Abstract: P1416

A rare case of dilated cardiomyopathy associated with cocaine use. The role of speckle tracking echocardiography in assessing myocardial function

Authors:
M G Brutaru¹, D M Dorobantu¹, A Calin¹, C Gingham¹, B A Popescu¹, I M Coman¹, ¹Institute of Cardiovascular Diseases Prof. C.C. Iliescu - Bucharest - Romania

Topic(s):
Tissue Doppler, Speckle Tracking and Strain Imaging

Citation:
European Heart Journal - Cardiovascular Imaging (2019) 20 (Supplement 1), i990

Introduction: Cocaine is a drug often associated with myocardial infarction, dilated cardiomyopathy (DCM), arrhythmias or hypertension. Several mechanisms have been proposed to contribute to the cardiac complications of cocaine use, such as coronary vasoconstriction, oxidative stress, and increased platelet aggregation and thrombus formation.

Case description: We present the case of a 32 year old man who came to our clinic for reevaluation, with a recent history of atypical chest pain and fatigue. He had been diagnosed a month previously with DCM, with a left ventricular ejection fraction (LVEF) of 30%. Of note, the coronary arteries were normal. The patient was a smoker for and consumed cocaine, designer drugs and alcohol, occasionally, for the last 8 years. He stopped all drug use when first diagnosed.

The physical examination was normal and the ECG showed left bundle branch block (LBBB). During hospitalization he had an episode of angina, with subtle ischemic changes, as shown by biphasic inferior T waves, different from baseline and not concordant with the QRS complex.

Echocardiography showed diffuse mild left ventricular hypokinesia with a calculated LVEF of 44% (Panel A), septum dyssynchrony due to LBBB and mild mitral regurgitation. The basal inferoseptal and basal inferior segments appeared more pronouncedly hypokinetic at the visual assessment, and in the light of the ECG changes a segmental strain analysis was performed. This showed early longitudinal lengthening (Panel B, yellow arrow), early shortening (panel B, blue arrow), and postsystolic shortening after aortic valve closure (Panel B, red arrow). The global longitudinal strain was reduced at -13.2%. Additionally, the segments with decreased strain were not localized to one coronary territory, but rather diffuse.

The patient is treated with a beta-blocker, ACE inhibitor, spironolactone and aspirin. After one month being drug free there has been a significant improvement in the symptoms and also in the LVEF (44% vs 30%). He is scheduled to undergo a CMR and also an echocardiographic reevaluation at 3 months to assess whether any long-lasting effects might be observed.

Discussions: This case highlights the cardiac toxic effects associated with cocaine in a young, otherwise healthy man. An ischemic cause for the cardiomyopathy should be considered, even with normal coronary arteries, since vasoospasm or transient coronary thrombus may still lead to myocardial infarction. Visual assessment of wall motion abnormalities has limitations, especially in the presence of LBBB. Speckle tracking imaging showed that the wall motion abnormalities did not correspond to a coronary territory, being diffuse and with a pattern concordant with the septum dyssynchrony.

Conclusions: Cocaine abuse can lead to myocardial damage either by direct action or ischemia. CMR remains the gold standard in evaluating such patients, but in situations where it is not readily available, echocardiography can be used.
Abstract: P1416
A rare case of dilated cardiomyopathy associated with cocaine use... the role of speckle tracking echocardiography in assessing myocardial function.

Introduction: Cocaine is a drug often associated with myocardial infarction, dilated cardiomyopathy (DCM), arrhythmias or hypertension. Several mechanisms have been proposed to contribute to the cardiac complications of cocaine use, such as coronary vasoconstriction, oxidative stress, and increased platelet aggregation and thrombus formation.

Case description: We present the case of a 32 year old man who came to our clinic for reevaluation, with a recent history of atypical chest pain and fatigue. He had been diagnosed a month previously with DCM, with a left ventricular ejection fraction (LVEF) of 30%. Of note, the coronary arteries were normal. The patient was a smoker for and consumed cocaine, designer drugs and alcohol, occasionally, for the last 8 years. He stopped all drug use when first diagnosed.

The physical examination was normal and the ECG showed left bundle branch block (LBBB). During hospitalization he had an episode of angina, with subtle ischemic changes, as shown by biphasic inferior T waves, different from baseline and not concordant with the QRS complex.

Echocardiography showed diffuse mild left ventricular hypokinesia with a calculated LVEF of 44% (Panel A), septum dyssynchrony due to LBBB and mild mitral regurgitation. The basal inferoseptal and basal inferior segments appeared more pronouncedly hypokinetic at the visual assessment, and in the light of the ECG changes a segmental strain analysis was performed. This showed early longitudinal lengthening (Panel B, yellow arrow), early shortening (panel B, blue arrow), and postsystolic shortening after aortic valve closure (Panel B, red arrow). The global longitudinal strain was reduced at ~13.2%. Additionally, the segments with decreased strain were not localized to one coronary territory, but rather diffuse.

The patient is treated with a beta-blocker, ACE inhibitor, spironolactone and aspirin. After one month being drug free there has been a significant improvement in the symptoms and also in the LVEF (44% vs 30%). He is scheduled to undergo a CMR and also an echocardiographic reevaluation at 3 months to assess whether any long-lasting effects might be observed.

Discussions: This case highlights the cardiac toxic effects associated with cocaine in a young, otherwise healthy man. An ischemic cause for the cardiomyopathy should be considered, even with normal coronary arteries, since vasospasm or transient coronary thrombus may still lead to myocardial infarction. Visual assessment of wall motion abnormalities has limitations, especially in the presence of LBBB. Speckle tracking imaging showed that the wall motion abnormalities did not correspond to a coronary territory, being diffuse and with a pattern concordant with the septum dyssynchrony.

Conclusions: Cocaine abuse can lead to myocardial damage either by direct action or ischemia. CMR remains the gold standard in evaluating such patients, but in situations where it is not readily available, echocardiography can be used.