



HEART FAILURE WITH RECOVERED LEFT VENTRICLE EJECTION FRACTION. CASE REPORT

INSUFICIENCIA CARDIACA CON FRACCIÓN DE EYECCIÓN DEL VENTRÍCULO IZQUIERDO RECUPERADA. REPORTE DE CASO

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ABSTRACT

Introduction: Heart failure with recovered ejection fraction is defined as the improvement of left ventricular systolic function (LVEF) to values $\geq 40\%$, after having presented a reduced LVEF ($< 40\%$) at a previous time and $\geq 10\%$ absolute improvement in LVEF. It is a chronic and dynamic condition with a high risk of relapse and deterioration, so close monitoring and optimal medical therapy are essential for its management. **Clinical case:** A case is presented of a young male patient with dilated cardiomyopathy with severe left ventricular systolic dysfunction, who with neurohormonal therapy for heart failure, showed rapid clinical recovery and improvement of ejection fraction and left ventricular remodeling. **Conclusion:** Further studies are needed to better understand this condition and its long-term treatment.

Keywords: Heart failure; Pharmacotherapy; Dilated cardiomyopathy; Ventricular remodeling. (Source: MESH-NLM)

RESUMEN

Introducción: La insuficiencia cardíaca con fracción de eyección recuperada se define como la mejoría de la función sistólica del ventrículo izquierdo (FEVI) hasta valores $\geq 40\%$, después de haber presentado una FEVI reducida ($< 40\%$) en un momento previo y con un incremento absoluto de la FEVI $\geq 10\%$. Es una condición crónica y dinámica con alto riesgo de recaída y deterioro, por lo que, el seguimiento estrecho y la terapia médica óptima son fundamentales para su manejo. **Caso clínico:** Se presenta el caso de un paciente varón joven con cardiopatía dilatada con grave disfunción sistólica del ventrículo izquierdo, que con terapia neurohormonal de falla cardíaca, presenta una rápida recuperación clínica y mejoría de la fracción de eyección y remodelado ventricular izquierdo. **Conclusión:** Se requieren más estudios para comprender mejor esta condición y su tratamiento a largo plazo.

Palabras clave: Insuficiencia cardíaca; Farmacoterapia; Cardiomiopatía dilatada; Remodelación ventricular. (Fuente: DeCS- BIREME)

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INTRODUCTION

Heart failure with recovered ejection fraction is defined as the improvement of left ventricular systolic function (LVEF) to values $\geq 40\%$ after having presented a reduced LVEF ($< 40\%$) at a previous time and with an absolute increase in LVEF $\geq 10\%$. Although it is associated with lower mortality and hospitalization than heart failure with reduced or preserved ejection fraction, it remains a chronic and dynamic condition with a high risk of relapse and deterioration. It is essential to maintain close follow-up and appropriate medical therapy⁽¹⁻³⁾.

The evidence related to the improvement of LVEF is still limited, so the objective of this report was to describe the clinical characteristics, comorbidities, auxiliary examinations and analyze the evolution of LVEF in a patient with heart failure with recovered ejection fraction.

CLINICAL CASE

37-year-old male from Lima-Peru, cargo transportation supervisor. Male without a history of high blood pressure, diabetes, ischemic heart disease, or arrhythmias. Mother died suddenly at age 33 with an apparent cardiac cause by autopsy, maternal uncle with heart failure, maternal uncle with a cardiac pacemaker. The patient came to the outpatient clinic with an illness of 4 months due to lower limb edema, palpitations, 3-pillow orthopnea, and progressive exertional dyspnea

until reaching mild exertion. He is found with BP 110/70 mmHg, HR 120 bpm, RR 18x', normal temperature. Weight 95kg, size 170cm. Positive jugular engorgement, edema +++/++++. The cardiovascular examination showed displaced tip shock, rhythmic, tachycardic heart sounds, presence of third heart sound, and mitral II/VI systolic murmur. In addition, he had crackles and decreased murmur at the lung bases. The rest of the exam was non-contributory. The blood count, blood glucose, kidney function, serum electrolytes, thyroid and liver profile were within normal values. ProBNP was 953 pg/ml.

The electrocardiogram showed sinus tachycardia, left axis, left atrial enlargement, left ventricular hypertrophy, poor r-wave progression in the precordials, and QRS of 100 ms. The chest x-ray revealed cardiomegaly and pulmonary congestion. Holter monitoring showed sinus rhythm and simple low-load ventricular extrasystoles. The baseline echocardiogram showed dilated cardiomyopathy with severely depressed left ventricular systolic function (EF 26%), moderately depressed right ventricular systolic function (FAC 23%, TAPSE 14mm), grade III diastolic dysfunction, functional grade II/VI mitral regurgitation, high probability of pulmonary hypertension (PSAP 44mmHg, TAP 72ms). (Figure 1).

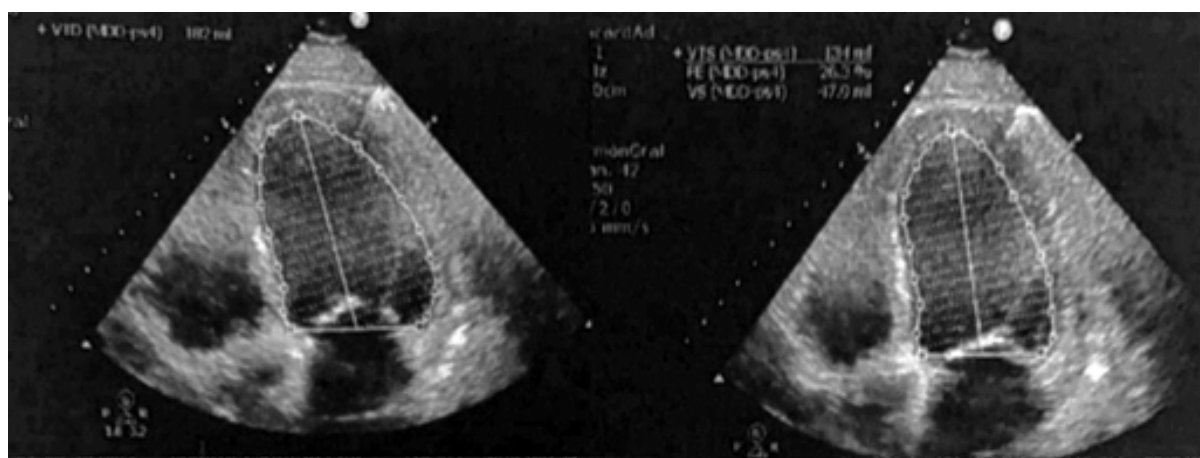


Figure 1. Baseline echocardiogram. Shows severely depressed systolic function.

A magnetic resonance study was performed that showed non-ischemic dilated cardiomyopathy, with severe biventricular systolic dysfunction (LVEF 18% and RVEF 22%), severe biventricular dilation, as well as intramyocardial linear fibrosis equivalent to 5% of the myocardial mass located in the lateral wall, anterior and septal of the left ventricle, and, in the lateral and inferior wall of the right ventricle. Coronary CT angiography showed coronary arteries without significant alterations and a calcium score of 0.

Patient was managed on an outpatient basis due to refusal and fear of being hospitalized during a health emergency due to Covid-19. Treatment was started with spironolactone 25mg/day, furosemide 40mg bid, sacubitril/valsartan 50mg bid and water restriction to 1000ml/day. Telephone follow-up was carried out after two weeks, reporting adherence to treatment, with improvement in orthopnea and edema, but still with dyspnea on mild exertion. Bisoprolol 2.5 mg/day was added. He attended an in-person appointment after a month, showing a 10kg weight loss, BP 100/60 mmHg,

HR 82bpm, negative jugular engorgement, lower limb edema +/++++, absence of third sound. Control echocardiogram showed LVEF 30-33% and improvement in LV filling pressures (figure 2). The dose of sacubitril/valsartan was increased to 100 mg bid, bisoprolol 5 mg/day. The dose of furosemide was reduced to 40 mg/day, and spironolactone was continued. Water restriction of up to 1500ml/day is also indicated.

Three months later, it is evident that he tolerates medication. He is found to have BP 110/70 mmHg, HR 70bpm, absence of edema, tolerance of recumbency, and functional class improvement to NYHA II. A control echocardiogram was performed, showing LVEF 50% with normal diastolic function, even with significant dilation of the left ventricle (Figure 3). Medication was maintained except for sacubitril/valsartan, which was suspended as the patient could not afford it. It was changed to enalapril 10 mg bid, with good adherence and tolerance.

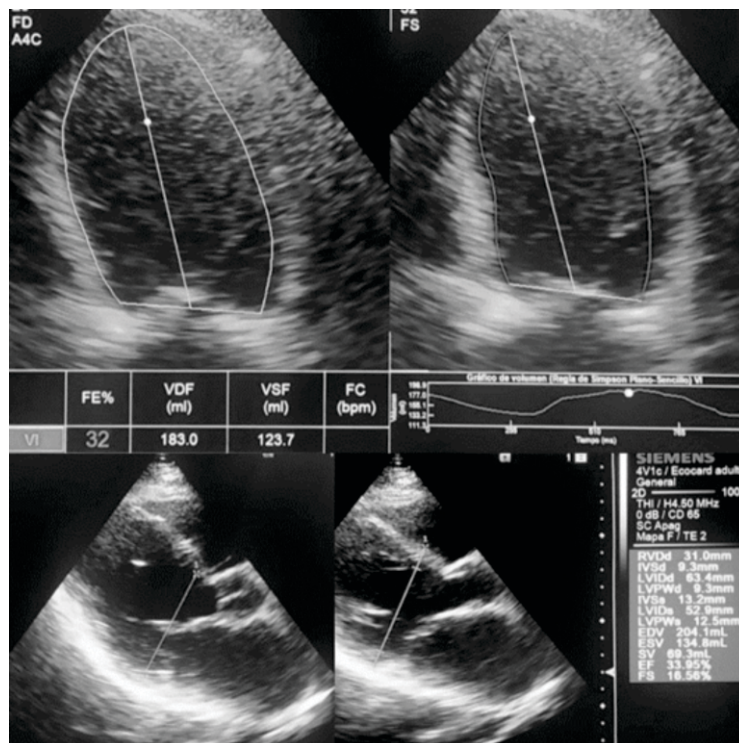


Figure 2. Echocardiogram one month after treatment. Presents an increase in FEVI to 33%. Additionally, it was found: TAPSE 20mm, E/e'15, PSAP 38mmHg.

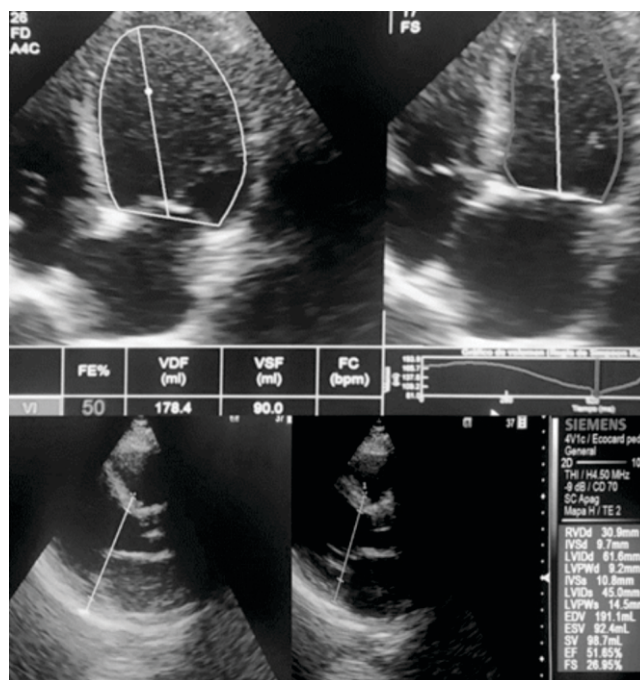


Figure 3. Echocardiogram after the third month of treatment. Shows improvement in LVEF to 50% even with dilation of the left cavities. Also: E/e’6. PSAP 35mmHg.

The echocardiogram after one year showed an LVEF of 65%, normal diastolic function and no dilation of the left ventricle (figure 4). The follow-up electrocardiogram no longer showed signs of left atrial enlargement or left ventricular hypertrophy (figure 5). Functional class improvement was observed through a

stress test performed with a beta blocker, in which it reached 70% of the maximum heart rate and 9.1 METS. It was decided to suspend furosemide, reduce the dose of bisoprolol to 2.5 mg, and continue with the rest of the medications at established doses. The control showed a good response after one month.



Figure 4. Echocardiogram one year after treatment. Preserved LVEF and decreased left ventricular volume are seen.

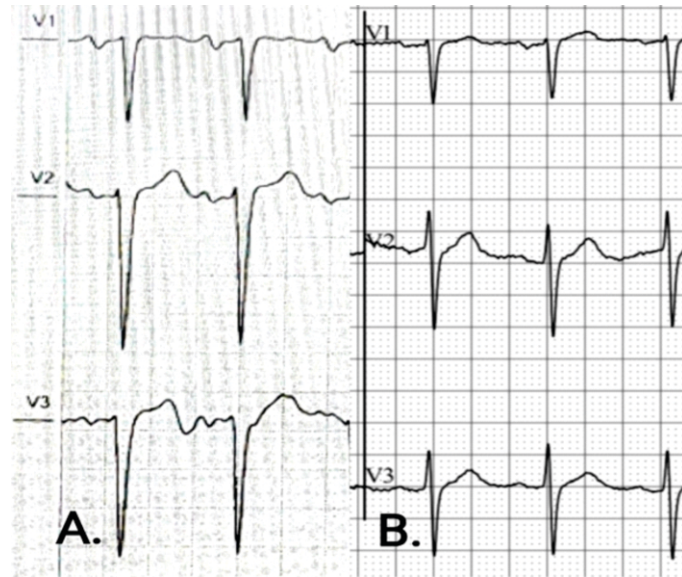


Figure 5. A. Baseline ECG. B. ECG at one year. Both taken at 10mm/mV and 25mm/s. Changes in voltage are evident. Mainly lower amplitude in the P wave and S wave, improved R wave progression and less wide QRS complex.

DISCUSSION

Dilated cardiomyopathy is defined as dilation of the left ventricle (or both), associated with depressed ejection fraction in the absence of hypertensive heart disease, congenital heart disease, coronary artery disease, and valvular disease. It is the most common indication for heart transplant. Its etiology can be idiopathic, genetic, viral, autoimmune and toxic (alcohol, drugs). In 50% of cases it is considered idiopathic. Dilated heart disease has familial origin in 20 to 50% and it should be considered if it is present in two or more first-degree relatives or in the case of a relative with sudden death at an early age⁽⁴⁻⁶⁾.

Dilated heart disease frequently presents as congestive heart failure. To reach the diagnosis, in addition to a detailed history and a thorough physical examination, it is essential to perform a transthoracic echocardiogram, since it allows estimating the ejection fraction, estimating ventricular volumes and ruling out other causes⁽⁶⁾. Likewise, magnetic resonance also has an important role, since it allows identifying the pattern of myocardial fibrosis in late gadolinium enhancement, which is associated with advanced stages of the disease⁽⁷⁾. Mortality due to dilated heart disease is estimated between 20 to 30% per year, and can reach 50% 2 years

after diagnosis. Death is sudden in approximately half of the cases. Optimal medical treatment with angiotensin-converting enzyme inhibitors, angiotensin II receptor blockers, dual neprilysin and angiotensin receptor inhibitors, beta-blockers, mineralocorticoid receptor antagonists, and recently, sodium-glucose cotransporter inhibitors type 2 has significantly reduced mortality in these patients^(2,3,6). In the case presented, our patient showed rapid clinical recovery with neurohormonal management of heart failure, showing reverse remodeling in the left ventricle and improvement in ejection fraction.

Entity known as heart failure with recovered LVEF, which has different characteristics in etiology, comorbidities, response to treatment and prognosis compared to failure with reduced or preserved LVEF. Patients with heart failure with recovered LVEF have an improved quality of life and a lower rate of hospitalization and mortality⁽¹⁾. Although the patient's recovery could also be the result of the natural history of a self-limiting background myocardial disease, the current recommendation is to continue treatment due to the high risk of relapse, as well as close monitoring of the patient⁽⁸⁻¹⁰⁾.



CONCLUSION

It is not uncommon for patients with dilated heart disease to show improvement in LVEF with neurohormonal treatment, even when myocardial dysfunction has been severe; however, information on the management of heart failure with recovered

LVEF is still limited. The risk of relapse is high. Therefore, it is advisable to maintain neurohormonal therapy for heart failure. Experimental studies are required on new therapeutic targets that allow lasting remission of heart failure.

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