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# Sodium Nitroprusside in Acutely Decompensated Heart Failure with Elevated Lactate: A Single-Centre Experience

**Luka Antolković[1]*, Aleksandar Blivajs[1], Tomislav Čikara[1], Anita Pleško[1], Ivan Kralj[2]**

[1]Dubrava University Hospital, Zagreb, Croatia
[2]University of Zagreb, School of Medicine, Zagreb, Croatia

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***ADDRESS FOR CORRESPONDENCE:** Luka Antolković, Klinička bolnica Dubrava, Av. G. Šuška 6, HR-10000 Zagreb, Croatia. / Phone: +385-91-7860-886 / E-mail: lukaantolkovic0@gmail.com

**ORCID:** Luka Antolković, https://orcid.org/0000-0002-5313-2213 • Aleksandar Blivajs, https://orcid.org/0000-0003-3404-3837 Tomislav Čikara, https://orcid.org/0000-0001-8012-4481 • Anita Pleško, https://orcid.org/0009-0009-7659-7011 Ivan Kralj, https://orcid.org/0009-0009-2469-7440

**Introduction:** Acute decompensated heart failure with biochemical evidence of hypoperfusion remains a therapeutic challenge, particularly in patients with preserved arterial pressure. Sodium nitroprusside may improve hemodynamics through controlled afterload reduction[1]. We aimed to describe changes in lactate levels and renal function, 30-day survival, and treatment-related adverse events in patients receiving sodium nitroprusside.

**Patients and Methods:** This retrospective, single-centre observational study included 16 critically ill patients treated in a cardiac intensive care unit between March 2022 and September 2026. All patients had clinical congestion, elevated lactate, and a baseline mean arterial pressure (MAP) above 65 mmHg. Sodium nitroprusside was titrated under invasive arterial pressure monitoring, with a minimum target MAP of 55 mmHg. Hemodynamic assessment was primarily echocardiographic. Outcomes included serial lactate levels at baseline, 24, 48, and 72 hours, and 7 days; renal function; 30-day survival; and adverse events. Lactate levels were analyzed using the Friedman test, followed by Wilcoxon signed-rank tests with Bonferroni correction

**Results:** Sixteen patients were included (mean age 61.4 ± 12.6 years). Biventricular heart failure was present in 13 patients (81.3%), and non-ischemic dilated cardiomyopathy in 10 (62.5%). Eleven patients (68.8%) received concomitant low-dose inotropic support. One patient required venoarterial extracorporeal membrane oxygenation (VA-ECMO) before nitroprusside initiation, and one received an axial-flow pump. Median baseline lactate was 4.7 mmol/L (interquartile range [IQR] 3.6–7.4). Lactate levels changed significantly over time (Friedman test, $p < 0.001$), with significant reductions from baseline at 24 hours (1.93 mmol/L, IQR 1.1–3.4), 48 hours (1.09 mmol/L, IQR 0.78–1.5), and 72 hours (0.96 mmol/L, IQR 0.75–1.68; all adjusted $p < 0.001$). Mean serum creatinine decreased from 145.7 ± 45.9 to 110.0 ± 44.5 µmol/L after seven days ($p = 0.01$). Thirty-day survival was 75% (12/16). Ten patients were discharged home, while two underwent heart transplantation. Mean nitroprusside infusion duration was 45.3 ± 23.1 hours, with no observed nitroprusside-related adverse events

**Conclusion:** In this small retrospective cohort, sodium nitroprusside treatment was associated with reductions in lactate levels and serum creatinine over time. These findings support further investigation of controlled afterload reduction in selected patients with acute decompensated heart failure, elevated lactate, and preserved baseline arterial pressure. The absence of a control group and the small sample size preclude conclusions regarding causality or comparative efficacy.

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## LITERATURE

1. Choi EYK, Lim HS. Treatment of Cardiogenic Shock: Inotropes, Vasopressors and Machines. Br J Hosp Med (Lond). 2024 Dec 30;85(12):1-17. https://doi.org/10.12968/hmed.2024.0396