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A cohort study to compare the benefits of Levosimendan vs Dobutamine for acute decompensated heart failure cases

Shyvin K S[a], Christopher Mathew [a],MohdRameez Mustafa [a], Aqib Raj [a],ArunVarghese[b], Mohammed AadalKhuraishi[a]

[a]*Departmentof Internal Medicine, Aster Dr Moopen's Medical College Hospital, Kerala,India*
[b]*Department of Community Medicine, Aster Dr Moopen's Medical College Hospital, Kerala, India*

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ABSTRACT
There is a need for medications that at least improve hemodynamics and alleviate symptoms without having a negative impact on survival since acute decompensated heart failure significantly increases morbidity and death.
Keywords:- Dobutamine, levosimendan, heart failure

I. METHODS

We assigned 52 patients who took treatment with acute decompensated heart failure and a left ventricular ejection fraction (LVEF) of 35 percent or less to comparing the efficacy and safety of intravenous levosimendan (12 µg/kg bolus in 10 min, followed by a dose of 0.1-0.2 µg/kg/ min for 24 hours) or dobutamine (5-10 mcg/kg/min IV for 24 hours).The primary end point was death from any cause.The randomization was done with EDGAR®

II. RESULTS

At 180 days, 13 (26%) of the levosimendan group's patients and 14 (28%) of the dobutamine group's patients experienced all-cause mortality. (hazard ratio, 0.91; 95% confidence interval, 0.74-1.13; P = .40). In comparison to the dobutamine group, the levosimendan group experienced higher reductions in B-type natriuretic peptide levels at 24 hours, which remained for 5 days. (P<.001 for all time points). For the other secondary end goals, there were no statistical differences between treatment groups (all-cause mortality at 31 days, number of days alive and out of the hospital, patient global assessment, patient assessment of dyspnea at 24 hours, and cardiovascular mortality at 180 days).

III. CONCLUSIONS

Levosimendan did not significantly lower all-cause mortality at 180 days or impact any secondary clinical outcomes, despite an early decrease in plasma B-type natriuretic peptide levels in patients in the levosimendan group compared to patients in the dobutamine group.

IV. DISCUSSION

Levosimendan

A pyridazone-dinitrile subsidiary is levosimendan whichcreates two basic impacts. Its fundamental design is to amplify cardiac contraction and this is accomplished by a pharmacological system known as calcium sensitization. It will not raise intracellular levels calcium quantities that are free. It attaches to cardiac troponin C in a calcium-dependent manner and settles troponin C. This results in actin-myosin cross-bridges, without expanding myocardial consumption of adenosine triphosphate (ATP). The contractility and cardiac efficiencyimproved enormously without expanding the total myocardial oxygen and energy prerequisites. The potential for arrhythmia is likewise diminished as total intracellular calcium levels are not raised. An extra advantage is that the stabilization effectis calcium dependant and levosimendanexerts it effects during systole; it will not influence the term of diastole thus ventricular relaxation is not debilitated. Therefore, satisfactory ventricular filling and optimal coronary perfusion happens. Levosimendan also causes venous, arterial and coronary vasodilation, presumably by opening ATP sensitive potassium channels in smooth muscle. Dose-dependent hypotension may also happen. Levosimendanis also of benefit in the setting of pulmonary vasoconstriction and right ventricular dysfunction and lessens pulmonary vascular resistance.

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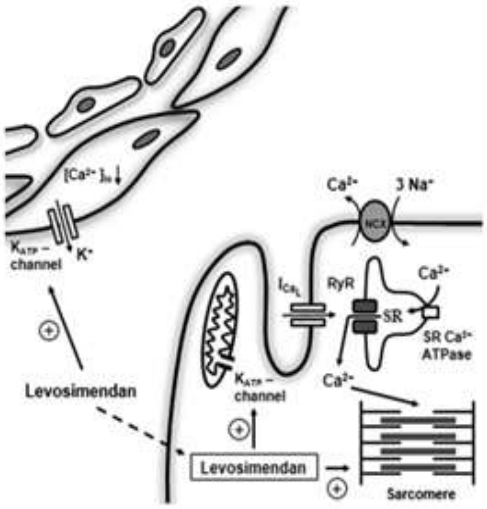

Figure 1

Triple mechanism of action of levosimendan. Levosimendan activates ATP-sensitive K+ (KATP) channels in vascular smooth muscle cells. The consequent hyperpolarization inhibits inward Ca2+ currents resulting in vasorelaxation. Additionally, levosimendan exerts a Ca2+ sensitizing effect in cardiomyocytes due to the interaction with cardiac troponin C. Activating of the mitochondrial KATP in the cardiomyocytes results in short- or long-term cardioprotection. KATP channel, ATP-sensitive K+ channel; NCX, sodium-potassium exchanger; ICaL, inward calcium current; SR Ca2+ ATPase, Sarcoplasmic reticulum calcium ATPase.

V. SIDE EFFECTS

Levosimendan is all around endured, with most adverse occasions (migraine & hypotension) being dose related and emerging from the vasodilatory actions of the medication. In order to avoid excessive hypotension, it could be reasonable to briefly stop milrinone and other vasodilators while administering this medication. The other side effects that are impending from trials include prolongation of remedied QT span and rarely, ventricular tachycardia. Nevertheless, it should be avoided in patients with Torsadesor some other strange rhythm.

Conflicts of Interests:- NIL. All relevant permissions received.

REFERENCES:-

[1]. Lombardi, C., Peveri, G., Cani, D., Latta, F., Bonelli, A., Tomasoni, D., Sbolli, M., Ravera, A., Carubelli, V., Saccani, N., Specchia, C., and Metra, M. (2020) In-hospital and long-term mortality for acute heart failure: analysis at the time of admission to the emergency department. ESC Heart Failure, 7: 2650– 2661. https://doi.org/10.1002/ehf2.12847

[2]. Pathak, A., Lebrin, M., Vaccaro, A., Senard, J.M. and Despas, F. (2013), Pharmacology of levosimendan: inotropic, vasodilatory and cardioprotective effects. J Clin Pharm Ther, 38: 341-349. https://doi.org/10.1111/jcpt.12067

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[3]. Nicolina Conti, Milo Gatti, Emanuel Raschi, Igor Diemberger, Luciano Potena. (2021) Evidence and Current Use of Levosimendan in the Treatment of Heart Failure: Filling the Gap. Drug Design, Development and Therapy 15, pages 3391-3409.

[4]. Hansen MS, Andersen A, Nielsen-Kudsk JE. Levosimendan in pulmonary hypertension and right heart failure. Pulm Circ. 2018 Jul-Sep;8(3):2045894018790905. doi: 10.1177/2045894018790905

[5]. Kasikcioglu HA, Cam N. A review of levosimendan in the treatment of heart failure. Vasc Health Risk Manag. 2006;2(4):389-400. doi: 10.2147/vhrm.2006.2.4.389

[6]. MariuszKowalczyk, MaciejBanach, Gregory Lip, Dariusz Kozlowski, Dimitri P. Mikhailidis, et al. Levosimendan – a calcium sensitising agent with potential antiarrhythmic properties. International Journal of Clinical Practice, Wiley, 2010, 64 (8), pp.1148. ff10.1111/j.1742-1241.2010.02396.xff. ffhal00552654f