A 66-year-old Caucasian woman with a history of hypertension was admitted to our hospital with a recent diagnosis of Burkitt lymphoma.
Her lactate dehydrogenase level was high and the diameter of her mediastinal mass was more than 10cm.
Her physical examination was normal, and basal electrocardiography (ECG) showed sinus rhythm with a heart rate of 72 beats/minute.
Two-dimensional transthoracic echocardiography (TTE; Siemens, Acuson Sequoia, C512) revealed normal biventricular functions with an LV ejection fraction (LVEF) of 60%, mild mitral and tricuspid regurgitation, and moderate pericardial effusion.
A risk assessment of the patient put her into a high-risk category and she underwent rituximab-hyperfractionated-cyclophosphamide-vincristine-doxorubicin-dexamethasone (R-Hyper-CVAD) chemotherapy protocol.
Her laboratory values are summarized in Table 1.
She received high-dose cyclophosphamide 300mg/m2 twice daily for 3 days, doxorubicin 25mg/m2/day for 2 days, rituximab 375mg/m2/day for 1 day, dexamethasone 40mg/day for 4 days, and vincristine 2mg/day for 2 days.
The total treatment dose of cyclophosphamide and doxorubicin received was 1800mg/m2 and 50mg/m2, respectively.
She was given allopurinol 300mg/day perorally, sodium bicarbonate (8.4%, 10 flacon/day) infusion for 24 hours before chemotherapy, and mesna 600mg/m2/day for 2 days as prophylaxis against tumor lysis syndrome and hemorrhagic cystitis, respectively.
She also received granisetron 2mg/day and lansoprazole 30mg/day as antiemetogenic and gastric prophylaxis, respectively.
The patient developed dyspnea on the seventh day of therapy.
A physical examination revealed blood pressure of 100/60mmHg and a heart rate of 110 beats/minute.
On chest auscultation, no inspiratory sounds were heard at lower zones and inspiratory crackles were heard at middle zones.
Neither cardiac murmurs nor S3 were heard.
An ECG showed low voltage in the limb and precordial leads.
TTE showed diffusely increased myocardial echogenicity, mild pericardial effusion, and generally impaired biventricular systolic functions with an LVEF 31% and right ventricular mid-apical akinesis.
Manifest pleural effusion was also detected.
Drug-induced cardiotoxicity (myocarditis) was suspected.
Furosemide and ramipril were started.
The beta-blocker therapy the patient was already taking for hypertension was continued.
After 12 days, TTE showed an LVEF of 37% and normal right ventricular functions.
Her dyspnea decreased and she was discharged on day 20.
After 1 month, TTE showed normal biventricular functions with an LVEF of 60%.
After the first course of the R-Hyper-CVAD chemotherapy protocol, she underwent a high-dose methotrexate and cytarabine cycle.
She had severe neutropenia and pneumonia.
She had no cardiac failure symptoms during this chemotherapy course, but she declined another course of chemotherapy.
She is still in remission despite the abbreviated course of chemotherapy.