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Levosimendan as an Adjunctive Treatment to Sinus Rhythm Restoration in Arrhythmia-Induced Cardiomyopathy

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Abstract

Arrhythmia-induced cardiomyopathy is characterized by ventricular remodeling and impaired contractile function due to persistent or paroxysmal atrial or ventricular arrhythmias, leading to increased heart rate. This condition is reversible upon restoring sinus rhythm. Levosimendan, a drug that enhances contractility without raising myocardial oxygen consumption, shows promise in augmenting ventricular function recovery when combined with sinus rhythm restoration. We present the case of a 68-year-old with atrial flutter who developed LV performance deterioration due to tachyarrhythmia, in the form of arrhythmia-induced cardiomyopathy, causing acute heart failure. Levosimendan, adjuntive to sinus rythm restoration, enhanced myocardial recovery.

Introduction

Arrhythmia-induced cardiomyopathy is a clinical entity characterized by ventricular remodeling and impaired contractile function, secondary to the presence of persistent or paroxysmal atrial or ventricular arrhythmias, resulting in an elevated heart rate. It is characterized by its reversibility following the restoration of sinus rhythm [1]. Levosimendan, on the other hand, is a drug that enhances contractility without increasing myocardial oxygen consumption by sensitizing myofilaments to intracellular calcium. This molecule presents potential as an enhancer of ventricular function recovery in conjunction with the restoration of sinus rhythm in arrhythmia-induced cardiomyopathy.

Case Presentation

We present the case of a 68-year-old male patient, a smoker, hypertensive, and dyslipidemic. He was admitted due to an acute inferior ST-elevation myocardial infarction (Killip I), and a primary angioplasty was performed on the mid-right coronary artery, which was the only diseased vessel, with a good angiographic result. Echocardiography revealed a non-dilated left ventricle with preserved ejection fraction and severe hypokinesia in the middle and apical inferior segments. The patient experienced typical counterclockwise atrial flutter with 2:1 conduction, as the only incidence during hospitalization, prior to discharge. The initial strategy involved heart rate control and subsequent programmed cavotricuspid isthmus ablation.

A month later, the patient presented to the Emergency Department with dyspnea on minimal exertion and palpitations. A new echocardiogram showed a dilated left ventricle (65mm) with severely depressed ejection fraction (25%) and a dysfunctional right ventricle with a TAPSE of 12 (Figure 1).

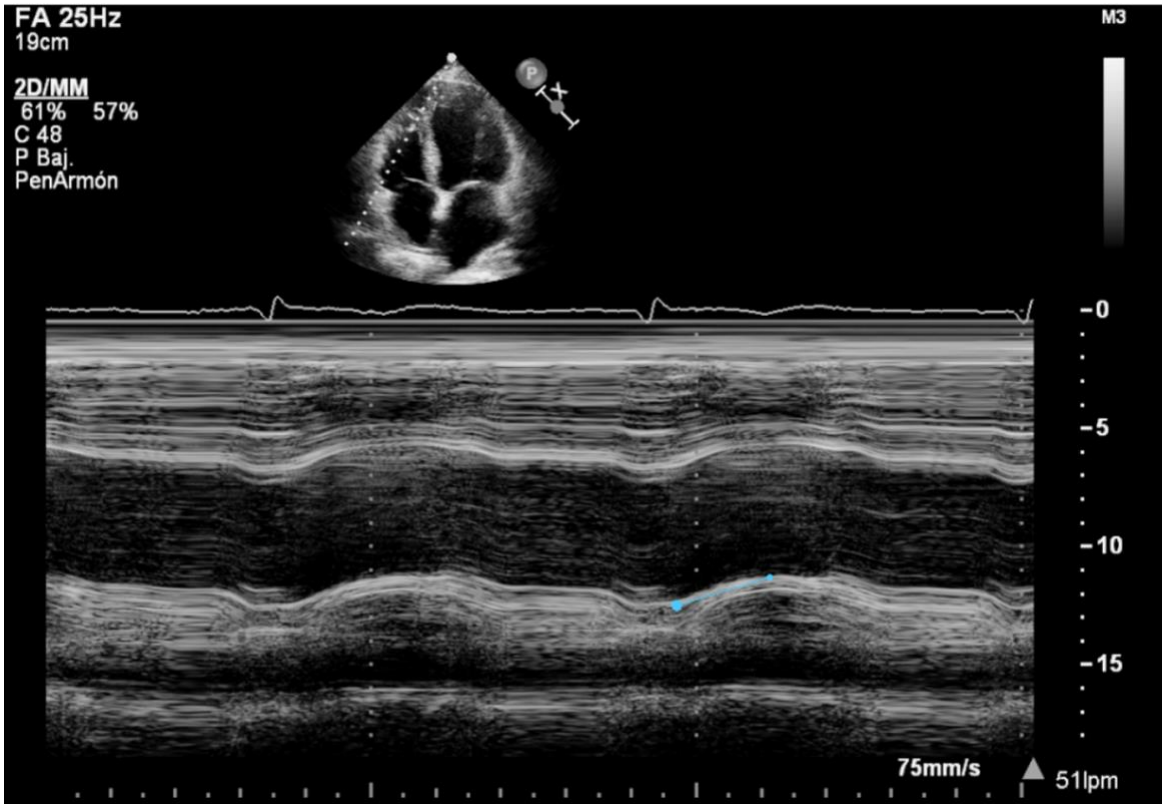

Figure 1: TAPSE of 12 mm.

Immediate admission to the Acute Cardiac Care Unit was arranged and synchronized electrical cardioversion at 100 J was performed under sedoanalgesia, effectively restoring sinus rhythm at 60 beats per minute. After 8 hours in sinus rhythm, the patient developed clinical signs of low cardiac output, with distal coolness, clouded consciousness, and oliguria. As a result, a 24-hour infusion of Levosimendan was administered, which the patient tolerated at maximum doses.

Following the treatment, signs of low cardiac output disappeared. A repeat echocardiogram showed preserved biventricular systolic function. The patient was discharged in sinus rhythm, without signs of heart failure, with a scheduled appointment for cavotricuspid isthmus ablation.

Discussion

Arrhythmia-induced cardiomyopathy is an uncommon etiology of ventricular dysfunction, where the temporary persistence of a high heart rate leads to increased filling pressures, reduced cardiac output, and increased peripheral vascular resistance. Moreover, tachyarrhythmia causes diastolic dysfunction by impeding proper myocardial relaxation, resulting in what Langer GA et al. described as myocardial contracture [4].

In this state of inadequate myocardial relaxation, the sarcolemma's sodium-calcium exchanger is incapable of restoring diastolic calcium levels, leading to elevated cytosolic calcium levels that perpetuate the state of contracture. This situation results in ventricular remodeling, leading to dilation and systolic dysfunction. Additionally, the activation of compensatory neurohormonal mechanisms contributes to remodeling and altered contractility [2].

Due to the pathophysiology of ventricular dysfunction induced by arrhythmias, the use of classical catecholaminergic inotropic drugs seems inappropriate as increased contractility would come at the expense of increased myocardial oxygen consumption and cytoplasmic calcium content, perpetuating the state of contracture in a myocardium with limited contractile reserve. On the other hand, Levosimendan is an inodilator drug that, through its binding to troponin C, stabilizes its association with calcium, facilitating the intracellular calcium utilization and redistribution, optimizing inotropic and lusitropic functions. Furthermore, it causes peripheral vasodilation and reduces afterload by inhibiting phosphodiesterase-III [5].

Conclusions

Based on the pathophysiology of arrhythmia-induced cardiomyopathy described previously and the pharmacological properties of Levosimendan, it is proposed as an adjunctive treatment to restore sinus rhythm, expediting myocardial recovery.

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