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New era in HF treatment-Medical management

Case Report: We present a case of a 35-year-old female who presented to the emergency room with severe dyspnea with orthopnea and angina she denied any personal or family history of cardiovascular disease, but reported two recent episodes of pneumonia for which she received empirical antibiotic treatment with Cefuroxime and Clarithromycin. Physical examination revealed an overweight patient, mild bibasilar crackles and systolic cardiac murmur over the mitral area. Her blood pressure was 190/100 mmHg and heart rate 120 BPM. Initial laboratory data showed elevated CK-MB and NT-proBNP, elevated liver enzymes and normal renal function. There were no particular findings on the surface ECG. The echocardiography revealed a dilated Left Ventricle (LV), severe systolic dysfunction (ejection fraction 20%) due to global hypokinesia, intraventricular dyssynchrony despite a narrow QRS complex on the surface ECG and moderate secondary mitral regurgitation. In this context, the optimal treatment for heart failure with reduced ejection fraction was initiated (at first perindopril then combination Sacubitril-Valsartan, Spironolactone, Furosemide, Metoprolol). On follow-up echocardiographic examinations, the ejection fraction gradually increased up to 45% at eight months examination. To clarify the cause of chamber dilation, a CMR was pursued which confirmed both left and Right Ventricle (RV) dilation (LV 171 ml/m2, RV 136 ml/m2) and severe systolic dysfunction. In addition, no edema or areas of focal myocardial fibrosis were noticed. The apical region of the LV was hypertrabeculated with a non-compacted/compacted myocardium radio of 2.2 in long axis views suggestive of non-compaction. No thrombus was seen. Also, we excluded an ischemic etiology considering the absence of coronary lesions, the diffuse hypokinesia on echocardiography and the lack of ischemic changes on CMR. In addition, acute myocarditis was unlikely due to the non-suggestive CMR aspect. Finally, the 24-hour ECG monitoring showed very rare ventricular extrasystoles accounting for only 0.5% of total ventricular beats and no tachyarrhythmia, making a diagnosis of tachycardiomyopathies improbable. In conclusion, myocardial non-compaction may be the expression of a genetic cardiomyopathy or may be the phenotypic appearance of other causes of left ventricle dysfunction. The reversibility of the disease in our patient does not support the diagnosis of genetic non-compaction cardiomyopathy. The patient was nonetheless programmed for genetic testing given the psychological burden that this diagnosis implies. So far, we were not able to determine a trigger for the reversible ventricular dysfunction in this patient. A question remains whether the optimal medical treatment for heart failure with reduced ejection fraction could nowadays completely reverse even a genetic form of non-compaction.

Biography

Oana Gheorghe-Fronea is Head of the department in Carol Davila University of Medicine and Pharmacy.