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ONE DOSE, ONE WALL: ISOLATED SEPTAL HYPOKINESIA TRIGGERED BY FIRST DOXORUBICIN EXPOSURE

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ABSTRACT

Cardiotoxicity or cardiomyopathy is a common side effect of the use of anthracyclines for the treatment of cancer patients. Anthracyclines produce their harmful effect on the heart after several cumulative doses; however, it is rare to find the development of cardiotoxicity after only the first dose of chemotherapy. We report a case of a young male known to have acute lymphoblastic leukaemia (ALL) who developed cardiotoxicity following the first dose of doxorubicin. The patient presented with dyspnoea, orthopnoea and chest pain, and bilateral limb oedema. He had a significant hypoxaemia requiring oxygen, raised jugular venous pressure, bilateral rales and a third heart sound on examination. A transthoracic echocardiography was performed and showed a low left ventricular ejection fraction of 40%, marked left ventricular dilation and septal hypokinesia, with no global hypokinesia and normal lateral wall motion, confirming our diagnosis of anthracycline-induced cardiotoxicity following the first dose. This report discusses a rare finding of cardiotoxicity following the first dose of doxorubicin in a young male patient with ALL. Doxorubicin is known to cause dilated cardiomyopathy and global hypokinesia; nonetheless, our patient had an isolated septal hypokinesia with normal lateral wall motion. This case should raise awareness about early prevention of cardiotoxicity as it can occur early in the treatment, rather than waiting for late onset of toxicity.

KEYWORDS

Cardiotoxicity, cardiomyopathy, hypokinesia, doxorubicin, anthracycline, echocardiography

LEARNING POINTS

- Cardiotoxicity is a life-threatening side effect of chemotherapy that can occur after the first dose.
- Cardiotoxicity can present in a variety of atypical presentations and findings.
- Anthracyclines can cause cardiotoxicity following the first dose of therapy, causing isolated septal hypokinesia.

INTRODUCTION

Cardiotoxicity is defined as damage to the heart muscle or cardiac function ranging from irregular heartbeats to heart failure, due to the use of chemotherapeutic agents such as anthracyclines[1]. Chemotherapeutic agents are evolving and showing changes with respect to survival rate of cancer

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patients, but the adverse side effects of these drugs remain a challenge, specifically to the cardiovascular system[1]. To optimise toxicity reduction in patients on chemotherapy, we should assess the risk factors and comorbidities before treatment and observe close follow-up during treatment to ensure safety of the heart muscle and function, aiming to decrease the incidence of drug induced toxicity[2]. Guidelines have already included biomarkers as a tool to predict cardiotoxicity and should be used accordingly. The benefits of cardiac biomarkers in early detection of cardiotoxicity during the period of chemotherapy has been studied extensively[2]. Studies have shown that increased cumulative doses of anthracycline causes cardiotoxicity in 4% of patients, reaching 36% when using a dose of 500–550 m/m^2[3]. Although it is known that doxorubicin causes late cardiotoxicity following a cumulative dose of the drug it can, in rare cases, cause early onset toxicity, following the first dose of chemotherapy. We present a case of an 18-year-old male patient known to have acute lymphoblastic leukaemia (ALL) who experienced cardiotoxicity following his first dose of doxorubicin. Transthoracic echocardiography (TTE) was performed and showed a low left ventricular ejection fraction (LVEF) of 40%, marked left ventricular dilation and septal hypokinesia, with no global hypokinesia and normal lateral wall motion, confirming our diagnosis of anthracycline-induced cardiotoxicity following the first dose. This report is highly important as it shows that cardiotoxicity may manifest in patients within few days following the first session of chemotherapy, which is a rare finding in the literature. Furthermore, anthracyclines usually cause global hypokinesia; however, our patient had an isolated septal hypokinesia, which to our knowledge is reported for the first time.

CASE DESCRIPTION

An 18-year-old male patient, recently diagnosed with ALL, presented initially for his first chemotherapy session. He started with induction therapy including anthracycline, 50 mg on day 0 and day 1 in accordance with his body surface area (i.e. 30 mg/m^2). TTE pre-chemotherapy was performed and revealed a normal systolic function with an ejection fraction of 60%. One week later after his first dose of chemotherapy, the patient started experiencing dyspnoea, orthopnoea and chest pain, and was admitted to the ICU. His blood pressure was 166/63 mmHg, heart rate was 125 bpm and his temperature was 37.6°C. A physical examination was remarkable for bilateral basilar crackles heard on lung auscultation and mild bilateral lower leg pitting oedema. He had a significant hypoxaemia requiring oxygen. He had a raised jugular venous pressure, bilateral rales and a third heart sound on examination. A 12-lead ECG was done and found to be normal with no ST segment changes *(Fig. 1)*. Laboratory blood tests came back significant for hypokalaemia, severely elevated troponin I (28.2 ng/ml) and proBNP (4,065 pg/ml). C-reactive protein and D-dimer were elevated. The rest of the laboratory workup was normal. A chest X-ray revealed mildly congested lungs. A TTE was performed and showed a low LVEF of 40%, marked left ventricular dilation *(Fig. 2 and 3)* and septal hypokinesia *(Fig. 4)*, with no global hypokinesia and normal lateral wall motion *(Fig. 5)*. Left and right atria and right ventricle were all normal.

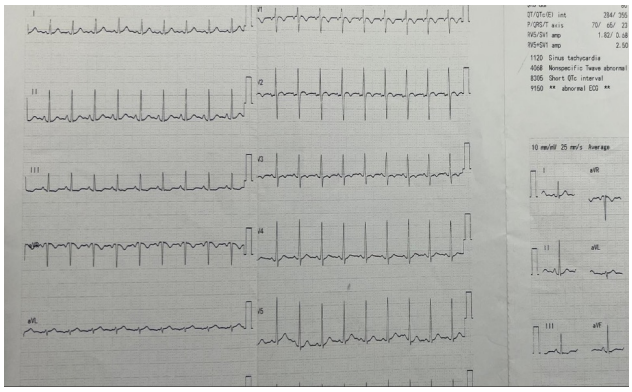


Figure 1. *ECG showing a sinus tachycardia rhythm with no ischaemic changes.*

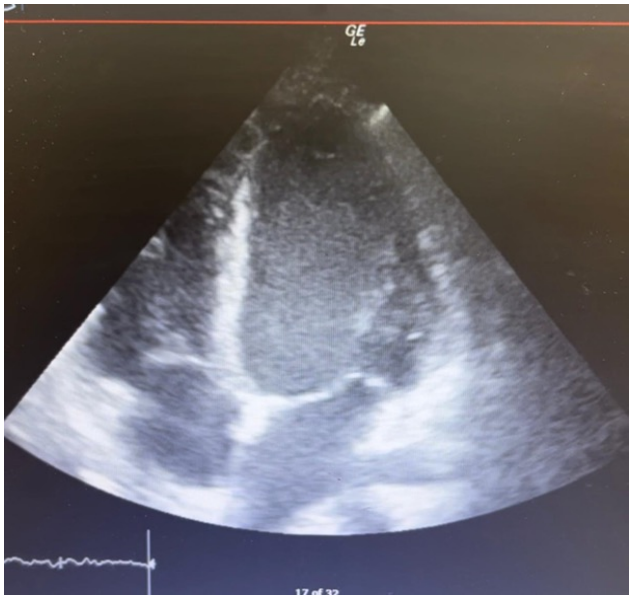


Figure 2. *Apical view on echocardiography showing a markedly dilated left ventricle.*



Figure 3. *Echocardiography showing left ventricle dilated to 3.54 cm.*

All four valves were normal, and no pericardial effusion was noted. Compared to the TTE performed pre-chemotherapy, the LVEF had decreased by 20%. The patient had developed chemotherapy-induced cardiomyopathy and acute heart failure and was immediately placed on a beta blocker (bisoprolol fumarate 2.5 mg) daily. Moreover, during his stay, he developed neutropenic fever with an absolute neutrophil count of 27.7, fever of 39°C and diarrhoea. He was placed on filgrastim, metronidazole 500 mg IV, fluconazole 200 mg IV, acyclovir 400 mg and meropenem 2 g IV. This patient had developed chemotherapy-induced cardiotoxicity after only the first session of anthracycline. This is a relatively rare finding, as cardiotoxicity usually occurs in the setting of cumulative doses.

DISCUSSION

Chemotherapy-induced cardiotoxicity can occur in the early or late phase of the treatment[3]. Its incidence depends on several factors including the drug type, dosing regimen, cumulative dose and patient-related factors such as age and history of previous cardiovascular disease[3]. Anthracycline is the most commonly known chemotherapy agent associated with cardiotoxicity. It produces its effect with an increasing cumulative doses and occurs in 4% of patients, reaching 36% in the case of a dose of 500–550 mg/m^2[3].

Anthracycline-induced cardiotoxicity has been divided into three categories: acute (occurring transiently immediately after infusion, with an incidence of <1%), early onset (within the first year after treatment with an incidence of 2%) and late onset occurring at least one year after completion of therapy[3].

Anthracycline cardiomyopathy manifests mainly as left ventricular dilation, heart failure, myocarditis and arrhythmia[4]. According to Florescu et al. (2013), chemotherapy-induced cardiotoxicity is defined as reduction of LVEF of 5-10% from baseline below 55%, either global or specific in the interventricular septum with symptoms or signs associated with heart failure[4].

In our case, the patient experienced cardiotoxicity following the first dose of anthracycline in the induction phase for treatment of ALL. This finding is very rare, as there are only two cases reported in the literature. The first occurred after 26 days with echocardiography revealing left atrial dilation and generalised hypokinetic wall motion with a LVEF of 23%, progressing to ventricular tachycardia and then fibrillation on day 28[5]. The second occurred 15 days after treatment. His TTE showed a severely reduced LVEF of 25%, a decrease in right ventricular function and pulmonary hypertension[6]. Our patient developed cardiomyopathy after one week of doxorubicin and echocardiography showed a septal hypokinesia, which is uncommon compared to the known global hypokinesia effect of doxorubicin, and he did not develop any arrhythmias.

Cardiotoxicity after the first dose of chemotherapy has been reported in drugs other than anthracycline, mainly 5-FU; however, it manifests with different symptoms and signs

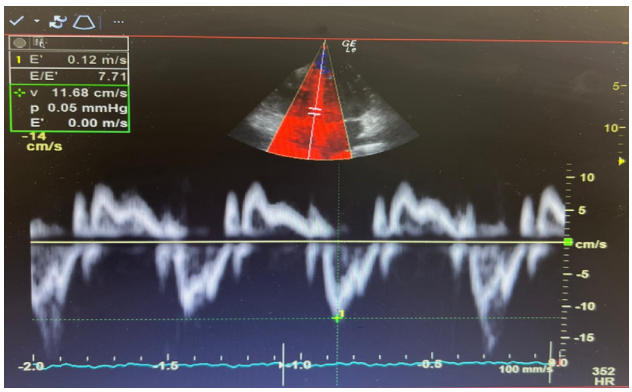


Figure 4. Echocardiography showing septal hypokinesia (E':0.12), and normal diastolic function.

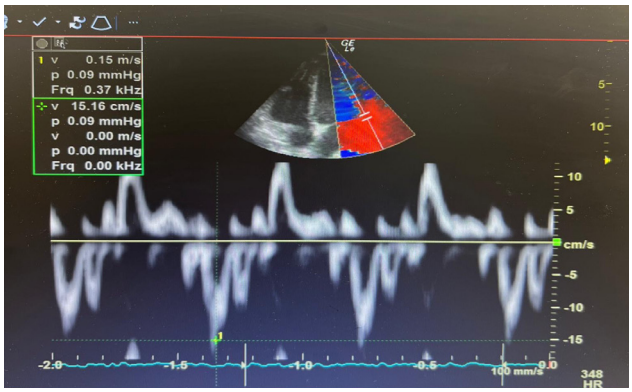


Figure 5. Normal motion of the lateral wall of the left ventricle (V: 0.15m/s) with no evidence of global hypokinesia.

such as coronary vasospasm with visible ST changes on ECG with a normal coronary angiogram. In addition, there may be the development of polymorphic ventricular tachycardia[7], myopericarditis, elevated troponin with global hypokinesia with left ventricular systolic dysfunction and LVEF of 45%[8]. Contrary to the commonly found global hypokinesia, our case showed isolated anteroseptal hypokinesia. In addition, elevated troponin was mostly reported in 5-FU induced cardiotoxicity; our patient had elevated troponin and NT-pro BNP following anthracycline, combining the features of both anthracycline and 5-FU-induced cardiotoxicities.

To our knowledge, this is the third case of doxorubicin-induced cardiotoxicity after the first dose reported. Although this toxicity occurs mainly in middle-aged or elderly people, our patient was a young man, 18 years of age. Septal hypokinesia has been reported in cases post myocardial infarction; however, it occurred in our patient following chemotherapy. It is the first reported case of isolated septal hypokinesia in the literature.

In 2022, the European Society of Cardiology (ESC) unveiled new guidelines regarding anthracycline-induced cardiotoxicity. A Class IIa recommendation is the use of cardio protective drugs for primary prevention such as ACE inhibitors and beta blockers in high-risk patients. Dexrazoxane must be administered to patients who require an ongoing anthracycline therapy regimen. Added to that, medications with less risk of cardiotoxicity must be administered, such as liposomal anthracycline formulations

(PEGylated liposomal doxorubicin), all with Class IIa evidence. The ESC recommends baseline measurement of NPc and/or cTnd in all patients with cancer at risk of cancer therapy-related cardiac dysfunction (CTRCD) and monitoring before every cycle of anthracycline, at 3 and 12 months after completion of therapy in very high-risk patients with Class I evidence[9]. Additional studies and clinical trials were performed to develop new potentials for this issue. For instance, empagliflozin reduced CTRCD in high-risk patients, and preserved the ejection fraction over the course of six months[10]. Similarly, statins, particularly atorvastatin, lowered CTRCD and reduced LVEF decline in a randomised controlled trial on breast cancer patients[11]. In patients with an elevated baseline heart rate, the rate-lowering drug ivabradine proved to be promising, as it preserved cardiac function and decreased both the incidence of asymptomatic cardiac dysfunction and troponin elevation in a randomised controlled trial, but caused multiple visual disturbances[12]. These findings show the potential that such therapies have in mitigating anthracycline cardiotoxicity and improving the quality of life for patients requiring anthracycline-based therapy.

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