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Letter to the Editor

Levosimendan in acute decompensation of anthracycline-induced cardiotoxicity

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1. Case report

A 42-year-old woman with metastatic breast cancer and previous normal cardiac function was treated during the last 2 years with chemotherapy regimens that included doxorubicin (total dosage of 841 mg/m^2). Four months after the last regimen, she was admitted to the ICU with decompensation of heart failure. She presented with severe dyspnea, anasarca and was dependent on supplemental oxygen. Chest radiograph showed bilateral pleural effusion and pulmonary edema. Transthoracic echocardiogram revealed severe diffuse myocardial dysfunction (left ventricular ejection fraction of 31%). The initial treatment included furosemide, spironolactone, deslanoside, non-invasive ventilation, ACE-inhibitors and dobutamine (started at 8.3 mcg/kg/min). On the third day of ICU, patient's clinical condition did not improve and tachyarrhythmia arose after the increase of dobutamine dose (20 mcg/kg/min). Therefore, levosimendan was started at a dosage of 0.14 mcg/kg/min. Dobutamine was weaned and taken off within the first 12 h of levosimendan infusion. Two days later, patient's symptoms and clinical signs of heart failure improved; levosimendan was stopped after 48 h of infusion. She was discharged from the ICU on the following day and finally discharged home 1 month later.

2. Discussion

Cardiomyopathy with congestive heart failure (CHF) is the most serious complication of long-term doxorubicin treatment [1]. The prognosis of this clinical condition is poor since it is usually refractory to conventional therapy for CHF [1]. In episodes of acute decompensation, patients may need ICU admission and inotropic support.

Levosimendan is a calcium sensitizer that has recently been introduced in the treatment of acute heart failure and has shown advantages in terms of tolerability and long-term prognosis when compared to dobutamine [2]. Tachyarrhythmias and increases in the energetic consumption of the myocardium are potential side effects induced by increased intracellular calcium concentration associated with the use of dobutamine and phosphodiesterase inhibitors [3]. Levosimendan has been used for the treatment of cardiac failure in different scenarios [2–5]. While levosimendan is currently widely employed in the treatment of decompensated heart failure, to the best of our knowledge, there is no report of the use of levosimendan in the treatment of acute decompensation of anthracycline-induced cardiotoxicity.

A limitation of our report was that brain natriuretic peptides (BNP) were not measured. Increases in NT-proBNP levels of patients who received chemotherapy with anthracyclines can predict drug-induced cardiotoxicity [6,7]. Moreover, NT-

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proBNP levels correlate inversely with left ventricular mass in children with anthracycline-induced cardiomyopathy [8]. It was also demonstrated that levosimendan decreases NT-proBNP levels in parallel to the improvement of hemodynamic parameters of patients with severe CHF [9,10]. Therefore, sequential assessment of NT-proBNP levels could potentially be a useful biomarker in monitoring treatment response to levosimendan in patients with anthracycline cardiomyopathy.

In conclusion, although further studies are needed, levosimendan may be an interesting therapeutic option for the treatment of acute decompensation of doxorubicin-induced cardiomyopathy, especially in the setting of failure or intolerance to conventional inotropes such as dobutamine.

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