



# Cocaine-Induced Cardiomyopathy? Think Again: A Case of Noncompaction Cardiomyopathy

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## Background

Non-compaction cardiomyopathy is a rare congenital disorder characterized by a compacted epicardial layer and a noncompacted endocardial layer with trabeculations. We present a unique case of a patient presenting with heart failure symptoms who had evidence of non-compaction cardiomyopathy.

## Case Presentation

A 26-year-old male with past medical history of nicotine dependence, history of cocaine use presents with heart failure symptoms. Specifically, he was presenting with bilateral leg swelling and shortness of breath. Family history is significant for heart failure for dad in his 40s. Physical exam was notable for bilateral leg swelling. NT-proBNP was elevated at 6524. High-sensitivity troponin and TSH were within normal limits. Urine drug screen was positive for cocaine. Chest X-Ray revealed cardiomegaly. Transthoracic echocardiogram was done which revealed an LVEF of 20-25% with severe LV dilation, moderate pulmonary hypertension.

## Decision-Making

Initially it was thought that the CHF was secondary to cocaine use. Patient was started on Lasix as well as GDMT with Entresto, Farxiga, Toprol-XL, and Aldactone and was discharged. However, a few months later, the patient had cardiogenic shock in the setting of heart failure exacerbation complicated by congestive hepatopathy and respiratory failure. A repeat echocardiogram revealed LVEF < 20%, grade III diastolic dysfunction, moderate pericardial effusion, and a round mobile echodensity attached to the outer layer of pericardium located at the apex and within the pleural space.

## Noncompaction Cardiomyopathy on Imaging

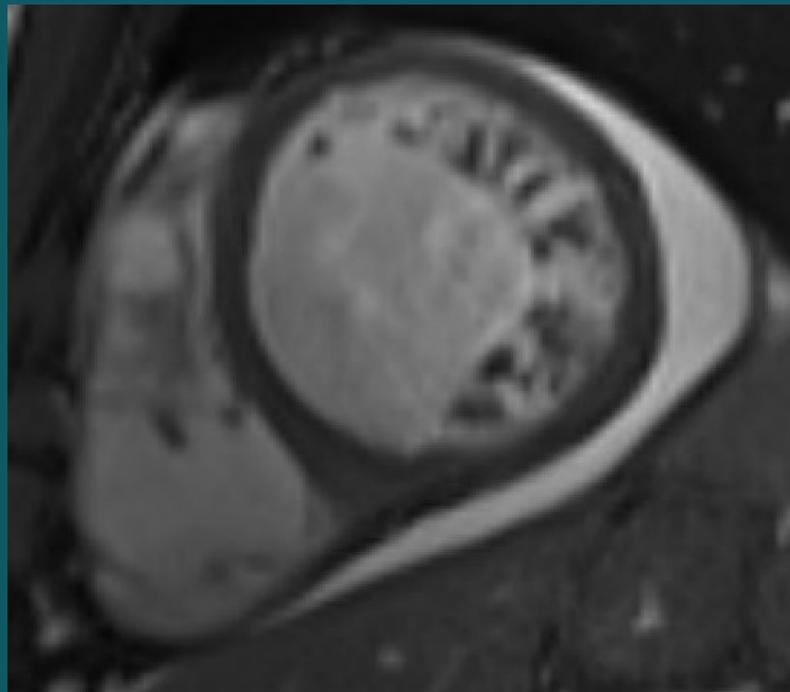


Figure 1: Cardiac MRI revealing noncompacted layer with trabeculations

## Decision-Making Continued

Right heart catheterization revealed severe biventricular failure and cardiogenic shock. Patient was started on inotropic agent milrinone and diuretic agent Lasix drip. Patient started to improve and slowly transitioned to GDMT. Nonischemic cardiomyopathy workup including ferritin, anti-CCP, and RF were within normal limits. Cardiac MRI were done which revealed evidence of noncompaction with trabeculations as well as late gadolinium enhancement concerning for active or prior pericarditis with moderate to large pericardial effusion. Genetic testing was positive for the titin mutation.

## Conclusions

The prevalence of noncompaction cardiomyopathy is about 0.014% to 0.26%. In this case, while the patient used cocaine, it was not convincing that cocaine was the primary etiology of his heart failure symptoms. In a young patient, it is important to consider hereditary causes of cardiomyopathy in the differential. Getting the cardiac MRI and genetic testing was necessary to establish the etiology.

## References

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## Disclosure Information

Nothing to disclose