

Dilated Cardiomyopathy Secondary to Toxicity from Anthracycline Chemotherapy in a Patient with a History of Acute Lymphoblastic Leukemia: A Case Report

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ABSTRACT

Dilated cardiomyopathy is defined by presence of left ventricular or biventricular dilation and left systolic dysfunction in absence of hypertension, coronary disease, or valvular disease that would justify it. Its causes can be divided into two large groups: those of genetic origin and those of nongenetic origin, which include infectious, toxic, and autoimmune disease causes, among others. Its natural course varies significantly based on the different etiologies and its diagnosis can influence the disease's management and prognosis.

Clinical case: 29-year-old male, with a history of having suffered from Acute Lymphoblastic Leukemia at the age of 4 with complete remission of the disease demonstrated at that time by bone marrow aspirate and immunophenotype, having been treated with anthracyclines. On this occasion, he attended due to the presence of sudden onset dyspnea and hemoptysis, for which in the first instance, due to the previously mentioned history, an attempt was made to rule out pulmonary thromboembolism with chest Angio tomography, which was corroborated by said study and demonstrating the presence of two intracavitary thrombi. Therefore, a transthoracic echocardiogram was performed, highlighting severe dilation of the left ventricle. Management for heart failure was adjusted based on structural alteration demonstrated by echocardiography, with favorable clinical evolution.

Conclusions: The natural history of dilated cardiomyopathy is not easy to establish due to the wide heterogeneity of its etiology and the different rates of progression depending on it. In some cases, there may be functional recovery after acute myocardial damage, for example, in cardiomyopathy induced by tachycardia or in that induced by cardiotoxic drugs. In other cases, stabilization of the disease and systolic dysfunction and, in others, sudden death may be observed.

KEYWORDS: Dilated cardiomyopathy, anthracyclines, male, dyspnea, heart failure.

ARTICLE DETAILS

Published On:
05 November 2025

Available on:
<https://ijmscrs.com/>

BACKGROUND

Dilated cardiomyopathy (DCM) is defined by the European Society of Cardiology (ESC) as the presence of left ventricular or biventricular dilation and systolic dysfunction in the absence of abnormal loading conditions (valve disease or hypertension) or coronary artery disease sufficient to account for global systolic impairment¹.

In 2016, the ESC presented a revised definition of DCM, emphasizing that this diagnosis encompasses a wide range of genetic and non-genetic diseases².

Determining the incidence and prevalence of DCM has been challenging due to geographic variability, incomplete penetrance of the disease³, or late onset of presentation⁴, as well as changes in diagnostic criteria⁵.

DCM is currently considered one of the leading causes of heart failure and heart transplantation. The prevalence of

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DCM has traditionally been estimated at 36 per 100,000 population, with an annual incidence of 6 per 100,000⁶. The ESC classifies the causes of DCM into two large groups: genetic and non-genetic causes, although the interaction between genetic predisposition and environmental factors is sometimes also relevant. DCM is recognized as a genetically transmitted disease in at least 30–40% of cases. 2 Other factors linked to the pathogenesis of DCM are anthracycline and doxorubicin derivatives, whose metabolites are believed to act as toxicants for myocytes, producing structural and functional changes in the myocardium. The common pathophysiological basis for the different etiologies of DCM is the loss of myocardial contractile capacity⁷.

CLINICAL CASE

This is a 29-year-old male patient, originally from and residing in Pabellón de Arteaga, Aguascalientes. He works as a warehouseman. He reports his civil status as a common-law spouse and completed high school. His family history only includes paternal genetics for hypertension. Of his significant medical history, he reports having suffered from acute lymphoblastic leukemia at age 4, undergoing treatment with anthracycline-based chemotherapy, with complete remission of the disease based on bone marrow aspiration and immunophenotyping during follow-up by the hematology department.

On this occasion, he presented to the unit with progressive dyspnea, which progressed from severe to mild exertion, lasting approximately 2 months. He reported that he had not previously experienced dyspnea, even performing physical activity without any difficulty, until presenting with difficulty, even at work, moving boxes of packages (clothing, electronics). He also progressively developed lower limb edema, which he had not previously experienced in his life. This edema worsened at the end of the day and improved with rest. In the last week prior to admission, he also mentioned having experienced hemoptysis, not associated with fever or chest pain, with an increase in the amount of hemoptysis during that week, which is why he decided to come to our unit for evaluation.

Upon arrival at the emergency department, his vital signs were reported within normal parameters, except for his oxygen saturation by pulse oximetry, which was 82%. This improved with the support of supplemental oxygen via nasal prongs at 2 liters/minute. No significant laboratory abnormalities were observed. However, due to his history of leukemia, the onset of hemoptysis accompanied by dyspnea and the need for supplemental oxygen, a chest Angio tomography was ordered. The etiology of the presenting symptoms was suspected to be pulmonary thromboembolism. The following were reported: pulmonary thromboembolism of the inferior lobar artery and right posterior basal segmental artery, and multiple intraventricular thrombi. The patient

presented with pleural effusion and atelectasis in the right region.

Based on the diagnosis reported on the imaging study, the patient was taken to the Internal Medicine floor for further treatment. Upon arrival at our unit, a further interview was conducted, confirming a clinical history consistent with heart failure (dyspnea, orthopnea, bendopnea, paroxysmal nocturnal dyspnea, electrocardiogram with sinus tachycardia, chest X-ray with bilateral pleural effusion). A significant physical examination revealed crackles in both lung bases and lower extremity edema (++/+++). He was managed as a patient in his third decade of life with heart failure and the presence of intracavitary thrombi seen on contrast-enhanced tomography, so an assessment was requested by the cardiology service who completed it with a transthoracic echocardiogram where the following were reported: 1. Severely dilated left ventricle (indexed TSV 63 ml/m²ASC, indexed LVSD: 31 mm/m²ASC), normal ventricular geometry. Indexed left ventricular mass: 96 g/m², RWT: 0.28. Intracavitary thrombi in the left ventricle measuring 30x16 and 10x13 mm. 2. Resting segmental motion alterations. 3. LV diastolic dysfunction type III/III. Restrictive pattern. 4. Normal-sized right cavities. 5. Slightly dilated left atrium. 6. Severe LV systolic dysfunction: LVEF 26% 7. RV systolic dysfunction. TAPSE 13 mm, S': 8.0 cms/s, Tei: 0.82 8. Mild mitral and pulmonary regurgitation. 9. Resting valvular flowmetry within normal parameters. No stenosis. 10. Low probability study for pulmonary hypertension. PASP 35 mmHg PMAP 28 mmHg. 11. Aortic arch with no alterations, no significant anterograde gradient. 12. Pericardium with normal characteristics. 13. No shunts at the time of the study. He was also evaluated by the cardiothoracic surgery service, who referred him as requiring tertiary level care for cardiopulmonary bypass surgery. Therefore, in our unit, optimal management was established for heart failure with reduced LVEF and anticoagulation due to the presence of intracavitary thrombi and the established pulmonary thromboembolism according to Spanish and American cardiology guidelines. The patient presented adequate clinical progress, without requiring tertiary level care upon evaluation. Therefore, the patient was discharged from the service without presenting new conditions.

DISCUSSION

A thorough clinical history must be taken to arrive at a diagnosis, identifying possible treatable or reversible causes of the disease. In our case, there was a well-established history of myocardial damage, such as exposure to anthracyclines.

The symptoms and signs are a consequence of left-sided heart failure, generally with nonspecific symptoms such as asthenia and exertional dyspnea. Depending on the degree of increased left atrial pressure, dyspnea can present with varying degrees of severity⁸, as was the case in our patient, with progressive

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dyspnea, even leading to hemoptysis due to the established structural damage, combined with concomitant pulmonary thromboembolism and the presence of intracavitary thrombi. If heart failure is suspected, we should order various complementary tests to confirm our diagnosis:

1. Chest X-ray: Cardiomegaly is usually observed; however, we seek to rule out acute decompensation with findings such as signs of pulmonary venous congestion and, in advanced stages, signs of interstitial or alveolar pulmonary edema⁹.
2. Electrocardiogram: This is nonspecific; signs of atrial enlargement and left ventricular hypertrophy are common⁹.
3. Echocardiography: This is a very useful technique for confirming the diagnosis and assessing cardiac function. DCM is defined by the presence of left ventricular or biventricular systolic dysfunction (LVEF less than 45%) associated with ventricular dilatation. Ventricular dilatation is defined by an end-diastolic volume or end-diastolic diameter indexed by body surface area greater than the upper limit of normal, defined as two standard deviations above the mean. It is recommended to use normal values calculated for the patient's age and sex as a reference⁹.
4. Cardiac magnetic resonance imaging: Cardiac magnetic resonance imaging (CMR) is currently considered the gold standard for measuring ventricular volumes, mass, and ejection fraction. In routine clinical practice, its use is sometimes limited by its low availability and high cost. It is a tool that should be considered at least once in every patient diagnosed with DCM. It can provide important information regarding both the etiological diagnosis and prognostic stratification⁹.

Within our diagnostic approach, the echocardiogram results were able to be performed, meeting the previously described characteristics. This justified adjusting management based on guidelines for heart failure with reduced LVEF and adequate clinical evolution. However, as previously mentioned in the literature review, it would be important to perform an magnetic resonance for the first time in our patient and evaluate the need for genetic testing due to its relationship with the described pathology.

CONCLUSIONS

The fundamental objectives of DCM treatment are the improvement of symptoms and quality of life in these patients. Therefore, patient education on the control of heart failure triggers, such as non-adherence to treatment or diet, is very important, as well as the early initiation of appropriate treatment, both with pharmacological¹⁰ and non-pharmacological measures¹¹.

Left ventricular reverse remodeling is the most significant prognostic determinant in the progression of DCM¹² and should be our primary therapeutic target¹³.

Male sex and age over 60 years have been associated with a worse prognosis in patients with DCM and HF¹⁴. On the other hand, higher levels of systolic function and lower levels of

ventricular dilation are associated with a higher likelihood of reverse remodeling¹³.

Patient management requires follow-up by both primary care and cardiology to complete the diagnosis and treat complications that cannot be treated on an outpatient basis¹⁵.

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