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Case Report Open Access

Dilated Cardiomyopathy in a Young Man with Illicit Cocaine Use: A Case Report

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Abstract

Introduction: Dilated cardiomyopathy (DCM) is a progressive disease of the cardiac myocytes, characterized by enlargement in the ventricle and contractile dysfunction in the presence of normal ventricular wall thickness. Alcohol, Cocaine consumption, and Sarcoidosis are associated with development of DCM.

Case report: 31 year old male patient, unemployed for 3 years, was admitted to the emergency department with a chief complaint of dyspnea. His condition worsened over the last three days. His vital signs were as follows: TA 115/75 mmHg, pulse rate 100 bpm, and respiratory rate 27 bpm, temperature 36.5°C. Previously suspected of having sarcoidosis, the patient had a history of alcohol and cocaine abuse. Alcohol and cocaine abuse lasts 7 years. Necrosis and perforation were remarkable in the nasal septum due to cocaine abuse. ST segment elevation of 2 mm in V2-4 leads without any reciprocal changes was noted in the electrocardiogram taken on presentation. Besides, there was 2 mm ST-segment depression in lead V6. Chest X-ray revealed cardiomegaly, marked pleurisy on the right, bilateral hilar congestion, and increased reticular density. Echocardiogram disclosed significantly dilated heart chambers, accompanied by mitral and tricuspid valve regurgitation. Left ventricular ejection fraction was measured at 25%. Imaging and pulmonary findings suggested that the development of DCM might be multifactorial, due to sarcoidosis and cocaine abuse.

Conclusion: Heart failure secondary to DCM should be included in the differential diagnosis of patients with dyspnea, especially in those with prominent risk factors such as substance abuse.

Keywords: Dilated cardiomyopathy; Cardiac failure; Cocaine; Drug abuse; Sarcoidosis; Dyspnea

Introduction

Dilated cardiomyopathy (DCM) is a myocardial dysfunction leading to heart failure where ventricular enlargement and systolic dysfunction are prominent [1]. DCM is usually idiopathic. However, it can also be seen with familial or specific cardiac and systemic disorders. The idiopathic form constitutes approximately 25% of all cases of congestive heart failure. The prevalence of idiopathic DCM has been estimated as 36 in 100,000. DCM is the most common type of cardiomyopathy presenting with systolic and diastolic dysfunction [2].

As stated by Japp et al. in their compilation, among the causes of DCM there are many factors such as amphetamines, cocaine, ethanol, toxins such as chemotherapeutic agents, amyloidosis, infiltrative diseases such as sarcoidosis, nutritional disorders, inflammatory and autoimmune diseases, and genetic history [3]. The outcome is usually grave, since up to 70% of the patients die in 5 years [1].

In alcohol abuse, myocardial damage due to ethanol toxicity or secondary malnutrition is reported to be the possible causes of the development of DCM [4]. Chronic cocaine use is the primary cause of DCM and myocardial infarction secondary to cocaine use also paves the way for the development of DCM [5].

As a result of systolic pump failure, exercise dyspnea, orthopnea, paroxysmal nocturnal dyspnea; and embolization findings as a result of ventricular dysfunction such as the acute neurological deficit, chest and/or back pain, hematuria, and pulseless cyanotic extremity may ensue. In addition, findings of ischemic heart disease may also be present [2].

Treatment is directed to correct underlying causes while relieving the patient's symptomatology. Therefore in most cases, the management is limited to the treatment of heart failure. Corticosteroids, azathioprine, and anti-thymocyte globulin are no longer used. Antiviral drugs are of no benefit. Temporary or permanent pacemakers may be needed to treat cardiac rhythm disorders. Patients with DCM are usually candidates for heart transplantation because the prognosis is poor [1].

Case Report

31-year-old, 190 cm tall, a fit-looking male patient who weighed 84 kg was admitted to the emergency department with a chief complaint of shortness of breath. The patient had been dyspneic for about six months and the condition gradually worsened, so that the patient's daily activities were restricted in the last two weeks. Dyspnea was noted even while resting in the last three days; therefore, coupled with decreased oral intake, the patient had been admitted to various hospitals. He had a medical history of abdominal surgery at the age of

13 years due to a penetrating injury. In previous examinations, sarcoidosis was suspected. In his family history, his mother and father both had the chronic obstructive pulmonary disease and coronary artery disease. He was a cigarette smoker with a history of 20 packages year, regular daily alcohol for 7 years, and use of cocaine *via* nasal inhalation. Necrosis and perforation were detected in the midline of the nasal septum of the patient due to use of cocaine. The patient had no known allergies and no regular medication. He was a high school graduate without a regular occupation and was unemployed for 3 years. At baseline examination, blood pressure was 115/75 mmHg, heart rate was 100 bpm, respiratory rate was 27 bpm, the temperature was 36.5°C, and transcutaneous SaO_2 was found as 91 without oxygen support. Physical examination revealed his general condition as moderate, oriented, cooperative, conscious, and with a Glasgow coma scale of 15. His personal care was poor. Breath sounds were diminished on the right base, diffuse rhonchi were audible, and expiration was prolonged. The patient was tachypneic and was using accessory respiratory muscles. S_1 and S_2 sounds were audible in cardiac auscultation. He had withdrawal symptoms such as sweating, nausea, and palpitations as he had no intake of alcohol and cocaine for the last 3 days. His pupils were miotic and there was no light reflex. Other system examinations were normal.

Electrocardiography (Figure 1) showed 2 mm ST-segment elevation in leads V2-V4 without any reciprocal changes. There was 2 mm ST-segment depression in lead V6. His cardiac enzymes and blood tests including liver function tests, renal function tests, and complete blood counts were within normal limits. BNP and NT-pro BNP tests used in the diagnosis of heart failure could not be conducted as they were not available in our hospital. Chest radiography (Figure 2) revealed cardiomegaly and increased diffuse opacity, followed by computed chest tomography (Figure 3) revealing marked pleural effusion at the base of a right side, bilateral hilar congestion, and increased reticular density, as well as increased cardiothoracic index. Echocardiographic examination revealed significantly dilated cardiac chambers accompanied by mitral and tricuspid valve failure. Left ventricular ejection fraction was measured at 25%. The patient was thought to have pulmonary and cardiac involvement with the present imaging findings. It was determined that the volume load was increased through physical examination, imaging, and tests. When these findings were combined with the patient's history, it was thought that the patient had progressive heart failure and that the stage of heart failure had reached New York Heart Association (NYHA) Functional Classification III to IV in the last few weeks. The patient was admitted to the coronary care unit with the diagnosis of DCM. One or more among DCM etiologies of sarcoidosis, alcohol and cocaine use, and cocaine-related ischemic heart disease were thought to be responsible. Definitive etiology could not be determined as endocardial biopsy was not conducted in our hospital. Symptoms of heart failure improved within 72 hour under medical treatment with angiotensin-converting enzyme inhibitor and diuretic. The patient who prescribed antipsychotic drug was directed to receive psychosocial support. He was a candidate for heart transplantation.

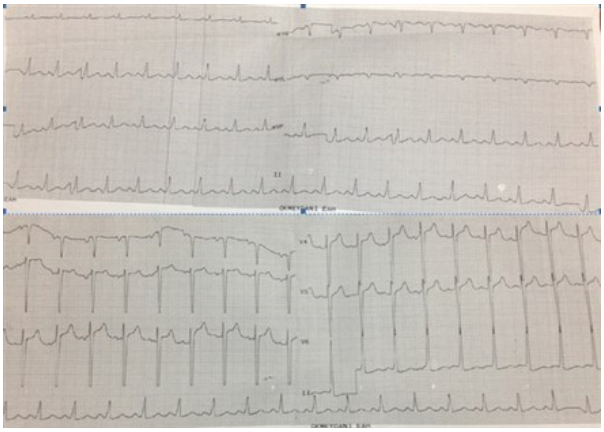

Figure 1: Electrocardiography.

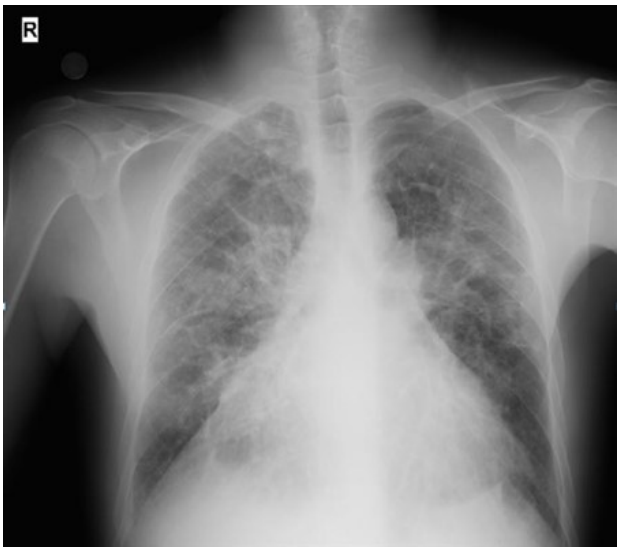

Figure 2: Chest radiography.

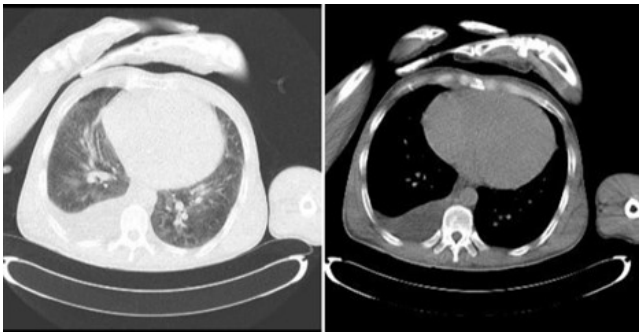

Figure 3: Computed Chest Tomography.

Discussion

The complaints of the patient admitted to the emergency department with respiratory distress were initially thought to be due to restrictive pulmonary disease. The patient was oxygenated and intravenous fluid treatment was started due to poor oral intake during the recent days. Detailed medical history was obtained revealing the history of chronic alcohol and cocaine use. After this stage, chest radiography followed by computed tomography was performed where cardiomegaly and marked pleural effusion were detected in the patient. Heart failure treatment protocol was thereupon initiated. Failure to consider cardiac insufficiency in the foreground due to the patient's young age delayed the primary treatment and the applied 500 ml isotonic fluid therapy increased the patient's existing respiratory distress. Although anticipated mostly in infants and in the elderly patient group, cardiac insufficiency can also be observed in younger patients depending on the risk factors. For the present case, these risk factors were identified as sarcoidosis, cocaine, and alcohol use.

In this case, cocaine was being used by nasal inhalation. Perforation was detected in the midline of the nasal septum of the patient due to the use of cocaine. Cocaine, and crack cocaine as an effective form thereof, are absorbed through all mucous membranes of the body, can be inhaled through the nasal passage, injected, or smoked in the form of cigarettes [6]. Hemodynamic changes associated with the sympathetic stimulation by cocaine in the body increase myocardial oxygen demand. Chronic cocaine use may cause recurrent attacks of coronary spasm and hypertensive crises, which may result in endothelial damage, coronary artery dissection, and acceleration of atherosclerosis [7,8].

Acute cocaine poisoning reduces myocardial contractility and ejection fraction and increased left ventricular end-diastolic pressure and endsystolic volume. Long-term cocaine use is associated with left ventricular hypertrophy and prolonged deceleration time. However, the pathophysiology of cocaine-related cardiomyopathy is not clear. Contributing factors in cocaine-related cardiomyopathy include the blocking of sodium and potassium channels in the myocardium, changes in the uptake of calcium ions, myocardial inflammation with necrosis and fibrosis, hypertrophy of the left ventricle, changes in gene expression, and concurrent alcohol consumption [9].

According to the study by Bhargava et al., it was reported that catecholamine increase and oxidative stress due to cocaine use accelerated the formation of free oxygen radicals and this contributed to cocaine-related left ventricular dysfunction [10].

In this study, the patient used cocaine as well as chronic alcohol. In addition to being one of the factors of cardiac insufficiency, alcohol also potentiates the impact of cocaine. This association might be among responsible factors for the development of the patient's progressive heart failure. In the studies, it was found that coca ethylene, which is the metabolite of cocaine when taken with alcohol, was more cardiotoxic than the main compound [11].

As the patient was suspected of having sarcoidosis, it was thought that his cardiac insufficiency might also be caused by myocardial involvement due to sarcoidosis. In a presentation by Meyer et al. where myocardial sarcoidosis cases were introduced, it was emphasized that myocardial involvement might occur up to 25% of patients diagnosed with sarcoidosis and that sarcoidosis should be kept in consideration especially in young patients with cardiomyopathy [12].

Conclusion

In this case report, we wanted to emphasize that heart failure and DCM should be considered in the differential diagnosis in even young patients presenting with shortness of breath. Detailed work-up of the respiratory and circulatory systems at an early stage would have a significant impact in diagnosis and treatment, and an elaborate history can also be of vital importance.

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