreased left ventricular ejection fraction which is recognised to be reversible has been reported as 0.1–4.1% (adjuvant studies) and 7–27% (metastatic studies) in previous studies about this molecule [2, 3]. It was noted by Yavas et al. that although trastuzumab therapy is known to be associated with congestive heart failure, its arrhythmogenic potential has not been studied in detail [1]. The study by Yavas et al. investigated acute electrocardiographic changes due to trastuzumab infusion and attempted to show the potential arrhythmogenic effects of this molecule by QT dispersion. Unfortunately, we think that a small but important detail was overlooked in this study. We noticed that electrolyte imbalances were not mentioned in the excluding criterion of the study, and that this topic was also not included in the discussion. It is known that hypokalemia and hypocalcemia cause prolongation in the QT interval, whereas hyperkalemia and hypercalcemia cause a shortening. Some studies report that hypokalemia greatly affects QT dispersion, whereas hypocalcemia, hypercalcemia and hyperkalemia do not cause significant changes [4]. For this reason, the authors should have mentioned this situation in the method and discussion part of the article.

When we consider that the study sample consists of patients with metastatic stage, electrolyte imbalance can be assumed with a certain frequency in these patients. Electrolyte imbalances, which are among the reasons of non-cardiac QT dispersion, can affect the working group measurements. This situation may change all statistical analyses. Thus, we think that the authors should seek to respond the questions listed below: 1) Are the serum calcium, potassium and magnesium values of patients considered? 2) How many of the patients had bone metastases and how many of them were treated with bisphosphonates? 3) How many of the patients were cachectic or suffering from weight loss? Was hypoalbuminemia detected in these patients? Were the corrected calcium values for patients with hypoalbuminemia calculated?

We thank the authors for this compelling study based on the hypothesis that her2 receptor inhibition might cause a disorder of the normal heart cell signalling pathway. We will be awaiting the reply of authors with great interest.

Correspondence: *Ozgur Tanriverdi, MD, Department of Medical Oncology, Ministry of Health Mugla State Hospital, Mugla University Research and Practice Center, TR-4800 Mugla, ozgurtanriverdi@hotmail.com*

Reply to this Letter to the Editor:
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