Case report

Dilated cardiomyopathy associated with cocaine use: clinical case and review

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Miocardiopatía dilatada asociada a uso de cocaína: caso clínico y revisión

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Abstract
Case presentation. A 33 year old male patient, with recently diagnosed hypertension and chronic cocaine use, presented with symptoms of congestive heart failure. He underwent an echocardiogram that reported dilatation of all four cardiac chambers and decreased left ventricular ejection fraction. The etiology of the dilated cardiomyopathy was investigated, and infectious causes were ruled out. **Treatment.** The patient was managed in the acute phase with diuretics, dobutamine drip, beta-blockers, and digitalis, in addition to vericiguat, and presented with evident clinical improvement. **Outcome.** A catheterization was recommended to rule out ischemic coronary artery disease as a differential diagnosis, in addition to magnetic resonance or endomyocardial biopsy to rule out other infiltrative pathologies; however, these were not performed due to the patient's refusal and study limitations. The patient requested voluntary discharge, and medical management was indicated, with continued outpatient study in a clinic.

Keywords
Heart Failure, Stroke Volume, Left Ventricular Dysfunction, Dilated Cardiomyopathy, Cocaine-Related Disorders.

Resumen
Presentación del caso. Se describe el caso clínico de un paciente masculino, de 33 años, con hipertensión arterial de reciente diagnóstico y consumo crónico de cocaína, que debutó con síntomas de insuficiencia cardíaca congestiva. Se le realizó un ecocardiograma que reportaba dilatación de las cuatro cavidades cardíacas y una fracción de eyección del ventrículo izquierdo disminuida. Se investigó la etiología de la cardiomiopatía dilatada, y se descartaron las causas infecciosas. **Intervención terapéutica.** Se manejó en la fase aguda con diuréticos, goteo de dobutamina, betabloqueadores, digitálicos, además de uso de vericiguat, y el paciente presentó evidente mejora clínica. **Evolución clínica.** Se recomendó realizar un cateterismo para descartar enfermedad coronaria isquémica como diagnóstico diferencial, además resonancia magnética o biopsia endomiocárdica para descartar otras patologías infiltrativas, sin embargo, no se realizaron por negativa de paciente y limitaciones de estudios. El paciente solicitó el alta voluntaria, y se indicó manejo médico y continuar estudio ambulatorio en consulta externa.

Palabras clave
Insuficiencia Cardíaca, Volumen Sistólico, Disfunción Ventricular Izquierda, Miocardiopatía Dilatada, Trastornos Relacionados con Cocaína.

Introduction

Dilated cardiomyopathy (DCM) is defined as the presence of left ventricular dilatation and global or regional systolic dysfunction not explained solely by abnormal overload conditions or coronary artery disease.[i] This is one of five types of cardiomyopathy (hypertrophic, dilated, non-dilated left ventricular, arrhythmogenic right ventricular, and restrictive).[i]

It is an important cause of cardiovascular morbidity and mortality due to congestive heart failure, and in 20 % of cases, genetic origins have been identified.[ii] Mortality due to severe congestive heart failure can reach 50 % two years after diagnosis.[iii]

The prevalence of this disease is associated with 2 % to 3 % of left ventricular systolic dysfunction and 1.5 % of congestive heart failure in the general population.[iii] Although MD is most often diagnosed in mid-life,[i] it is estimated that up to 36 % of cases occur in young patients.[iv] These patients usually present in emergency departments with symptoms of frank heart failure, and upon clinical evaluation, it is possible to identify ventricular dilatation and remodeling, the presence of myocardial fibrosis, and systolic and/or diastolic function dysfunction.[v] Therefore, a clinical case is presented as an opportunity to describe the most frequent etiologies of dilated cardiomyopathy, as well as to address possible associated risk factors and therapeutic options, based on a review of the current literature.

Presentation of the case

A 33 year old male patient, originally from Guatemala, with a history of arterial hypertension diagnosed six months prior to consultation, has been under pharmacological treatment since diagnosis with losartan 50 mg orally, once a day. He denied the use of alcohol, tobacco, or illicit drugs; however, he reported a clinical picture of one month of evolution, characterized by bilateral edema of the lower limbs with an ascending pattern up to the infrapatellar region.

This was accompanied by dyspnea initially induced by maximal efforts, with gradual progression over two weeks, until it was limited to minimal efforts. One day prior to the consultation, he presented with intolerance to dorsal decubitus, compatible with orthopnea of recent onset. For this reason, the patient was referred to a public hospital for clinical evaluation, where signs of pulmonary congestion and vital signs were noted: blood pressure 130/80 mmHg, respiratory rate 20 breaths per minute, and oxygen saturation 99 %.

In addition, the patient verbally expressed the history of cardiomegaly, so it was decided to perform an echocardiogram, which reported: dilatation of the four cardiac cavities, akinesia of the apex, anterior, septal, and lateral walls, and severe hypokinesia of apical and middle segments of the inferior wall, in addition to severely decreased left ventricular systolic dysfunction with LVEF (left ventricular ejection fraction) of 22 %, right ventricular systolic dysfunction, mitral and tricuspid insufficiency, and mild pulmonary arterial hypertension PSAP (pulmonary artery systolic pressure) of 32.9 mmHg.

For this reason, it was classified as congetive heart failure, which was managed with intravenous furosemide at a dose of one ampoule every eight hours and oxygen therapy with a nasal cannula at five liters per minute. However, the patient was transferred due to hemodynamic instability to a hospital of higher complexity, where he was received with a blood pressure of 130/80 mmHg, a pulse of 110 bpm, a respiratory rate of 20 rpm, and an O_2 saturation of 97 %.

On physical examination, the jugular veins were found to be ingurgitated in grade II, the pulmonary semiology evaluation revealed bilateral congestive type crepitant rales. A gallop heart rhythm, systolic murmur in mitral and tricuspid foci, Levine II, and displacement of the maximum impulse point towards the sixth intercostal space were identified. At the abdominal level, moderate ascites was found, hepatojugular reflux was present, and in the extremities, lower limb edema grade II.

On admission, laboratory tests were requested (Table 1); however, troponin measurement was not included due to the hospital center's unavailability at the time of care. A posteroanterior chest X-ray showed grade IV cardiomegaly. The electrocardiogram showed sinus tachycardia, atrial enlargement, and a T-wave inversion on the lateral aspect (Figure 1).

On the third day of admission, new laboratory studies were sent (Table 2). The results included: IgG antibodies against *Trypanosoma cruzi*, with negative results, and a non-reactive rapid HIV (human immunodeficiency virus) test. Surface antigen for hepatitis B with a value of 0.3 S/CO (signal to cut-off index, reference value: 0.0-0.9), antibodies for hepatitis C with a value of 0.2 S/CO (reference: 0.0-0.9), and antistreptolysin O (ASO) of 125 IU/ml (reference value: 0.0-200). All three had negative results for these diagnostic suspicions.

Therapeutic intervention

The patient continued treatment with intravenous diuretics (furosemide) as part of the management of acute heart failure, in addition to dobutamine 6 mg/min due to its positive inotropic effect. According to what was documented in the medical evolution, the patient presented clinical improvement during his hospital stay (however, the specific criteria supporting this assessment were not detailed), so spironolactone was added to the treatment, in a dose of half a tablet orally every day, enalapril 5 mg orally every day, beta-blockers such as carvedilol 6.25 mg orally every day, clopidogrel 75 mg via orally every day. In addition, the use of dapagliflozin 5 mg daily and vericiguat 2.5 mg orally daily was indicated to improve prognosis and reduce hospitalizations.

An evaluation by a cardiologist was requested, who indicated starting digoxin via a digitalization scheme with an initial dose of 0.25 mg intravenously, followed by 0.25 mg intravenously every eight hours, and then continuing with 0.25 mg intravenously once a day as a maintenance dose.

Clinical course

Since his admission to the referral hospital, the patient was managed for acute decompensation of heart failure, and simultaneously, the etiological investigation of MD was initiated. Infectious causes such as Chagas disease, HIV, Hepatitis B and C, and a streptococcal infection, which could have triggered the indirect cardiac damage, were ruled out. In the absence of a clear etiology after initial examinations, cardiaccatheterization was recommended for further diagnostic evaluation; however, the patient declined such a procedure for personal reasons.

Table 1. Laboratory tests

Examination	Day 1	Day 3	Day 4	Units	Reference value
Leukocytes	7,5	9,7	9,2	$10^3/mm^3$	5-10
Neutrophils (%)	66,1	63,3	63,4	%	50-70
Lymphocytes (%)	24,7	25,7	24,9	%	20-40
Monocytes (%)	7,4	9,6	10,2	%	3-12
Hemoglobin	16,7	17,5	16,9	g/dL	12-16
Hematocrit (%)	52,0	52,5	49,4	%	37-54
Platelets	211	240	221	$10^3/\mu L$	150-400
Creatinine	1,2	1,1	0,9	mg/dl	0,4-1,2
Urea nitrogen	23,9	18,0	18,0	mg/dl	5-18
Sodium	137,0	141,0	139,0	mEq/l	135-150
Potassium	4,0	3,8	3,4	mEq/l	3,5-5,5
Calcium	-	9,4	9,0	mg/dl	8,5-10,5
CPK	-	52,0	-	UI/L	38-174
CPK-MB	32,2	14,2	13,9	UI/L	0-25
Albumin	-	3,9	-	g/dL	3,5-5,0
TPT	25,0	-	-	Seg	24,2-32,8
TP	15,8	-	-	Seg	8,6-11,6
INR	1,4	-	-	-	-
C Reactive Protein	-	0,2	-	mg/dl	0-8

Table 2. Complementary laboratory tests

Examination	Day 4	Day 9	Day 10	Units	Reference value
Total Cholesterol	101,0	-	-	mg/dL	140-200
HDL	33,0	-	-	mg/dL	35-65
LDL	53,0	-	-	mg/dL	75-100
Triglycerides	102,0	-	-	mg/dL	40-150
Free T3	3,4	-	-	pg/mL	2,5-3,9
Free T4	1,0	-	-	ng/dL	0,61-1,12
TSH 3rd generation	1,4	-	-	UI/mL	0,34-5,6
Rheumatoid factor	-	2,0	-	UI/mL	0-15
Uric acid	-	-	4,3	mg/L	2,3-6,1
Serum iron	-	-	177,8	µg/dL	60-180
Ferritin	-	-	262,5	µg/L	10-120

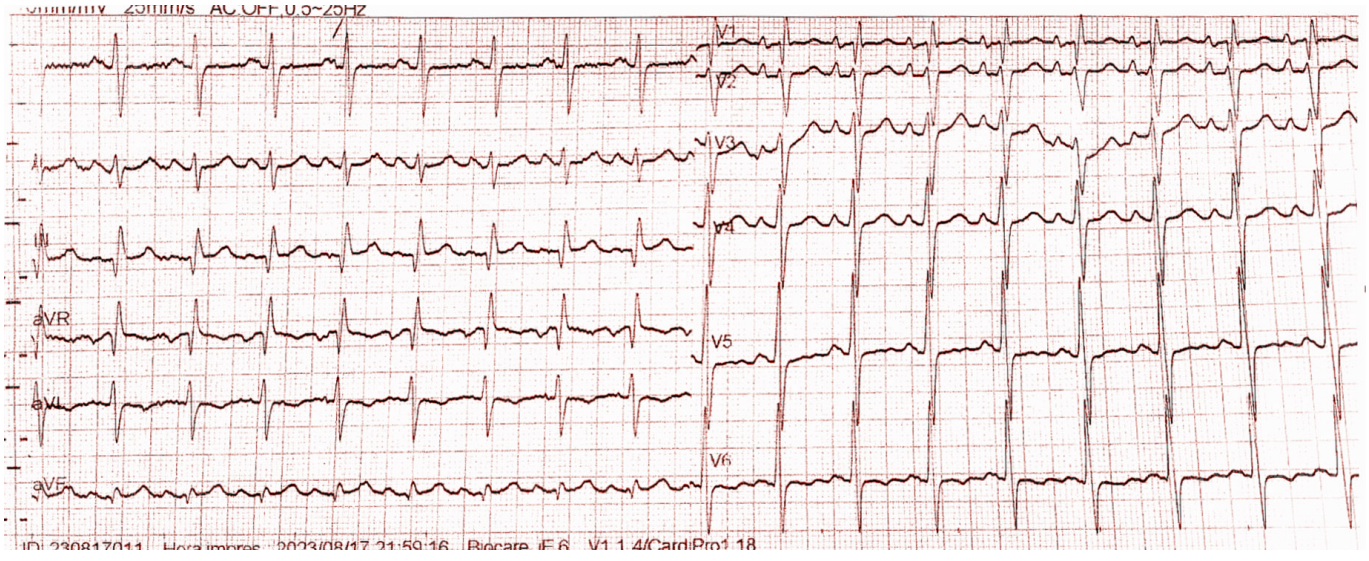

Figure 1. ECG. A 12-lead electrocardiogram is presented, velocity of 20 mm/s and voltage of 10 mm/mV. Heart rate of 110 bpm. QRS complex: 0.12 sec, P wave of 0.12 sec. T wave of 0.20 sec. Sinus tachycardia, atrial enlargement and T wave inversion on the lateral side.

Also, other etiologies could have been explored in this patient by complementary studies such as cardiac magnetic resonance imaging or an endomyocardial biopsy with Congo red staining, specific for amyloid, which could have been very useful for the differential diagnosis of infiltrative heart disease, including sarcoidosis, amyloidosis, and deposition cardiomyopathies such as hemochromatosis. These studies are essential because management and prognosis vary significantly according to etiology; however, they were not possible to perform due to limitations in the hospital center's resources.

During hospitalization, and after multiple clinical interrogations, the patient admitted a history of chronic cocaine use lasting approximately ten years. However, a confirmatory toxicological study was not performed due to a lack of resources and because the patient requested voluntary discharge. The patient was discharged with oral therapy already established, outpatient cardiology and internal medicine controls were indicated; however, after discharge, the patient did not attend his controls.

Clinical diagnosis

Congestive heart failure, cocaine-induced dilated cardiomyopathy, biventricular systolic dysfunction was diagnosed.

Discussion

DCM consists of ventricular dilatation and decreased systolic function, leading to heart failure.[vi] It has a prevalence of 36 to 40 per 100 000 inhabitants, with a predominance in men under 50 years of age.[i] Well-known causes have been described as poorly controlled high blood pressure and secondary causes such as coronary artery disease,[vii] or excessive alcohol consumptioni. In this patient, the most relevant risk factors were arterial hypertension and chronic cocaine consumption. Arterial hypertension favors ventricular pressure overload and myocardial remodeling. At the same time, cocaine, one of the most widely consumed psychoactive substances worldwide, induces coronary vasoconstriction, hypertrophy, and myocardial fibrosis, which together cause remodeling, accelerating the development of DCM.[vii,viii]

Studies have shown an increase in the left ventricular end-systolic volume mass index and a decrease in LVEF in patients with long-term cocaine use, changes that, for the most part, were found in the patient.[vii] The common pathophysiological basis is the loss of myocardial contractile capacity.[ix]

The causes can be grouped into two broad categories: genetic and non-genetic.[ix] Among the non-genetic causes, those of inflammatory-immunological origin stand out, with viral myocarditis being one of the most frequent etiologies, related to agents such as cytomegalovirus (CMV), HIV, or infiltrative diseases such as amyloidosis or sarcoidosis. In this case, pathologies such as HIV, diabetes *mellitus*, and thyroid dysfunction were ruled out. However, no specific study for CMV, amyloidosis, or sarcoidosis was performed, for reasons previously stated, and given the patient's refusal to continue with complementary studies.

Other possible causes, such as coronary artery disease, previously known, should also be considered. The main symptom that motivated the patient to consult was dyspnea, which is considered the most frequent reason for consultation.[x] The manifestations of cardiomyopathies vary according to the type of systolic, diastolic, or both dysfunction.[i] In addition, cardiac arrhythmias (especially atrial fibrillation), weakness, or fatigue may be found.[xi] Radiological findings may include cardiomegaly and evidence of pulmonary congestion.[xii]

Electrocardiographic findings are usually nonspecific, sinus tachycardia, cavity growth,[ix] left systolic overload,[ix] T-wave abnormalities, atrial fibrillationxi, and LBBB (left bundle branch block). It is worth noting that the latter finding is associated with ventricular mechanical dyssynchrony, which compromises hemodynamic efficiency by reducing cardiac output.

The gold standard, at present, is cardiac magnetic resonance imaging,[ix] since it provides information on etiology and prognostic stratification;[ix] however, in the patient described in this case report, it was not performed due to economic aspects.

Other resources that can be considered are positron emission tomography, endomyocardial biopsy, and coronary angiography (cardiac catheterization) in persons with no known history of coronary artery disease, to better define the coronary anatomy and rule out occult ischemic disease,[xi] or to measure intracardiac pressures, which are useful when evaluating the degree of evolution of the disease, response to treatment, or to establish an indication for cardiac transplantation.

For the management of acute complications, such as heart failure, the use of four drugs is indicated: beta-blockers, mineralocorticoid receptor antagonists, SGLT-2 inhibitors, and angiotensin/neprilysin receptor antagonists (ARNI), as well as other medications, including angiotensin-converting enzyme inhibitors (ACE inhibitors), diuretics, and digoxin.[xiii]

Beta-blockers can improve ventricular remodeling, function, and clinical efficacy.[xiii] Ivabradine can improve cardiac function by reducing heart rate, which decreases myocardial oxygen demand, improves coronary perfusion, and optimizes ventricular filling, thus favoring cardiac output.[xiii,xiv]

It should be taken into account whether the etiology of DCM is known, in which case it will have a specific treatment.[xv] Reduced ejection fraction is associated with high morbidity and mortality.[xvi] Vericiguat, which has been shown to be effective and safe[xvi] by stimulating soluble guanylate cyclase (sGC), promotes vasodilatation and improves ventricular function, contributing to hemodynamic optimization in patients with heart failure. While ACE inhibitors have been shown to prolong survival.[xvii] Nevertheless, MD treatment aims to reduce mortality and improve quality of life.[xviii]

This clinical case highlights the importance of considering chronic cocaine consumption as a relevant etiological factor, given that in this young patient, arterial hypertension and possible coronary artery disease could be secondarily induced by such consumption. In the absence of an alternative etiology to explain the clinical picture, and considering the well-documented cardiovascular effects of this substance, the case was classified as cocaine- induced dilated cardiomyopathy.

This condition is a secondary form of DCM, due to chronic cocaine use, characterized by dilatation of ventricular cavities (mainly left ventricle), systolic dysfunction; in addition, it is secondary to catecholaminergic toxicity, fibrosis, ischemia, and myocarditis caused by this drug.

Importantly, in patients with cocaine-induced DCM, total abstinence can lead to significant recovery of ventricular function.[xix] Nevertheless, continued use increases the risk of progression to advanced heart failure, the development of life-threatening arrhythmias, and increased mortality from acute ischemic events.[xix]

Therefore, any young patient presenting with clinical findings compatible with heart failure and a history of cocaine use should be considered as a probable diagnosis and approached as heart failure with reduced LVEF. In addition, for adequate etiological characterization, it is essential to perform cardiac magnetic resonance imaging and cardiac catheterization if the exclusion of coronary artery disease is required. The recurrent challenge of dealing with cases with multiple possible etiologies is evident, in which the patient's therapeutic compliance and access to specialized studies can make the difference between clinical uncertainty and a precise therapeutic intervention.

Ethical aspects

The Helsinki Declaration and international ethical guidelines for health-related research were followed. Patient confidentiality was guaranteed, and an informed consent form was prepared, in which the patient authorized the use of their information and images for publication.

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