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RESEARCH ARTICLE

Use of levosimendan in cardiogenic shock [version 1; peer review: peer review discontinued]

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Abstract

Cardiogenic shock (CS) is acute inadequate tissue perfusion caused by the heart's inability to pump an adequate amount of blood. Due to the failure of classic inotrope agents, a sensitizer agent, levosimendan, has been used as a rescue therapy in such situations. In order to assess the effectiveness of levosimendan to treat CS, we studied its hemodynamic effects on patients with CS. A retrospective study was conducted at the ICU of the Military Hospital of Tunis between January 2004 and December 2009, and between January 2011 and December 2013. Twenty-six patients with CS refractory to catecholamines were included in our study. When catecholamines failed to improve the hemodynamic condition, levosimendan was introduced. This treatment was administered in two steps: a loading dose of 12 µg/kg/min was infused for 30 min; and then continuous infusion was given for 24 h at a dose of 0.1 µg/kg/min. Levosimendan significantly increased mean arterial pressure to 76 ± 7 mmHg at 48 h and cardiac index to 3.19 ± 0.68 L/min/m² and decreased pulmonary wedge pressure to 17 ± 3 mmHg at 48 h. Pulmonary arterial systolic pressure, pulmonary arterial diastolic pressure, and mean pulmonary arterial pressure were significantly reduced at 24 h. A significant decrease in lactate from 3.77 ± 2.93 to 1.60 ± 1.32 mmol/L, by 72 h, was also noted. Levosimendan significantly reduced systemic vascular resistance and pulmonary vascular resistances. Administration of levosimendan also reduced the need for catecholamines. Our study confirms the efficacy of levosimendan to stabilize hemodynamic parameters in patients with CS.

Keywords

levosimendan, cardiogenic shock, hemodynamic variables, mortality

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Introduction

Cardiogenic shock (CS) is a clinical condition of acute inadequate tissue perfusion caused by the heart's inability to pump an adequate amount of blood^{1,2}. Pharmacological treatment of CS is based on positive inotropic agents^{3,4}. Due to the failure of classic inotrope agents, a sensitizer agent, levosimendan, has been used as rescue therapy in such situations. This drug has both positive inotropic and myocardial relaxing properties, which increase the affinity of myofilaments within the myocardial cell to calcium without raising intracellular concentrations of calcium or AMPc⁵⁻⁸. Levosimendan can improve the hemodynamic status of patients with CS without increasing the consumption of myocardial oxygen and without increasing the risk of arrhythmia^{9,10}; however, few studies have been conducted on the use of levosimendan to treat CS.

The purpose of our study was to assess the effectiveness of levosimendan to treat CS by studying its hemodynamic effects on 26 patients with CS and who were refractory to catecholamines.

Materials and methods

A retrospective study was conducted at the ICU in the Military Hospital of Tunis for two periods: between January 2004 and December 2009, and from January 2011 until December 2013. All patients had CS and were aged >18 years. Patients who died within 48 h were excluded from the study. The diagnosis of CS was made if hypotension was <90 mmHg for more than 30 min and there were signs of low perfusion in the absence of a hypovolemia or cardiac arrhythmia. These signs were associated with a cardiac index of <2.2 L/min/m², a pulmonary capillary wedge pressure of >18 mmHg, and high doses of catecholamines had been ineffective to restore mean arterial pressure to 65 mmHg (i.e., dobutamine at >15 µg/kg/min and noradrenaline at >0.6 µg/kg/min).

When catecholamines failed to improve the hemodynamic condition and there were persistent signs of low peripheral perfusion, levosimendan was introduced (Orion Pharma, Espoo, Finlande). This treatment was administered in two steps: a loading dose of 12 µg/kg/min was infused for 30 min; and then continuous infusion was given for 24 h at a dose of 0.1 µg/kg/min. The pulmonary artery was catheterized using a 7.5-Fr Swan Ganz catheter with continuous cardiac output and mixed venous saturation measurements (Edwards Life sciences, North Carolina, USA). This catheter allowed us to collect hemodynamic parameters from each patient at just before administration of levosimendan (i.e., T0), at 30 and 90 min, and at 2, 4, 8, 12, 24, and 48 h.

Doses of catecholamine and plasma-lactate concentrations were recorded on days 0, 1, 2, and 3. Values of the left-ventricular ejection fraction were collected on days 0, 1, 2, 7, and 15. The primary endpoints were the evolution of hemodynamic parameters (the cardiac index, pulmonary pressure, and SvO₂). Data were recorded and analyzed using Microsoft Excel (2007) software and Epi Info 6.0.4 (<http://www.cdc.gov>). Continuous variables were expressed as their means ± standard deviations and were compared between groups using Student's two-tailed t-test. Non-parametric tests were also used where necessary (Mann-Whitney U test). Fisher's exact (or the chi-squared) test was used to compare categorical variables, as appropriate. A *p*-value of <0.05 was considered statistically significant.

The local ethics committee approved the use of the patients' data for this study.

Results

Dataset 1. Data used for retrospective analysis of the effects of levosimendan for cardiogenic shock

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Data was collected at the ICU in the Military Hospital of Tunis between Jan 2004 and Dec 2009, and between Jan 2011 and Dec 2013. PAM: Mean Arterial Pressure (mmHg); HR: Heart Rate (beat/min); POD: Right Atrial Pressure (mmHg); IC: Cardiac Index (L/min/m²); PAPO: Pulmonary Wedge Pressure (mmHg); RVS: Systemic Vascular Resistance (dyne/s/cm²); SVO2: Mixed Venous Oxygen Saturation (%); PAP: Pulmonary arterial systolic pressure (mmHg); Mean Pulmonary Arterial Pressure (mmHg); PAP D: Pulmonary arterial diastolic pressure (mmHg); RVP: Pulmonary Vascular Resistance (mmHg); Noradre: Norepinephrine (ug/kg/min); DOBU: Dobutamine (ug/kg/min); Adre: Epinephrine (ug/Kg/min); Lact: Arterial Lactate (mmol/L); FE: Left Ventricular Ejection fraction (%).

Twenty-six patients who were hospitalized for CS met our criterion for inclusion in this study. The status of CS was mainly secondary to myocardial infarction (in 42% of cases) and to cardiomyopathy of the peripartum (in 31% of cases). The demographic data are shown in Table 1.

The hemodynamic data at inclusion showed a mean arterial pressure of 63 ± 7 mmHg, a cardiac index of 1.96 ± 0.29 l/min/m², and a pulmonary capillary wedge pressure of 28 ± 4 mmHg.

Table 1. Distribution of demographic parameters.

Parameters	Value
Number of patients	26
Age (years)	51 ± 18
Gender ratio (male/female)	0.85
APACHE II medium	20 ± 7
Multiple organ-failure score	3.4 ± 1
Average length of stay in ICU (days)	13 ± 12
Etiology of CS	
- Myocardial infarction	12
- Peripartum cardiomyopathy	6
- Dilated cardiomyopathy	3
- Other pathology	5
History of patients	
- Coronary-artery disease	5
- Diabetes	5
- Arteritis ischemic lower limbs	3
- Smoking	4
Evolution	
- Survival	14
- Death	12

Levosimendan resulted in significantly improved mean arterial pressure to 76 ± 7 mmHg at 48 h ($p=0.001$ compared to T0) and cardiac index to 3.19 ± 0.68 L/min/m² ($p<0.0001$ compared to T0) (Figure 1) without any significant modification to either heart rate (101 ± 28 to 86 ± 13 , $p=0.26$) or the emergence of rhythm disturbance.

Levosimendan decreased pulmonary wedge pressure to 17 ± 3 mmHg at 48h ($p=0.001$ compared to T0). Pulmonary arterial systolic pressure was significantly reduced to 38 ± 5 mmHg after 48 h, compared to 49 ± 6 mmHg at inclusion ($p=0.016$ compared to T0). Pulmonary arterial diastolic pressure was significantly reduced to 20 ± 4 mmHg at 24 h, compared to 29 ± 4 mmHg at inclusion ($p<0.001$ compared to T0). Mean pulmonary arterial pressure

was significantly reduced to 26 ± 5 mmHg after 24 h compared to 35 ± 5 mmHg at inclusion ($p=0.001$ compared to T0).

There was a significant decrease in lactate from 3.77 ± 2.93 to 1.60 ± 1.32 mmol/L after 72 h. Levosimendan significantly reduced systemic vascular resistance (1834 ± 308 to 1321 ± 304 dyne/s/cm², $p<0.0001$) and pulmonary vascular resistances (289 ± 64 to 204 ± 78 dyne/s/cm², $p=0.001$). Administration of levosimendan also reduced the need for catecholamines (Figure 2), thus leading to a gradual reduction in the need for catecholamines in six patients.

Administration of levosimendan improved SvO₂ to $70 \pm 5\%$ after 24 h of infusion. The evolution of the left-ventricle ejection fraction

Figure 1. Data are expressed as mean values of cardiac index of all our patients.

Figure 2. Data are expressed as mean values from 15 patients who received norepinephrine, 15 patients who received Dobutamine and 5 patients who received epinephrine.

showed bottom-up kinetics as a function of time, which ranged from $26 \pm 5\%$ at inclusion to $54 \pm 5\%$ by day 15.

Discussion

Levosimendan is a calcium sensitizer. Its main mechanism of action is to increase the binding affinity of troponin C to Ca^{2+} and to stabilize its conformation. It also directly increases cardiac contractility without raising intracellular concentrations of calcium or AMPc. This makes levosimendan one of the best inotropic agents and causes least arrhythmia. In addition to its positive inotropic effects, levosimendan can cause vasodilatation of many vascular areas by opening ATP-dependent potassium channels. This increased coronary blood flow without increasing consumption of myocardial oxygen, thus explains the good tolerance of levosimendan in cases of coronary syndrome and also its effects as an anti-ischemic and a cardio-protector.

Many clinical studies have been conducted to assess the effects of levosimendan on hemodynamic parameters. The first studies focused on patients with decompensated heart failure. The main aim of these authors was to compare levosimendan with dobutamine (i.e., the main type of catecholamine used during low cardiac output). Three main studies have been published: the LIDO, SURVIVE, and CASINO studies.

In the LIDO study, 203 patients were hospitalized for severe cardiac failure and received either levosimendan (a loading dose of $24 \mu\text{g/kg/min}$ infused for 10 min; and then continuous infusion for 24 h at a dose of $0.1 \mu\text{g/kg/min}$, $n=103$) or dobutamine (a dose of $5 \mu\text{g/kg/min}$ was given without a loading dose, $n=100$). Levosimendan increased the cardiac index by $\geq 30\%$ and decreased pulmonary capillary wedge pressure by $\geq 25\%$ in 29 patients compared to only 15 patients receiving dobutamine (hazard ratio=1.9, CI= of 95%, $p=0.022$)^{11,12}. A decline in the need for catecholamines and the resumption of beta blockers occurred more frequently in patients that received levosimendan¹⁴. Mortality rate was also lower in patients that received levosimendan compared to dobutamine after 1 month (7.8% vs. 17%, $p=0.045$) and at 6 months (26% vs. 38%, $p=0.029$)^{9,11-13}.

The SURVIVE study was a multicenter study that included 1327 randomized patients with severe cardiac failure and who had a left-ventricle ejection fraction of $<30\%$. This study chose mortality as the main criterion for judgment and assessed the effectiveness of levosimendan compared to dobutamine¹¹. When the analyses focused on early mortality rates during the first 5 days, levosimendan was more effective than dobutamine (4.4% vs. 6%), indicating that levosimendan could be an alternative to dobutamine for patients with acute cardiac insufficiency. However, this favorable trend was not found in the longer term¹¹. During the 180 days of follow-up, there were 173 deaths (26%) in the group receiving levosimendan ($n=64$) compared to 185 (28%) in the group receiving dobutamine ($n=63$)¹⁵. However, the main criterion of mortality at 6 months may not be appropriate to judge the effectiveness of this treatment when its main benefits were observed at an early stage¹⁶.

The CASINO study was a randomized, controlled, double-blind study that included 299 patients with decompensated congestive heart failure. The study compared levosimendan therapy with dobutamine, and with a placebo. The study showed positive results for

its first assessment criterion: i.e., mortality rate at 6 months. The authors reported that improved survival was associated with the use of levosimendan¹⁶. In the light of these results, levosimendan represents a significant advance in the treatment of cardiac insufficiency. These studies have helped to expand levosimendan's indications and have also assessed its contribution to treat CS.

Many authors have evaluated the hemodynamic effects of levosimendan in the course of CS. Delle *et al.* concluded that administration of levosimendan to 10 patients with CS led to a significant increase in the cardiac index and significantly decreased vascular resistance¹⁷.

Russ *et al.* obtained similar results when they prescribed levosimendan to 56 patients with CS. They reported a significant increase in the cardiac index (from 2.1 ± 0.56 to $3.0 \pm 1.11 \text{ l/min/m}^2$ at 24 h, $p<0.01$) associated with a significant decrease in systemic vascular resistance (from 1208 ± 333 to $858 \pm 299 \text{ dynes/s/mm}^4$ at 24 h, $p<0.01$), whereas mean arterial pressure and pulmonary wedge pressure were slightly but non-significantly decreased. An improved SOFA score was found after 72 h of levosimendan treatment, suggesting improved organic function¹⁸.

Labriola *et al.* also reported the same variations in hemodynamic parameters as described by Delle *et al.* and Russ *et al.*, which were distinguished by the development of a significant increase in the left-ventricle ejection fraction¹⁹.

According to the study of Berry *et al.*, a continuous decrease in noradrenalin, dobutamine, and milrinone was associated with improved hemodynamic parameters in 93 patients with CS who received a continuous infusion of levosimendan for 26 h at a dose of $0.096 \mu\text{g/kg/min}$ ²⁰.

Labbene *et al.* analyzed the hemodynamic effects of levosimendan in 16 patients hospitalized with CS and who were refractory to catecholamine²¹. The evolution of hemodynamic parameters after infusion of levosimendan was marked by a significant increase in the cardiac index (from 2.01 ± 0.41 to $3.4 \pm 0.65 \text{ l/min/m}^2$ at 48 h) of SvO_2 (from $57\% \pm 5\%$ to $70\% \pm 3\%$) and the left-ventricular ejection fraction (from $26 \pm 6\%$ to $52 \pm 5\%$). There was also a significant decrease in pulmonary wedge pressure from 28 ± 5 to $17 \pm 5 \text{ mmHg}$, and of pulmonary arterial systolic pressure, of diastolic arterial systolic pressure, of average arterial systolic pressure of systemic vascular resistance (from 1833 ± 308 to $1216 \pm 304 \text{ dyne/s/cm}^2$ at 48 h), and of pulmonary vascular resistances (from 289 ± 64 at T0 to $179 \pm 44 \text{ dyne/s/cm}^2$ at 4 h)²¹. Our results agree with those from the literature and show the effectiveness of levosimendan to treat CS when it is refractory to catecholamines. At the recommended dose, levosimendan improves hemodynamic parameters by increasing the cardiac index and SvO_2 . It is responsible for the drop in systemic vascular resistance and pulmonary vascular resistances, and reduces the need for catecholamines. Other studies have also confirmed the efficacy of levosimendan to stabilize hemodynamic parameters in patients with CS²²⁻²⁵.

Conclusion

Our results show that levosimendan was efficacious at treating CS, and that it has several advantages compared to other isotropic

agents. However, it is expensive (compared to dobutamine) and the absence of a net profit on long-term survival represents a limit to its wide-spread use in pathologies with a poor prognosis, such as CS. Further randomized studies that include a placebo and a larger patient population with CS are needed to precisely determine the indications for levosimendan and to assess its cost in relation to patient survival.

Data availability

F1000Research: Dataset 1. Data used for retrospective analysis of the effects of levosimendan for cardiogenic shock, [10.5256/f1000research.5820.d40172](https://doi.org/10.5256/f1000research.5820.d40172)²⁶

Author contributions

IL and MF conceived the study. IL and FJ designed the experiments. KEA, CR and IL carried out the research. KEA and FJ prepared

the first draft of the manuscript. IL, FJ and KEA contributed to the experimental design and preparation of the manuscript. All authors were involved in the revision of the draft manuscript and have agreed to the final content.

Competing interests

No competing interests were disclosed.

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References

- Leurent G: [Ischaemic cardiogenic shock. Where do we stand in 2012?]. *Ann Cardiol Angelol (Paris)*. 2012; **61**: 417–422.
[PubMed Abstract](#) | [Publisher Full Text](#)
- Lambert Y, Boutout F: **Etat de choc cardiogénique dans les premières heures**. *Encycl Méd Chir*. Elsevier, Masson, Paris. Médecine d'urgence, 25-020-A-20, 2007; 14.
[Reference Source](#)
- Pirracchio R: **Etat de choc cardiogénique**. *Encycl Méd Chir*. Elsevier, Masson, Paris, Anesthésie-Réanimation, 36-840-C-10, 2012; 9.
[Reference Source](#)
- Salem JE, Aissaoui A: **Choc cardiogénique**. *Encycl Méd Chir*. Elsevier, Masson, Paris, Cardiologie, 11-038-B-10, 2014; 10.
[Reference Source](#)
- Pathak A, Lebrin M, Vaccaro A, *et al.*: **Pharmacology of levosimendan: inotropic, vasodilatory and cardioprotective effects**. *J Clin Pharm Ther*. 2013; **38**(5): 341–349.
[PubMed Abstract](#) | [Publisher Full Text](#)
- Parissis JT, Rafouli-Stergiou P, Paraskevaidis I, *et al.*: **Levosimendan: from basic science to clinical practice**. *Heart Fail Rev*. 2009; **14**(4): 265–275.
[PubMed Abstract](#) | [Publisher Full Text](#)
- Robertson IM, Sun YB, Li MX, *et al.*: **A structural and functional perspective into the mechanism of Ca²⁺-sensitizers that target the cardiac troponin complex**. *J Mol Cell Cardiol*. 2010; **49**(6): 1031–1041.
[PubMed Abstract](#) | [Publisher Full Text](#) | [Free Full Text](#)
- Papp Z, Édes I, Fruhwald S, *et al.*: **Levosimendan: molecular mechanisms and clinical implications: consensus of experts on the mechanisms of action of levosimendan**. *Int J Cardiol*. 2012; **159**(2): 82–87.
[PubMed Abstract](#) | [Publisher Full Text](#)
- Rami L, Giunti C, Mebazaa A, *et al.*: **Choc cardiogénique: quel inotrope choisir?** In: *Conférences d'actualisation*. Elsevier-SFAR, 2004; 425–440.
[Reference Source](#)
- Fotbolcu H, Duman D: **A promising new inotrope: levosimendan**. *Anadolu Kardiyol Derg*. 2010; **10**(2): 176–82.
[PubMed Abstract](#) | [Publisher Full Text](#)
- De Luca L, Colucci WS, Nieminen MS, *et al.*: **Evidence-based use of levosimendan different clinical settings**. *Eur Heart J*. 2006; **27**(16): 1908–1920.
[PubMed Abstract](#) | [Publisher Full Text](#)
- Follah F, Celand JG, Just H, *et al.*: **Efficacy and safety of intravenous levosimendan compared with dobutamine in severe low-output heart failure (the LIDO study): a randomized double-blind trial**. *Lancet*. 2002; **360**(9328): 196–202.
[PubMed Abstract](#) | [Publisher Full Text](#)
- Nieminen MS, Fruhwald S, Heunks LM, *et al.*: **Levosimendan: current data clinical use and future development**. *Heart Lung Vessel*. 2013; **5**(4): 227–245.
[PubMed Abstract](#) | [Free Full Text](#)
- Labbene I, Belhadj AM, Lebbi A, *et al.*: **Intérêt du lévosimendan dans les défaillances myocardiques aiguës**. *J Magh A Réa Méd Urg*. 2006; **8**: 34–38.
[Reference Source](#)
- Mebazaa A, Nieminen MS, Packer M, *et al.*: **Levosimendan vs dobutamine for patients with acute decompensated heart failure: the SURVIVE Randomized Trial**. *JAMA*. 2007; **297**(17): 1883–1891.
[PubMed Abstract](#) | [Publisher Full Text](#)
- Ferjani M: **Le lévosimendan une révolution dans la prise en charge des défaillances myocardiques aiguës?** *J Magh A Réa Méd Urg*. 2006; **8**: 2.
[Reference Source](#)
- Delle Karth G, Buberl A, Geppert A, *et al.*: **Hemodynamic effects of a continuous infusion of levosimendan in critically ill patients with cardiogenic shock requiring catecholamines**. *Acta Anaesthesiol Scand*. 2003; **47**(10): 1251–1256.
[PubMed Abstract](#) | [Publisher Full Text](#)
- Russ MA, Prondzinsky R, Christoph A, *et al.*: **Hemodynamic improvement following levosimendan treatment in patients with acute myocardial infarction and cardiogenic shock**. *Crit Care Med*. 2007; **35**(12): 2732–2739.
[PubMed Abstract](#) | [Publisher Full Text](#)
- Labriola C, Siro-Brigiani M, Carrata F, *et al.*: **Hemodynamic effects of levosimendan in patients with low-output heart after cardiac surgery**. *Int J Clin Pharmacol Ther*. 2004; **42**(4): 204–211.
[PubMed Abstract](#)
- Berry WT, Hewson RW, Langrish CJ, *et al.*: **Levosimendan: a retrospective single-center case series**. *J Crit Care*. 2013; **28**(6): 1075–1078.
[PubMed Abstract](#) | [Publisher Full Text](#)
- Labbene I, Hichri N, Thabet S, *et al.*: **Hemodynamic Effects of levosimendan during cardiogenic shock**. *Ann Fr Anesth Reanim*. 2007; **26**: S167–S170.
- Lehmann A, Lang J, Boldt J, *et al.*: **Levosimendan in patients with cardiogenic shock undergoing surgical revascularization: a case series**. *Med Sci Monit*. 2004; **10**(8): MT89–93.
[PubMed Abstract](#)
- Katsytadze I, Amosova E, Purdkiy I, *et al.*: **Long term effects of levosimendan therapy in patients with cardiogenic shock**. *Resuscitation*. 2013; **84**(Supplement 1): S8–S98.
[Publisher Full Text](#)
- Zobel C, Reuter H, Schwinger RH: **[Treatment of cardiogenic shock with the Ca²⁺ sensitizer levosimendan]**. *Med Klin (Munich)*. 2004; **99**(12): 742–746.
[PubMed Abstract](#) | [Publisher Full Text](#)
- Greif M, Zwermann L, Reithmann C, *et al.*: **Levosimendan as rescue therapy in severe cardiogenic shock after ST-elevation myocardial infarction**. *Acute Card Care*. 2008; **10**(3): 185–190.
[PubMed Abstract](#) | [Publisher Full Text](#)
- Janen F, El Arayedh K, Labbene I, *et al.*: **Dataset 1 in: Use of levosimendan in cardiogenic shock**. *F1000Research*. 2014.
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