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Efficacy and Safety of Vasopressors and Inotropes in AMI-Related Cardiogenic Shock: A Systematic Review and Meta-Analysis

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Abstract

In patients with acute myocardial infarction (AMI) complicated by cardiogenic shock (CS), vasopressors and inotropes are widely used to stabilize circulation. However, their effect on survival remains uncertain. We performed a systematic review of MEDLINE, EMBASE, and CENTRAL through 20 February 2019, including both randomized and observational studies reporting mortality in AMI-related CS. Eligible studies compared patients receiving at least one vasopressor or inotrope with those who did not receive such therapy. Studies limited to post-cardiac surgery patients, case reports, and correspondence were excluded. Nineteen studies (six randomized trials) involving 2,478 patients were analyzed, though the overall quality of evidence was low. No vasopressor or inotrope—including adrenaline, noradrenaline, vasopressin, milrinone, levosimendan, dobutamine, or dopamine—was consistently associated with reduced mortality. Levosimendan showed a trend toward improved outcomes (RR 0.69, 95% CI 0.47–1.00). These results highlight the limited evidence supporting survival benefits from standard vasopressors or inotropes in AMI-related CS and underscore the need for rigorously designed randomized trials to clarify their role.

Keywords: Cardiogenic shock, Inotropes, Vasopressors, Myocardial infarction, Survival, Systematic review

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Introduction

Cardiogenic shock (CS) is a critical condition characterized by reduced cardiac output, leading to tissue hypoperfusion and multi-organ dysfunction, with high associated mortality [1, 2]. Clinically, CS is commonly defined based on hemodynamic parameters and signs of inadequate perfusion [3].

Vasopressors and inotropes are standard therapeutic options in CS to support cardiac output and improve organ perfusion. Current guidelines recommend noradrenaline as the first-line agent, with inotropes added in cases of persistent low cardiac output [4, 5].

Acute myocardial infarction (AMI) is a frequent cause of CS, responsible for roughly 30% of cases [6–8]. In this context, vasopressors and inotropes are often administered to maintain coronary perfusion and cardiac output. However, their use carries risks, including arrhythmias and increased myocardial oxygen demand due to enhanced contractility, elevated afterload, or compromised coronary perfusion [9].

To better understand their impact, we conducted a systematic review evaluating the effects of commonly

used vasopressors and inotropes on survival in AMI-related CS. Specifically, we sought to answer: (1) whether administration of adrenaline, noradrenaline, vasopressin, milrinone, levosimendan, dobutamine, or dopamine reduces mortality in these patients, and (2) how these agents affect safety outcomes, including ICU stay duration, need for supportive measures, hemodynamic response, organ failure, and therapy-related complications.

Experimental Section

This systematic review adhered to the PRISMA guidelines for reporting systematic reviews and meta-analyses [10]. The study protocol was registered in the PROSPERO database (CRD42018107644).

Selection criteria

Studies were eligible if they reported mortality outcomes in patients with AMI-related CS and included at least one treatment group receiving a vasopressor or inotrope, along with a control group not exposed to that therapy. The interventions of interest were:

1. Adrenaline
2. Noradrenaline
3. Vasopressin
4. Milrinone
5. Levosimendan
6. Dobutamine
7. Dopamine

We excluded studies that only compared different doses of the same drug without an unexposed control group, as well as studies limited to post-cardiac surgery patients. Due to the anticipated scarcity of randomized controlled trials, all study designs were included except for case reports and correspondence.

Search strategy

A medical information specialist (JL) systematically searched MEDLINE (OVID), EMBASE (OVID), and the Cochrane Central Register of Controlled Trials (CENTRAL) from their inception until 20 February 2019. The search strategy combined both controlled vocabulary (e.g., MeSH terms) and free-text keywords related to: (1) cardiogenic shock (including shock or low cardiac output in the context of myocardial infarction) and (2) vasopressor or inotrope therapy (see Appendix A). Studies involving animals, narrative reviews, and editorials were excluded. No additional restrictions were applied. Reference lists and citations of relevant articles were also checked to identify further eligible studies. The bibliographic data were imported into EndNote X8, and duplicates were removed.

Data extraction and quality assessment

Two researchers (MK, WL, DO, or VH) independently screened titles and abstracts, excluding studies that did not meet inclusion criteria. Full texts of potentially eligible studies were then reviewed independently by two researchers to confirm eligibility. Conference abstracts were considered if sufficient data were available. Data extraction was conducted independently by two researchers (MK and VH), and attempts were made to obtain missing or unclear data by contacting study authors. The methodological quality of randomized trials was evaluated using the Revised Cochrane risk-of-bias tool (RoB 2.0), and non-randomized studies were assessed with the Newcastle–Ottawa Scale, focusing on mortality outcomes [11, 12]. The overall certainty of evidence was graded using the GRADE framework [13]. Any disagreements were resolved through discussion or consultation with a third researcher (JH).

Data analysis

The primary endpoint was mortality, categorized as short-term (<90 days) and long-term (≥90 days). Treatment groups were defined by exposure to the specific vasopressor or inotrope, while control groups were composed of pooled comparators across studies. Mortality outcomes were synthesized quantitatively using a random-effects model, reported as relative risks (RR) with 95% confidence intervals (CI). Statistical heterogeneity was assessed using Chi-squared and I-squared tests, with $p < 0.10$ indicating significance. I-squared values exceeding 40% were considered indicative of substantial heterogeneity. Sensitivity analyses were performed when appropriate, such as excluding conference abstracts or observational studies. Analyses were performed using Review Manager version 5.3.

Secondary outcomes, including duration of supportive therapy, length of ICU stay, hemodynamic effects, organ dysfunction, and therapy-related complications, were summarized descriptively.

Results

The search identified 6187 unique records, of which 110 full-text articles were reviewed for eligibility. Ultimately, 19 studies met inclusion criteria for the systematic review. Reasons for exclusion are presented in **Figure 1**.

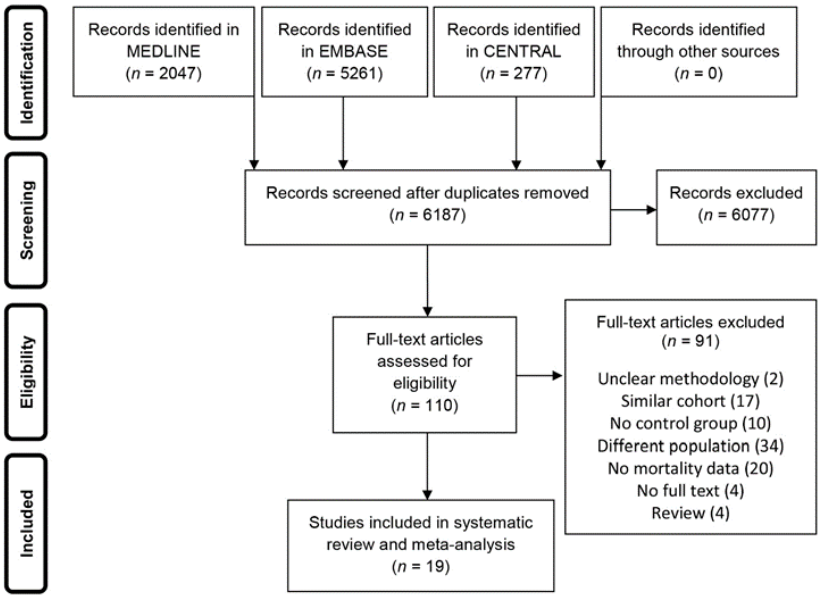

Figure 1. Flowchart of study selection

Study characteristics

Study characteristics of the 19 included studies are presented in **Table 1**.

Table 1. Summary of included studies ($n = 19$) on inotrope/vasopressor therapy in cardiogenic shock

Study	Year	Country	Center	Setting	Inclusion Period	Follow-up	Overall N	CS N
Cronin	1965	Canada	Single	Retrospective cohort	1952–1961	10 years	140	140
Moulopoulos	1993	Greece	Single	Retrospective cohort	1978–1991	1 month	55	55
Andriange	1971	Belgium	Single	Retrospective cohort	1967–1970	1 year	450	45
Samimi-Fard	2007	Spain	Single	Randomized trial	2003–2004	1 year	22	22
El Mokhtari	2007	Germany	Single	Retrospective cohort	-	1 year	20	20
Fuhrmann	2008	Germany	Single	Randomized trial	2003–2005	30 days	32	32
Myburgh	2008	Australia	Multi	Randomized trial	2004–2006	90 days	280	128
Christoph	2008	Germany	Single	Prospective cohort	2003–2005	-	22	22
De Backer	2010	Belgium	Multi	Randomized trial	2003–2007	1 year	1679	280
Omerovic	2010	Sweden	Single	Prospective cohort	2004–2006	1 year	94	94
Caetano	2012	Portugal	-	Retrospective cohort (conference paper)	-	10.6 ± 10.9 months	37	37
Huseby	2013	Norway	Single	Randomized trial	2006–2010	6 months	61	9
Affronti	2013	Italy	Single	Retrospective case-control	2011	-	17	17
Katsytadze	2013	Ukraine	-	Retrospective cohort (conference paper)	-	1 year	27	27
Yagi	2015	Japan	Multi	Prospective cohort (conference paper)	2012–2014	30 days	979	240
Tarvasmaki	2016	Finland	Multi	Prospective cohort	2010–2012	90 days	216	216
Levy	2018	France	Multi	Randomized trial	2011–2016	60 days	57	57
Vally	2019	France	Single	Retrospective cohort	2010–2017	30 days	150	150
Lewis	2018	USA	Single	Retrospective cohort	2013–2015	In-hospital	100	100

CS: cardiogenic shock.

Participants

The included studies comprised a total of 4441 patients, of whom 2478 were diagnosed with cardiogenic shock (CS). Detailed baseline characteristics are provided in Supplementary Materials, Table S1. All studies contained at least one subgroup with AMI-related CS, while ten studies focused exclusively on this patient population. Among the CS patients, 137 received adrenaline, 594 received noradrenaline, 8 received vasopressin, 50 received milrinone, 209 received levosimendan, 200 received dobutamine, and 367 received dopamine.

Intervention

Indications for initiating vasopressors or inotropes differed across the studies. Specific criteria for therapy initiation are summarized in Supplementary Materials, Table S1.

Comparison

Six randomized controlled trials were identified with varying intervention and control arms [14–19]. These included comparisons such as noradrenaline versus adrenaline in AMI-related CS [14], noradrenaline versus adrenaline for patients requiring any vasopressor [15], noradrenaline versus dopamine in all-cause shock ([16], SOAP II trial), dobutamine versus levosimendan in STEMI patients with post-PCI CS [17], levosimendan versus placebo in acute STEMI patients with heart failure within 48 hours post-PCI ([18], LEAF trial), and levosimendan versus enoximone in refractory CS under 2 hours after PCI [19]. In all studies, control groups received vasopressors or inotropes other than the intervention drug, most commonly noradrenaline.

Study quality

Using the GRADE approach, the overall certainty of evidence for mortality outcomes was rated as low. This was primarily due to limited RCT data, heterogeneity, and risk of bias. Individual study quality assessments are available in the Supplementary Materials (Tables S2 and S3).

Mortality outcomes

Short-term (<90 days) and long-term (≥90 days) mortality outcomes are presented in **Figures 2 and 3**. Individual study mortality data are detailed in Supplementary Materials, Table S4.

Adrenaline versus control

Three studies evaluated adrenaline [14, 15, 20]. Levy *et al.* and Myburgh *et al.* conducted RCTs comparing adrenaline to noradrenaline, focusing on hemodynamic endpoints such as cardiac index changes and achievement of MAP targets >24 hours. Tarvasmaki *et al.* performed an observational study comparing 90-day mortality in acute CS patients receiving adrenaline versus an unexposed control group. Pooled RCT results showed no statistically significant difference in short-term mortality with adrenaline (RR 1.22, 95% CI 0.60–2.50; $I^2 = 58\%$). Long-term mortality data similarly demonstrated no benefit (RR 1.37, 95% CI 0.45–4.16; $I^2 = 94\%$).

Noradrenaline versus control

Six studies, including three RCTs, reported outcomes for patients treated with noradrenaline [14–16, 20–22]. Analysis of short-term mortality revealed no significant difference (RR 0.84, 95% CI 0.63–1.10; n = 4 studies; $I^2 = 30\%$). Limiting the analysis to RCTs alone yielded consistent findings (RR 0.77, 95% CI 0.56–1.06; $I^2 = 26\%$). For long-term mortality, pooled results also showed no effect of noradrenaline (RR 1.31, 95% CI 0.80–2.15; n = 3 studies), with substantial heterogeneity observed ($I^2 = 81\%$).

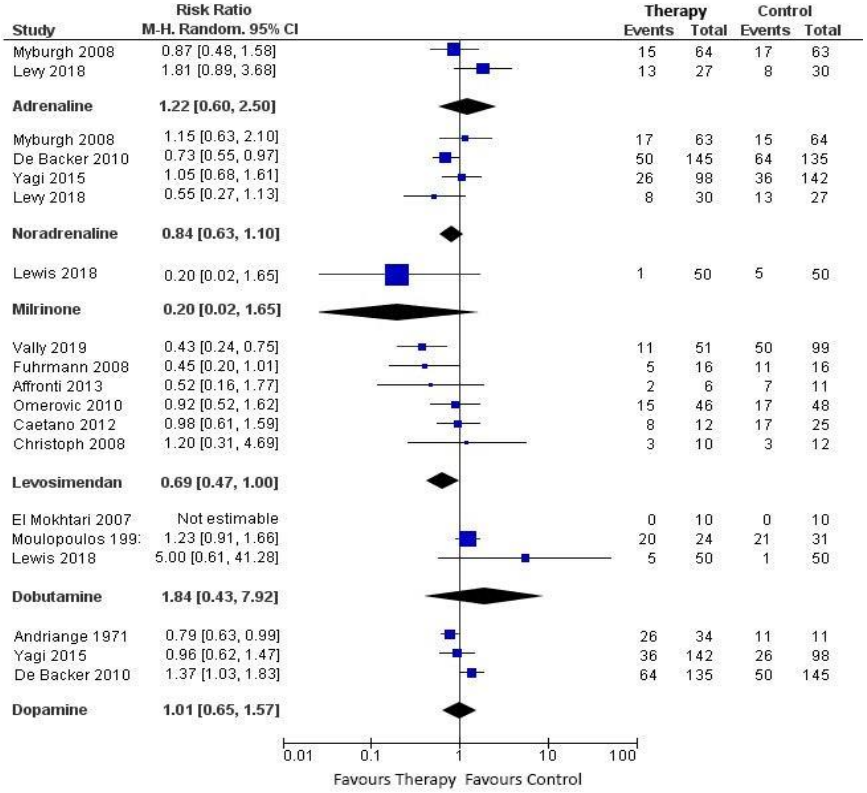

Figure 2. Forest plot demonstrating short-term (<90 day) mortality of cardiogenic shock patients treated with a vasopressor/inotrope versus a constructed control group

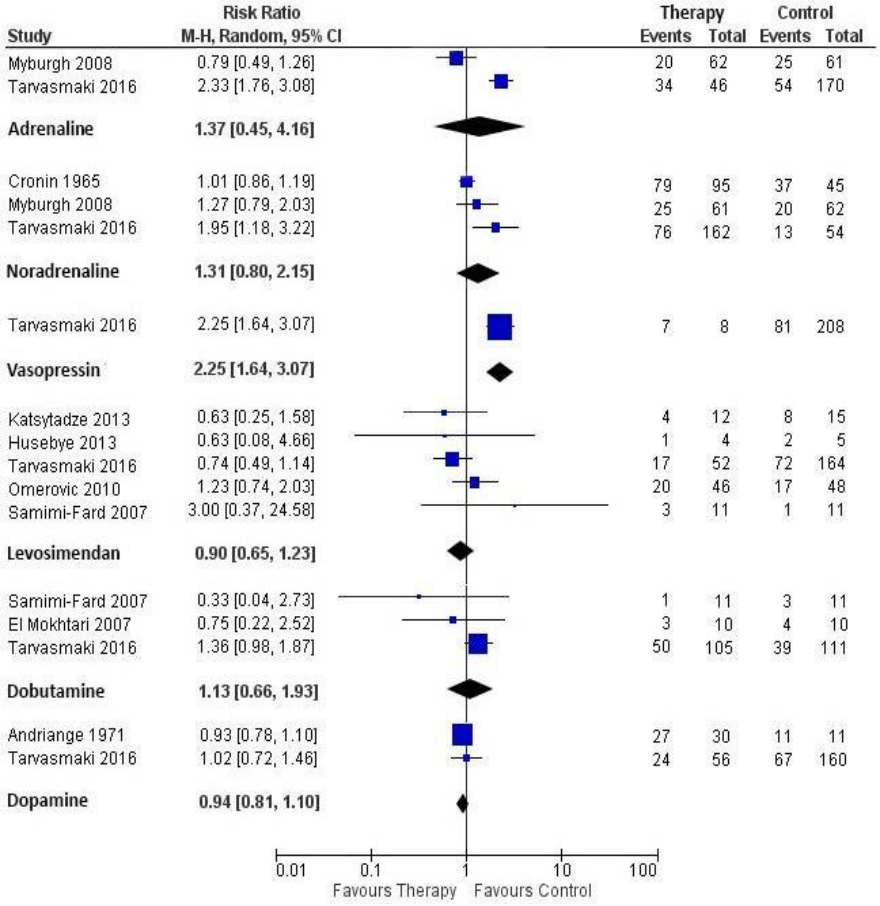

Figure 3.

Vasopressin versus control

Data on vasopressin were available from a single observational study [20]. In this cohort, 7 of 8 patients (87.5%) treated with vasopressin died within 90 days, compared with 81 of 208 patients (38.9%) in the control group, resulting in a relative risk (RR) of 2.25 (95% CI 1.64–3.07).

Milrinone versus control

Mortality outcomes for milrinone were reported in one observational study [23]. Lewis *et al.* found no statistically significant difference in in-hospital mortality between milrinone and dobutamine. Among 50 patients receiving milrinone, there was 1 death (2.0%), while 5 deaths (10.0%) occurred in the 50-patient control group (RR 0.20, 95% CI 0.02–1.65).

Levosimendan versus control

Ten studies, including three RCTs, assessed mortality in patients receiving levosimendan [17–20, 24–29]. Short-term mortality pooled across six studies suggested a trend toward reduced risk with levosimendan (RR 0.69, 95% CI 0.47–1.00; n = 352; I^2 = 39%). Sensitivity analysis excluding the conference paper strengthened this effect (RR 0.61, 95% CI 0.41–0.90; n = 5). For long-term mortality, pooled analysis of five studies showed no significant effect (RR 0.90, 95% CI 0.65–1.23; I^2 = 4%). Restricting to the two RCTs reporting long-term mortality yielded similar findings (RR 0.78, 95% CI 0.36–1.70; n = 3), as did an analysis excluding the conference paper (RR 0.95, 95% CI 0.65–1.40; n = 4).

Dobutamine versus control

Five studies, including one RCT, evaluated dobutamine [17, 20, 23, 30, 31]. Pooled short-term mortality showed no significant benefit (RR 1.84, 95% CI 0.43–7.92; I^2 = 56%). Similarly, long-term mortality pooled across three studies indicated no effect (RR 1.13, 95% CI 0.66–1.93; I^2 = 19%).

Dopamine versus control

Four studies, including one RCT, reported outcomes for dopamine [16, 20, 22, 32]. Short-term mortality from three studies demonstrated no significant effect (RR 1.01, 95% CI 0.65–1.57; I^2 = 84%). Sensitivity analysis excluding the conference paper confirmed this result (RR 1.04, 95% CI 0.51–2.12; n = 2). Long-term mortality pooled from two studies also showed no benefit (RR 0.94, 95% CI 0.81–1.10; I^2 = 0%).

Secondary outcomes

A summary of primary outcomes, treatment effects, and secondary endpoints from all included studies is provided in Supplementary Materials, Table S5. Reporting of safety outcomes and adverse events was inconsistent across studies, although arrhythmias were frequently noted.

Discussion

In this review, we evaluated the current literature on the use of vasopressors and inotropes in patients with AMI-related cardiogenic shock (CS). Overall, our findings indicate that commonly used agents—including adrenaline, noradrenaline, milrinone, levosimendan, dobutamine, and dopamine—did not demonstrate a significant effect on short-term or long-term mortality. The quality of available evidence was generally low, largely due to small sample sizes, heterogeneous study designs, and a predominance of observational data.

Notably, pooled estimates from six studies reporting short-term mortality suggested a potential trend toward improved outcomes with levosimendan (RR 0.69, 95% CI 0.47–1.00), although the certainty of this evidence remains low. Conversely, vasopressin was associated with higher mortality compared to control; however, this result derives from a single observational study involving only eight treated patients versus a substantially larger control group, introducing significant bias.

Our results complement and extend previous systematic reviews. A 2018 Cochrane review including 13 RCTs (n = 2001) [33] primarily evaluated patients with acute-on-chronic heart failure or post-cardiac surgery low cardiac output syndrome rather than AMI-related CS. That review suggested a modest short-term mortality benefit for levosimendan versus dobutamine, while other agents showed no significant differences. A separate 2016 Cochrane review examined vasopressor therapy in hypotensive shock from various etiologies [34]; patients with AMI-related CS were underrepresented, and no subgroup analysis by shock type was performed. Notably, dopamine increased the risk of arrhythmias compared to noradrenaline, while overall mortality differences were not observed.

Current ESC 2017 guidelines recommend dobutamine in patients with predominantly low cardiac output (Class IIb) and noradrenaline for CS with severe hypotension (Class IIb) [5]. The recommendation for noradrenaline is largely based on the SOAP II trial [16], which demonstrated lower arrhythmia rates and a trend toward reduced mortality versus dopamine. However, methodological concerns exist, including unstratified randomization and heterogeneous CS populations (AMI, chronic heart failure, post-cardiotomy). Recent evidence in older patients with vasodilatory hypotension suggests that a permissive hypotension strategy with reduced noradrenaline exposure may be safe and potentially advantageous [35].

Despite limited efficacy data, vasopressors and inotropes remain widely used in clinical practice. None of the studies included in this review incorporated a control group that received no pharmacologic therapy, so the current evidence only allows comparisons between agents rather than against placebo. Consequently, it remains unclear whether these drugs are genuinely effective in reducing mortality, or if they are simply equivalent in effect. Importantly, hemodynamic improvements do not always translate to improved tissue perfusion or clinical outcomes. Furthermore, variability in the definition of CS across studies contributes to additional uncertainty.

The findings underscore the urgent need for rigorously designed trials assessing the effectiveness of vasopressors and inotropes in AMI-related CS. Such trials should examine not only comparative efficacy among drugs and dosing strategies but also their impact on mortality and other patient-centered outcomes. Evidence from out-of-hospital cardiac arrest (OHCA) trials highlights the complexity of translating short-term hemodynamic benefits into meaningful survival gains [36–38], reinforcing the importance of high-quality, placebo-controlled RCTs in CS populations.

Our review has several limitations. Due to the scarcity of studies focused exclusively on AMI-related CS, we included studies with mixed CS etiologies that contained AMI subgroups. The included studies exhibited substantial heterogeneity in terms of patient populations, interventions, comparators, and outcomes, precluding detailed subgroup analyses. Many studies were single-center, retrospective, and small in size, which increases susceptibility to selection bias. While all study designs were included, conference abstracts were also considered to mitigate publication bias, though their methodological rigor is difficult to assess. Sensitivity analyses excluding conference papers and/or observational studies were conducted to evaluate the robustness of findings.

Conclusions

Currently, there is insufficient high-quality evidence to support the notion that routinely used vasopressors and inotropes reduce mortality in patients with AMI-related CS. Our findings highlight the critical need for well-designed randomized trials to establish the efficacy and safety of these therapies in this high-risk population.

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