Natriuretic peptide and cardiac troponin levels in doxorubicin-induced cardiotoxicity

To the Editor,

We read with a great interest the paper by Argun et al. (1) entitled "Cardioprotective effect of metformin against doxorubicin cardiotoxicity in rats'' published in the Anatolian Journal of Cardiology 2015 as Epub ahead of print. The authors aimed to investigate the effectivity of metformin in doxorubicin-induced cardiotoxicity using cardiac markers in blood and histopathological examination in the rat model. They concluded that metformin improved the left ventricular function, histopathologic change, and cardiomyocyte apoptosis. We congratulate the authors for this valuable investigation, and we have a few comments.

Doxorubicin (DXR) is a very effective and commonly used chemotherapeutic drug for the treatment of different types of cancers. It blocks cell division and growth by interacting DNA and RNA formation. However, it can cause a life-threatening heart damage, resulting in left ventricular dysfunction, thus limiting its usage (2).

Both atrial natriuretic peptide (ANP) and brain natriuretic peptide (BNP) are useful predictors of decreased left ventricular function in patients treated with DXR. ANP secretion from atria is triggered by atrial dilatation due to cardiac or noncardiac reasons. BNP is produced in the ventricle and is more specific for heart failure than ANP (1–3). Koh et al. (3) reported that plasma BNP levels significantly increased from 6 to 12 weeks in the doxorubicin-induced cardiotoxicity. In the study by Argun et al. (1), there was no statistical difference among groups in terms of ANP or BNP. This may be due to the design of the study, which is relatively short for the occurrence of chronic heart failure because of DXR.

Cardiac troponin (TnT) is a very specific and highly sensitive marker for myocardial damage and commonly used in clinical practice. Similar to BNP, TnT has been reported as an independent predictor of cardiac mortality in heart failure (2–4). In the study by Argun et al. (1), it would have been very helpful to measure TnT levels in terms of myocardial injury due to DXR. Thus, one could make an interpretation that TnT levels had increased in the early stage in the DXR-induced cardiotoxicity, but no change were observed in the BNP levels, which is very crucial for the early detection of DXR-induced cardiotoxicity before irreversible damage.

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References

1. Argun M, Üzüm K, Sönmez MF, Özyurt A, Karabulut D, Soyersarıca Z, et al. Cardioprotective effect of metformin against doxorubicin cardiotoxicity in rats. Anatol J Cardiol 2015 Apr 30. Epub ahead of print.
2. Kesik V, Yüksel R, Yiğit N, Saldır M, Karabacak E, Erdem G, et al. Ozone Ameliorates Doxorubicine-Induced Skin Necrosis - results from an animal model. Int J Low Extrem Wounds 2015 Aug 18. Epub ahead of print.
3. Koh E, Nakamura T, Takahashi H. Troponin-T and brain natriuretic peptide as predictors for adriamycin-induced cardiomyopathy in rats. Circ J 2004; 68: 163-7.
4. Nakamura Y, Yoshihisa A, Takiguchi M, Shimizu T, Yamauchi H, Iwaya S, et al. High-sensitivity cardiac troponin T predicts non-cardiac mortality in heart failure. Circ J 2014; 78: 890-5.

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