A broken heart can still be fixed: unexpected acute heart failure in a young patient

Authors:
M Belei1, DA Darabantiu1, A Sasu1, A Pop-Moldovan1, 1Emergency Hospital of Arad, Cardiology - Arad - Romania,

Topic(s):
Acute Heart Failure - Clinical

Citation:
Acute heart failure due to anthracycline-induced acute cardiotoxicity is an extremely rare finding. We present the case of a 36 year-old woman diagnosed with acute promyelocytic leukaemia, who underwent induction chemotherapy (idarubicine and all-trans-retinoic acid) in the previous 4 weeks. The patient had no cardiovascular history, baseline normal left ventricular (LV) function and was referred to our cardiology department for fatigue, atypical chest pain, dyspnea on mild exertion, palpitations, dry cough and peripheral oedema. Physical examination revealed no pulmonary rales, tachycardia, ventricular gallop, 2/6 mitral systolic murmur, blood pressure of 70/50 mmHg, ankle oedema 3+ and peripheral oxygen saturation of 98%.

Faced with a clinical setting of acute heart failure, we immediately proceeded to additional investigations to further confirm our initial diagnosis. The 12-lead electrocardiogram showed sinus tachycardia (145 bpm) and the chest X-ray was normal. Blood tests revealed an extremely elevated NT-proBNP of 35,000 pg/ml, an elevated hs-cTn I of 124,4 pg/ml (ULN- 29 pg/ml), severe pancytopenia and a significant inflammatory syndrome. The transthoracic echocardiogram (TTE) showed a normal-sized LV with apical akinesia, antero-lateral hypokinesia, moderate systolic dysfunction with an ejection fraction (EF) of 35%, apical thrombus and a moderate/severe mitral regurgitation. The patient was started on an intravenous infusion of inotropic agent (dobutamine) followed by a vasopressor (norepinephrine) in order to maintain systolic blood pressure. Intravenous loop diuretics, low-molecular-weight heparin, ivabradine and antibiotics were also administered. After stabilization, low-dose beta-blockers and ACE-inhibitors were added and continued at discharge. ECG, blood pressure, peripheral oxygen saturation and diuresis were closely monitored. A significant improvement in the patient’s clinical status was noted over the following days and a control TTE was performed one week after the initial assessment. It revealed a normal LV systolic function with an EF of 55%, no wall motion abnormality, mild mitral regurgitation but persistent LV apical thrombus. Serum levels of cardiac biomarkers registered a marked decrease with a NT-proBNP of 2941 pg/ml and a normal hs-cTnI.

The management problems in this patient were represented by the association of low blood pressure, high heart rate, congestion and thrombocytopenia (considering the need for anticoagulation).

Conclusions: Acute heart failure due to acute cardiotoxicity is one of the most noteworthy side effects of anthracycline-based chemotherapy regimens, despite being a rare event, especially after idarubicine. If detected early and treated properly with heart failure medication, a good functional recovery could be expected.
Abstract: A broken heart can still be fixed: unexpected acute heart failure in a young patient

Authors: M Belei, DA Darabantiu, A Sasu, A Pop-Moldovan

Emergency Hospital of Arad, Cardiology - Arad - Romania,

Topic(s): Acute Heart Failure - Clinical

Citation: Acute heart failure due to anthracycline-induced acute cardiotoxicity is an extremely rare finding. Among available anthracyclines, idarubicin has the lowest cardiotoxicity. We present the case of a 36 year-old woman diagnosed with acute promyelocytic leukaemia, who underwent induction chemotherapy (idarubicine and all-trans-retinoic acid) in the previous 4 weeks. The patient had no cardiovascular history, baseline normal left ventricular (LV) function and was referred to our cardiology department for fatigue, atypical chest pain, dyspnea on mild exertion, palpitations, dry cough and peripheral oedema. Physical examination revealed no pulmonary rales, tachycardia, ventricular gallop, 2/6 mitral systolic murmur, blood pressure of 70/50 mmHg, ankle oedema 3+ and peripheral oxygen saturation of 98%.

Faced with a clinical setting of acute heart failure, we immediately proceeded to additional investigations to further confirm our initial diagnosis. The 12-lead electrocardiogram showed sinus tachycardia (145 bpm) and the chest X-ray was normal. Blood tests revealed an extremely elevated NT-proBNP of 35,000 pg/ml, an elevated hs-cTnI of 124,4 pg/ml (ULN-29 pg/ml), severe pancytopenia and a significant inflammatory syndrome. The transthoracic echocardiogram (TTE) showed a normal-sized LV with apical akinesia, anterolateral hypokinesia, moderate systolic dysfunction with an ejection fraction (EF) of 35%, apical thrombus and a moderate/severe mitral regurgitation. The patient was started on an intravenous infusion of inotropic agent (dobutamine) followed by a vasopressor (norepinephrine) in order to maintain systolic blood pressure. Intravenous loop diuretics, low-molecular-weight heparin, ivabradine and antibiotics were also administered. After stabilization, low-dose beta-blockers and ACE-inhibitors were added and continued at discharge. ECG, blood pressure, peripheral oxygen saturation and diuresis were closely monitored. A significant improvement in the patient's clinical status was noted over the following days and a control TTE was performed one week after the initial assessment. It revealed a normal LV systolic function with an EF of 55%, no wall motion abnormality, mild mitral regurgitation but persistent LV apical thrombus. Serum levels of cardiac biomarkers registered a marked decrease with a NT-proBNP of 2941 pg/ml and a normal hs-cTnI.

Conclusions: Acute heart failure due to acute cardiotoxicity is one of the most noteworthy side effects of anthracycline-based chemotherapy regimens, despite being a rare event, especially after idarubicine. If detected early and treated properly with heart failure medication, a good functional recovery could be expected.