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Case Report

Levosimendan. A promising future drug for refractory cardiac failure in children?

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ABSTRACT

Intravenous positive inotropic agents play an important role in treating acute decompensation of patients with heart failure due to left ventricular systolic dysfunction. Levosimendan is a new positive inotropic agent having ATP-dependent potassium-channel opening, and calcium-sensitizing effects, which increases cardiac contractility and performance along with vasodilatatory action without increasing myocardial oxygen demand. We report a case of a 12-year-old girl with viral myocarditis, dilated cardiomyopathy, biventricular failure with severe left ventricular dysfunction, refractory to standard management, and who was successfully improved with levosimendan.

1. Introduction

Levosimendan is a new pyridazinone-dinitrile derivative with positive inotropic effects belonging to the category of ''inodilators'', which increases cardiac contractility and performance along with vasodilatatory action. It is a calcium-sensitizing drug, which causes vasodilatation by opening ATP-sensitive potassium channels without increasing myocardial oxygen demand.[1,2]

We present a case of viral myocarditis with dilated cardiomyopathy, biventricular failure, severe left ventricular dysfunction, and refractory to standard management, which successfully improved with levosimendan.

2. Case report

A 12-year-old girl, previously healthy, presented with moderate to severe grade fever since 10 days. Following this, the child developed abdominal pain and breathlessness, NYHA GRADE-1, since 7 days. Breathlessness increased progressively and on admission it was associated with orthopnea. The child received symptomatic treatment from a local doctor. The child had chest pain since 2 days and was referred to our hospital. There was no history of any previous cardiac problem, breathlessness, seizures, trauma, bluish discoloration, tubercular contact, or hospitalization. On admission, vital signs were as follows: pulse: 140/min, regular rhythm, low

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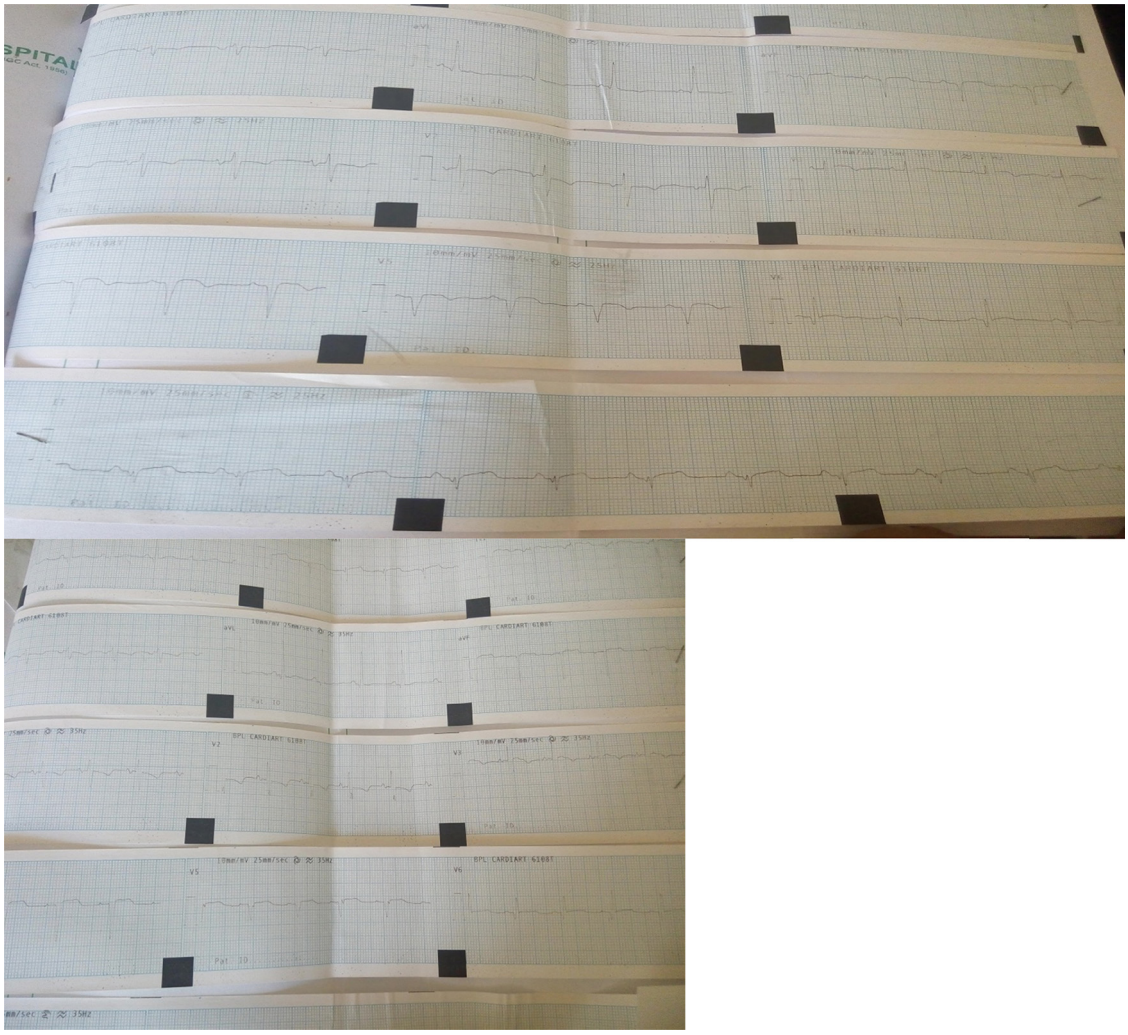

Figs. 1 and 2 – Nonspecific ST and T wave segments, suggestive of myocarditis.

volume, all pulses were felt but weak and thready; B.P: 110/84 mmHg; respiratory rate: 44/min; SpO_2: 96% at room air. Child had mild pallor, bilateral pitting pedal edema and raised JVP. Cardiovascular system examination revealed cardiomegaly and S3 gallop without any murmurs. Child had bilateral fine basal crepitations in chest with mild hepatomegaly. On admission, all routine blood and urine investigations were normal. Serum creatine kinase MB, Se. lactate, and Se. troponin were high and were 74 units/L, 4.5 mmol/L, and 2.3 ng/mL, respectively. ECG was suggestive of features of myocarditis. ECHO was done that was suggestive of dilated cardiomyopathy, and biventricular failure with severe left ventricular dysfunction with ejection fraction of only 15%.

Child was started on oxygen inhalation, iv fluids, dobutamine, Lasix, digoxin for DCM, and antibiotics and was monitored strictly. Carvedilol and enalapril were added on 2nd day as there were no signs of improvement. Cause of cardiomyopathy could never be established. On 2nd day, child developed supraventricular tachycardia and diltiazem was started. In view of ischemia heparin, aspirin and clopidogrel were started. As ejection fraction did not improve even after 3 days of dobutamine, levosimendan infusion was started and dobutamine was tapered and stopped. Patient improved gradually. On 6th day LVEF improved up to 35%. Heparin was stopped after 5 days and rest of the drugs were continued (Figs. 1–3).

3. Discussion

Main aim for reporting this case is that novel drug levosimendan can be used in refractory cardiac failure with low ventricular function.[3] Improvement after levosimendan was remarkable. Oxygen, catecholamines, inotropes, phosphodiesterase inhibitors, diuretics, and drugs for afterload reduction are still the basis for cardiac failure.

Levosimendan is a novel drug that acts as a myofilament Ca^{2+} sensitizer by acting on troponin with inotropic effects, increases myocardial performance without substantial changes in oxygen consumption, causes myocardial oxygen