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RESEARCH ARTICLE

Acute myocardial infarction vs acute decompensated heart failure in cardiogenic shock: a systematic review and meta-analysis of clinical phenotypes and mortality

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Abstract

Aims Cardiogenic shock (CS) encompasses distinct phenotypes driven by acute myocardial infarction-driven CS (AMI-CS) and acutely decompensated heart failure-driven CS (ADHF-CS). Consolidated comparative evidence remains limited. This meta-analysis compared presentation, management, and short-term mortality between AMI-CS and ADHF-CS.

Methods Observational studies between 2019 and 2025 comparing AMI-CS and ADHF-CS were summarized in accordance with Preferred Reporting Items for Systematic reviews and Meta-analyses (PRISMA) 2020. Random-effects models were used to estimate odds ratios (ORs) and mean differences with 95% confidence intervals. The primary outcome was short-term (in-hospital/30-day) mortality.

Results Twenty-nine studies (497 368 patients) were pooled. AMI-CS patients were older (mean difference 4.76 years, 95% confidence interval .37–9.14) with higher ejection fraction at presentation (mean difference 6.04%, 4.53–7.55). AMI-CS presented more fulminantly, with greater odds of cardiac arrest (OR 2.03, 1.04–3.98) and Society for Cardiovascular Angiography and Interventions Stage D/E shock (OR 1.50, 1.33–1.70); invasive filling pressures did not differ. Phenotype-defining variables (left ventricular ejection fraction, Society for Cardiovascular Angiography and Interventions stage, invasive haemodynamics) were extractable in <1.1% of the pooled cohort. Temporary mechanical circulatory support was more frequent in AMI-CS (any device OR 5.44, 2.83–10.46; intra-aortic balloon pump OR 4.73; percutaneous ventricular assist device OR 2.67; veno-arterial extracorporeal membrane oxygenation OR 3.27). Destination therapy was much less frequent (OR .09, .03–.24); major bleeding was modestly higher (OR 1.36, 1.02–1.82). Short-term mortality was higher in AMI-CS (OR 1.58, 1.06–2.37).

Conclusion AMI-CS presents more fulminantly than ADHF-CS, with markedly greater mechanical support use and higher short-term mortality. A dual-axis framework integrating shock severity with aetiology is proposed as an organizing principle for hypothesis generation; validation in prospective, phenotype-rich cohorts is required.

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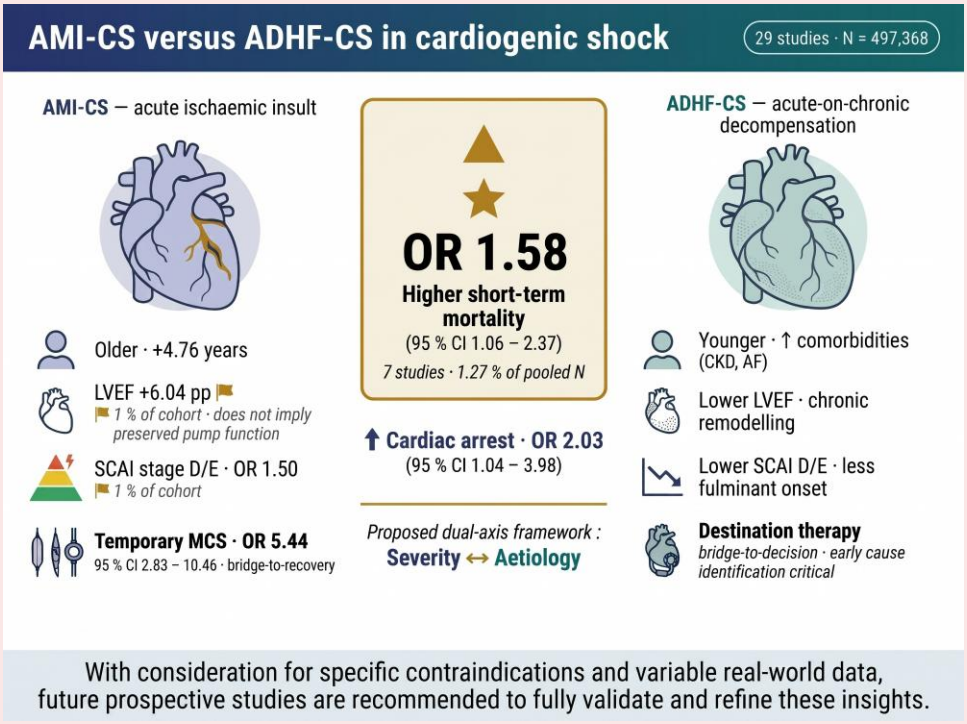
Graphical Abstract



Acute myocardial infarction versus acute decompensated heart failure in cardiogenic shock.

Keywords Cardiogenic shock • Acute myocardial infarction • Heart failure • Mechanical circulatory support • Meta-analysis • Mortality

Introduction

Cardiogenic shock (CS), a low-output state with critical end-organ hypoperfusion, remains the leading cause of in-hospital mortality among acute cardiovascular admissions despite advances in revascularization, mechanical support, and protocolized care.[1,2] The clinical paradigm has historically centred on acute myocardial infarction-driven CS (AMI-CS), as exemplified by the SHOCK trial, which established early revascularization as standard of care.[3,4]

Contemporary epidemiology has shifted substantially. Acutely decompensated heart failure-driven CS (ADHF-CS) now constitutes an equal or greater proportion of cardiac intensive care admissions, driven by population ageing, improved post-infarction survival, and rising heart failure prevalence.[5,6] The two phenotypes diverge pathophysiologically: AMI-CS arises from abrupt ischaemic loss of contractility with rapid haemodynamic deterioration and arrhythmic instability, whereas ADHF-CS reflects acute exacerbation of chronic remodelling, often with congestion, multi-organ dysfunction, and substantial comorbid burden.[7–13]

Historically, there has been a lack of consolidated information comparing aetiologies, with most studies focusing on discrete risk variables or mixed populations rather than direct comparisons. However, recent large observational registry studies have examined the differences in management and outcomes between AMI-CS and ADHF-CS.[14–17] This evidence gap has tangible clinical consequences in the era of Society for Cardiovascular Angiography and Interventions (SCAI) shock staging, hub-and-spoke shock systems, and increasingly aggressive temporary mechanical circulatory support (tMCS) strategies. This systematic review and meta-analysis aimed to compare presentation, management strategies, and short-term outcomes between AMI-CS and ADHF-CS, with prespecified emphasis on shock severity markers, mechanical support utilization, and short-term mortality.

Methods

Protocol and registration

This systematic review and meta-analysis was conducted in accordance with the Preferred Reporting Items for Systematic reviews and Meta-Analyses (PRISMA) 2020 statement.[18] The protocol was prospectively registered with International Prospective Register of Systematic Reviews (PROSPERO [CRD420251242655]).

Eligibility criteria

Eligible studies were observational cohorts, registry-based analyses, or randomized controlled trials directly comparing two adult phenotypes (≥18 years): AMI-CS and ADHF-CS. To preserve phenotype integrity of the ADHF-CS cohort, studies using broad terms such as 'non-ischaemic CS' or 'non-AMI-CS' were included only if aetiology breakdowns confirmed that >75% of patients had *de novo* or acute-on-chronic heart failure; studies grouping >25% other aetiologies (myocarditis, valvular emergencies, arrhythmias) without isolated HF-CS data were excluded. Inclusion was restricted to studies published or with patient enrolment predominantly from 2019 onwards to ensure contemporary relevance and the likely use of the SCAI shock classification.[19] Studies were required to report short-term (in-hospital or 30-day) all-cause mortality for both phenotypes in a format permitting calculation or extraction of a comparative effect estimate [adjusted or unadjusted odds ratio (OR) or Hazard ratio (HR) with 95%

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confidence interval (CI), or raw event counts] and were required to be full-text peer-reviewed manuscripts. A 10-point eligibility-scoring framework comprising critical and important criteria governed final selection (full rubric in Supplementary Methods, S-Methods S1). Case reports, case series with fewer than 20 patients per group, conference abstracts, narrative reviews, and editorials were excluded.

Information sources

PubMed/MEDLINE, Embase, the Cochrane Central Register of Controlled Trials (CENTRAL), Web of Science, Scopus, and Google Scholar were searched from inception through September 2025, combining MeSH and free-text terms for CS, acute myocardial infarction, heart failure, acute decompensated heart failure, non-ischaemic cardiomyopathy, and outcome descriptors (mortality, outcome, prognosis, comparative, versus), without language restriction. Reference lists of included articles, relevant systematic reviews, and major cardiology congress abstracts (American College of Cardiology, American Heart Association, European Society of Cardiology) were manually screened. The full search strategy is detailed in Supplementary Table S1.

Data collection process

Two investigators independently screened titles, abstracts, and full texts, with discrepancies resolved by consensus or a third senior investigator. Two investigators independently extracted data using a standardized six-domain form (study characteristics, demographics and comorbidities, presentation and shock severity markers, haemodynamic and echocardiographic parameters, management strategies, and clinical outcomes); the complete extraction template is reported in Supplementary Methods (S-Methods S2). Categorical data were captured as events/total (%), whereas continuous data were reported as mean ± standard deviation (SD) or median [interquartile range (IQR)], with medians and IQRs converted using validated estimators (e.g. Wan *et al.*). Discrepancies were resolved by consensus or third-reviewer adjudication; missing data were noted as 'not reported'.

Study risk of bias assessment

Methodological quality was independently assessed in duplicate using the Risk of Bias in Non-randomized Studies of Interventions (ROBINS-I) tool, evaluating seven domains.[20] Each study was categorized as low, moderate, serious, or critical risk, with disagreements adjudicated. Risk-of-bias judgements were not used to exclude studies but were incorporated into prespecified sensitivity analyses (excluding studies at serious or critical risk) and overall interpretation.

Statistical analysis

All statistical analyses were performed using R (version 4.3.3) with the meta, metafor, tidyverse, and ggplot2 packages.[21] Random-effects inverse-variance models with restricted maximum likelihood estimation were applied uniformly. The Hartung–Knapp adjustment was selected for CI construction over standard Wald intervals, given anticipated heterogeneity and modest contributing-study counts per outcome. Large language models served as supportive tools for eligibility screening, structured data extraction (Supplementary Table S2), and analytical code generation, with all outputs validated by human reviewers and final decisions remaining under investigator control; the full linear mixed models (LLM)-assisted workflow is documented in Supplementary Methods (S-Methods S3).

Pooled ORs with 95% CIs were estimated for dichotomous outcomes [cardiac arrest, SCAI D/E prevalence, tMCS utilization by device, complications, durable advanced therapies]. Pooled mean differences (MDs) with 95% CIs were calculated for continuous outcomes [demographics, left ventricular ejection fraction (LVEF), invasive haemodynamics, laboratory values, severity scores, resource utilization] using inverse-variance weighting. Effect measures were defined as AMI-CS minus ADHF-CS for continuous variables and AMI-CS vs ADHF-CS for binary outcomes. For haemodynamic continuous outcomes, complete-case inclusion was enforced; studies without means and SDs for both phenotypes were excluded *a priori*. Cohort overlap across publications from shared registries (CSWG, CCCTN, Altshock-2, Pittsburgh programmes) was managed through a dynamic per-outcome selection algorithm prioritizing the most recent publication with the largest sample size and greatest data completeness (Supplementary Table S3 and Figure S1), preventing patient double-counting whilst allowing different publications from the same registry to contribute to distinct analyses.

Prespecified sensitivity analyses examined robustness under alternative selection strata. Device-programme registries and mechanical support-focused cohorts were analysed separately to test whether estimates differed in procedure-exposed populations. Administrative and claims-based datasets were analysed in isolation, owing to differences in case ascertainment, diagnostic coding, covariate adjustment, and endpoint definitions, and were retained as parallel analyses rather than merged with primary syntheses. Heterogeneity was quantified using I^2 (low <25%; moderate 25–75%; high >75%) and τ^2. Sources of heterogeneity were explored through prespecified univariable meta-regression; the full covariate list and prespecified subgroup analyses appear in Supplementary Methods (S-Methods S4). Time-to-event data were pooled as hazard ratios where available. Publication bias was assessed using funnel plots, Egger's test, and trim-and-fill for outcomes with at least 10 contributing studies. A two-sided $P < .05$ was considered statistically significant.[22]

Results

Study selection

The systematic search across multiple electronic databases identified 4826 records (Figure 1): PubMed/MEDLINE ($n = 724$), Scopus ($n = 1689$), Web of Science ($n = 965$), Embase ($n = 1351$), and CENTRAL ($n = 97$). An additional 53 records were identified through citation searching of reference lists from relevant systematic reviews. After removing 1289 duplicate records and 44 non-English language publications, 3493 unique records underwent title and abstract screening.

During the initial screening, 3368 records were excluded because they consisted of review articles, case reports, editorials, conference abstracts, or studies that did not address phenotypic comparisons of CS. The remaining 125 reports from database searches and all 53 reports from citation searching were sought for full-text retrieval, with all 178 reports successfully obtained.

Full-text eligibility assessment was performed on 178 reports. Among database-sourced reports, 99 were excluded due to the absence of a direct AMI-CS vs ADHF-CS comparison ($n = 65$), ineligible comparator groups ($n = 22$), or review articles or editorials ($n = 12$). Among citation-sourced reports, 50 were excluded for no direct phenotypic comparison ($n = 28$), ineligible comparator groups ($n = 12$), and review or editorial format ($n = 10$).

Ultimately, 29 observational studies met all predetermined inclusion criteria and were included in the systematic review and meta-analysis. The study selection process adhered to PRISMA 2020 guidelines, ensuring methodological transparency and reproducibility.

Study characteristics

The 29 included studies (Table 1), published between 2019 and 2025, collectively enrolled 497 368 patients (264 684 AMI-CS; 232 684 ADHF-CS), reflecting nearly equal prevalence in contemporary practice.[17,23–50] The portfolio comprised multicentre registry analyses ($n = 14$), large-scale national database investigations ($n = 2$), and single-centre cohort studies ($n = 13$). Geographic distribution was predominantly North American ($n = 21$) and European ($n = 5$), with two large-scale Asian national registries. Sample sizes ranged from 32 patients in single-centre studies to over 329 000 in national database analyses. More recent data (≥2023) represented 69% of included studies ($n = 20$).

Risk of bias assessment

Using the ROBINS-I tool, none of the 29 studies achieved low overall risk of bias, reflecting the inherent challenges of observational CS research. The majority ($n = 19$; 65.5%) were judged at serious risk and

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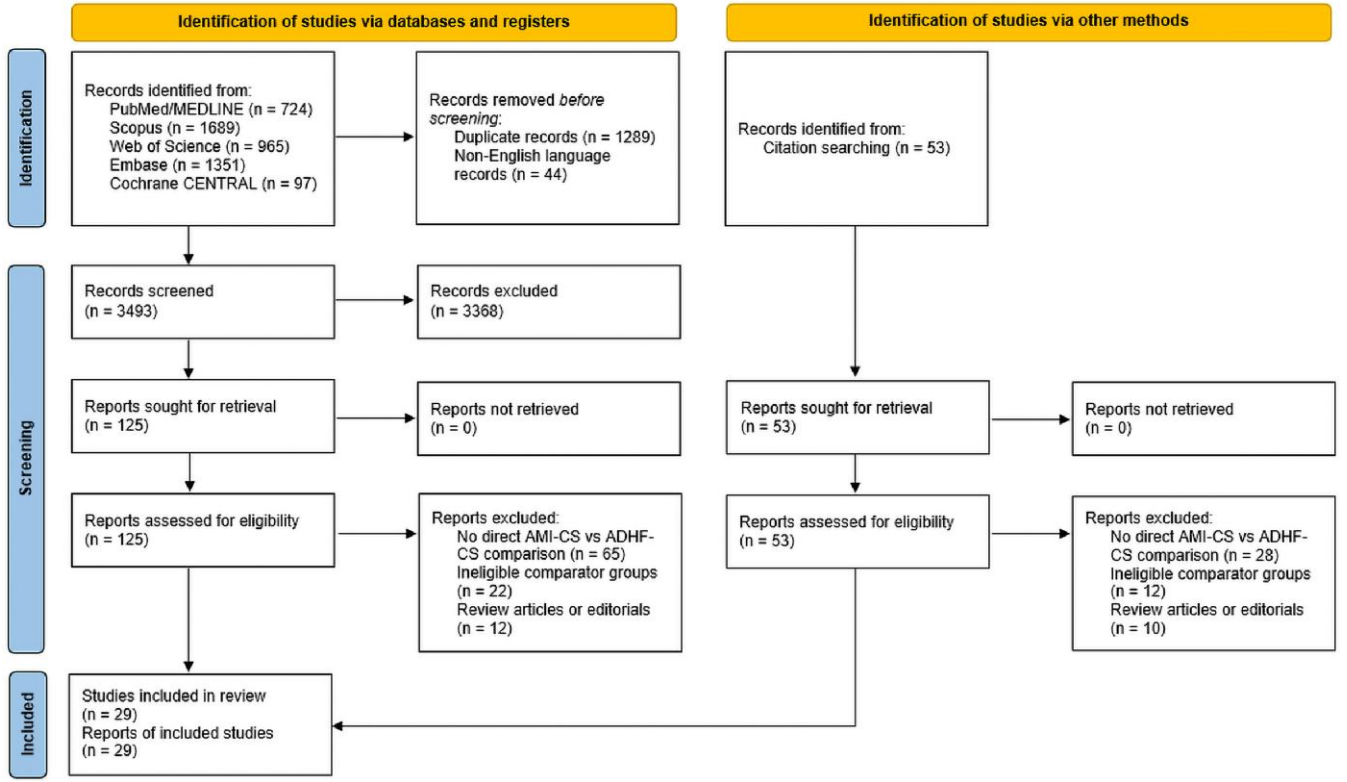

Figure 1 PRISMA flow diagram of study selection process.

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Figure 2 Risk of bias assessment summary using Risk of Bias in Non-randomized Studies of Interventions tool.

10 (34.5%) at moderate risk; no critical-risk classifications occurred. Confounding was the predominant methodological concern (62% serious risk), driven by unmeasured shock severity trajectories, treatment intensity, centre-level practice patterns, and transplant candidacy. Participant selection was the second leading bias source (24% serious risk), particularly in analyses restricted to mechanical support recipients.

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The absence of pre-registered analysis plans posed moderate risk across all studies in the selection-of-reported-results domain. Domain-specific evaluations are presented in *Figure 2*. Findings should be interpreted as adjusted prognostic associations rather than causal effects.

Clinical features at presentation

Pooled analyses revealed distinct baseline phenotypes. Patients with AMI-CS were older across seven studies (AMI-CS $n = 1505$; ADHF-CS $n = 1656$; MD 4.76 years, 95% CI .37–9.14; $P = .038$; $I^2 = 78\%$); a sensitivity analysis confirmed this with similar magnitude (Hartung-Knapp [HK] $P = .065$).

Patients with AMI-CS had higher ejection fraction at presentation (seven studies; AMI-CS $n = 1725$; ADHF-CS $n = 2635$; MD 6.04%, 95% CI 4.53–7.55; $P < .001$; $I^2 = 58\%$). LVEF was extractable in only ≈.88% of the overall pooled population (4360/497 368), so this estimate derives from selected sub-cohorts. The numerical difference should not be interpreted as preserved pump function: systolic reserve in AMI-CS is acutely exhausted, and LVEF is a poor surrogate for shock severity in this setting. A sensitivity analysis confined to three device-programme cohorts preserved direction and magnitude (common-effect MD 6.01 percentage points; $I^2 = 89\%$), likely reflecting candidacy criteria.[29,41,44]

Conversely, invasive haemodynamic parameters were similar between phenotypes. Across four studies, right atrial pressure (RAP)/central venous pressure (CVP) did not differ (AMI-CS $n = 236$; ADHF-CS $n = 595$; MD −1.40 mmHg, 95% CI −3.60–.81); pulmonary capillary wedge pressure across three studies was similarly non-discriminative (AMI-CS $n = 756$; ADHF-CS $n = 1659$; MD −.22 mmHg, 95% CI −4.51–4.07). A sensitivity analysis limited to two mechanical support programme studies confirmed this null result (HK $P = .350$).[41,44] These haemodynamic estimates derive from very small sub-cohorts [RAP/CVP, ≈.17%; pulmonary capillary wedge pressure (PCWP), ≈.49% of the pooled population]. Pooled MDs for age, ejection fraction, RAP/CVP, and PCWP appear in *Figure 3A–D*; contributing studies and patients per endpoint varied substantially (range 2–27 studies; 803–141 602 patients), and Supplementary Table S5 summarizes data availability for every pooled analysis.

Shock severity and cardiac arrest burden

AMI-CS patients exhibited more severe clinical phenotypes. Cardiac arrest occurred more frequently in AMI-CS across five studies (AMI-CS $n = 50\,778$; ADHF-CS $n = 90\,824$; representing 28.5% of the pooled population; OR 2.03, 95% CI 1.04–3.98; $P = .039$; $I^2 = 98\%$), with extreme heterogeneity reflecting variations in institutional practice. Out-of-hospital cardiac arrest exhibited a stronger association with AMI-CS (OR 3.04, 95% CI 2.09–4.43; four studies; AMI-CS $n = 726$; ADHF-CS $n = 1198$; $I^2 = 0\%$), consistent with ischaemic-substrate arrhythmic instability. SCAI Stage D/E was also more prevalent in AMI-CS (four studies; AMI-CS $n = 1708$; ADHF-CS $n = 3417$; representing 1.03% of the pooled population; OR 1.50, 95% CI 1.33–1.70; $P < .001$; $I^2 = 0\%$); a device-registry sensitivity yielded an enhanced effect (common-effect OR 3.16, 95% CI 2.01–4.95; HK $P = .072$), suggesting that AMI-CS is associated with greater haemodynamic compromise particularly among mechanical support populations. Pooled ORs for these outcomes appear in *Figure 4A–C*.

Temporary mechanical circulatory support utilization

Temporary mechanical circulatory support was consistently more frequent in AMI-CS than ADHF-CS. Across six studies (AMI-CS $n = 2259$; ADHF-CS $n = 3880$), any temporary support yielded an OR of 5.44 (95% CI 2.83–10.46; $P < .001$; $I^2 = 89\%$), likely reflecting institutional practice variation and temporal evolution in device availability. Device-specific syntheses confirmed increased utilization across all

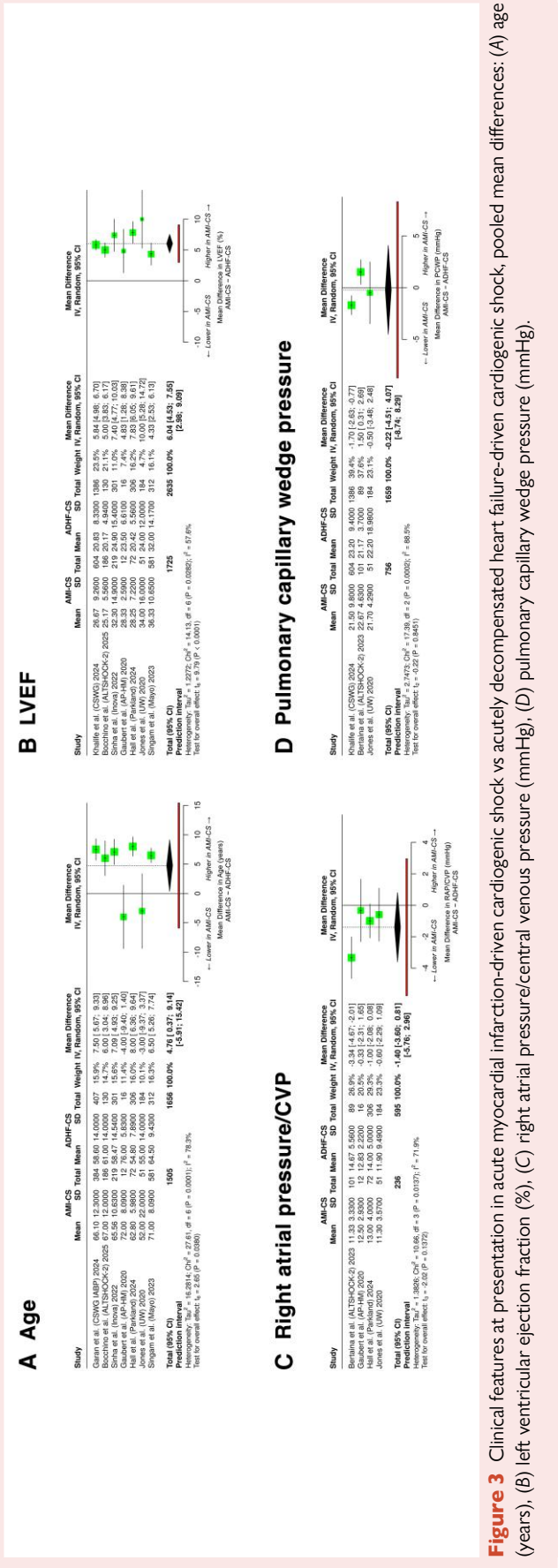

Figure 3 Clinical features at presentation in acute myocardial infarction-driven cardiogenic shock vs acutely decompensated heart failure-driven cardiogenic shock, pooled mean differences: (*A*) age (years), (*B*) left ventricular ejection fraction (%), (*C*) right atrial pressure/central venous pressure (mmHg), (*D*) pulmonary capillary wedge pressure (mmHg).

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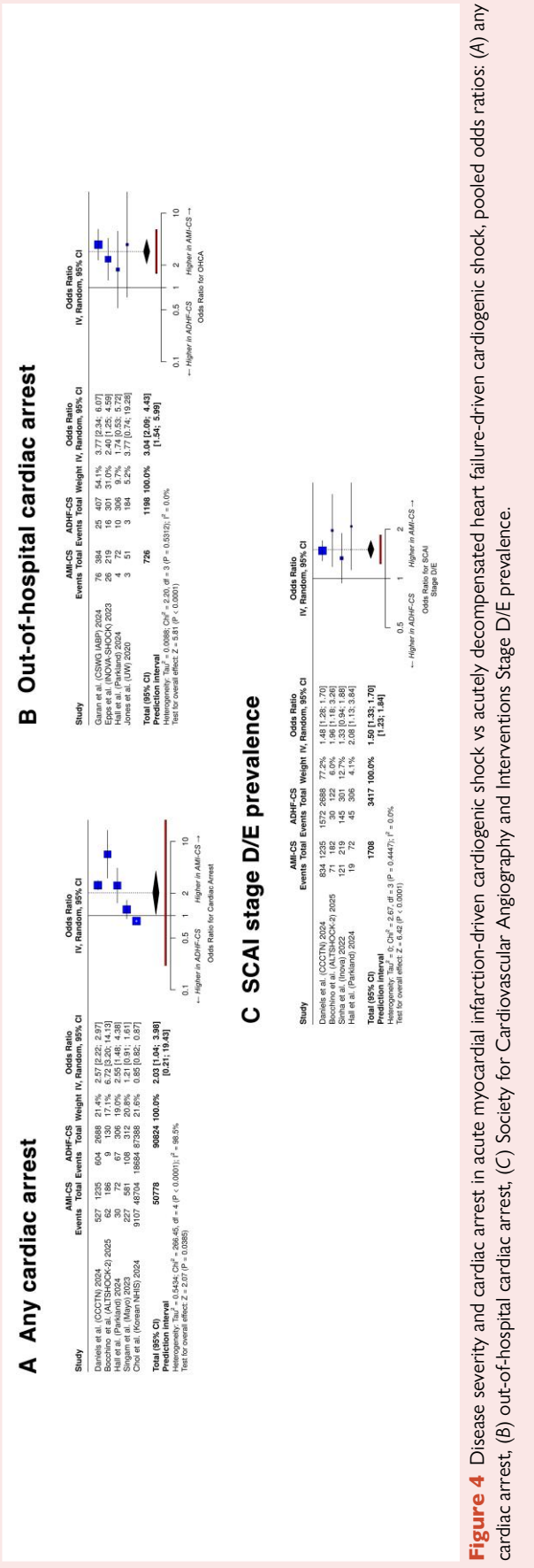

Figure 4 Disease severity and cardiac arrest in acute myocardial infarction-driven cardiogenic shock vs acutely decompensated heart failure-driven cardiogenic shock, pooled odds ratios: (*A*) any cardiac arrest, (*B*) out-of-hospital cardiac arrest, (*C*) Society for Cardiovascular Angiography and Interventions Stage D/E prevalence.

modalities: intra-aortic balloon pump (IABP) in five studies (AMI-CS $n = 1589$; ADHF-CS $n = 3638$; OR 4.73, 95% CI 2.02–11.08), percutaneous ventricular assist devices (pVADs) in four studies (AMI-CS $n = 975$; ADHF-CS $n = 1960$; OR 2.67, 95% CI 1.68–4.25), and veno-arterial extracorporeal membrane oxygenation (VA-ECMO) in three studies (AMI-CS $n = 371$; ADHF-CS $n = 574$; OR 3.27, 95% CI 1.23–8.69). Pooled ORs appear in *Figure 5A–D*.

Early clinical outcomes and resource utilization

AMI-CS was associated with higher short-term mortality across seven studies (AMI-CS $n = 2356$; ADHF-CS $n = 3937$; representing 1.27% of the pooled cohort; OR 1.58, 95% CI 1.06–2.37; $P = .026$; $I^2 = 82\%$). A device-programme sensitivity analysis demonstrated significantly amplified excess mortality (pooled OR 2.13, 95% CI 1.61–2.81; $P = .047$; Supplementary Table S4), indicating that the mortality differential is most pronounced in mechanical support-incorporated populations.

Heart transplant or durable left ventricular assist device (LVAD) during the index admission was significantly less frequent in AMI-CS (three studies; AMI-CS $n = 224$; ADHF-CS $n = 579$; OR .09, 95% CI .03–.24; $P < .001$; $I^2 = 0\%$), consistent with myocardial recovery potential after revascularization vs end-stage disease in ADHF-CS. Renal replacement therapy (RRT) did not differ across five studies (AMI-CS $n = 3854$; ADHF-CS $n = 7123$; OR .89, 95% CI .55–1.46; $I^2 = 91\%$) but was strongly setting dependent (Supplementary Table S4): device-incorporated cohorts showed significantly higher RRT use in AMI-CS (pooled OR 1.53, 95% CI 1.13–2.06; $P = .021$), whereas administrative and claims datasets indicated reduced odds in AMI-CS (pooled OR .60, 95% CI .59–.62), likely reflecting greater chronic kidney disease burden in claims-based ADHF-CS phenotyping.

Major bleeding was modestly more frequent in AMI-CS across two studies (AMI-CS $n = 1079$; ADHF-CS $n = 820$; OR 1.36, 95% CI 1.02–1.82; $P = .035$); the device-incorporated sensitivity preserved direction with larger effect (common-effect OR 2.29; Supplementary Table S4). Pooled ORs for short-term mortality, durable LVAD or transplant, RRT, and major bleeding appear in *Figure 6A–D*.

Sensitivity analyses: summary interpretation

Across sensitivity strata, primary findings remained directionally consistent except for RRT, where data source modified direction of association (registry OR 1.53 vs claims OR .60). Effect-magnitude amplification was observed in device cohorts for short-term mortality (OR 2.13 vs 1.58) and SCAI Stage D/E (OR 3.16 vs 1.50). The full-sensitivity matrix appears in Supplementary Table S4. Among dichotomous outcomes, only short-term mortality included sufficient studies for formal publication-bias assessment; funnel-plot inspection and Egger's test identified no evidence of bias (Supplementary Figure S2). *Table 2* summarizes phenotypic characteristics, management patterns, and clinical outcomes.

Discussion

This systematic synthesis of 29 observational studies establishes that AMI-CS and ADHF-CS represent clinically distinct presentations of CS. Three findings warrant emphasis: AMI-CS presents with higher SCAI stage and greater cardiac arrest burden, receives more temporary mechanical support across all device classes, and carries higher short-term mortality, with effect magnitudes varying considerably across data sources.

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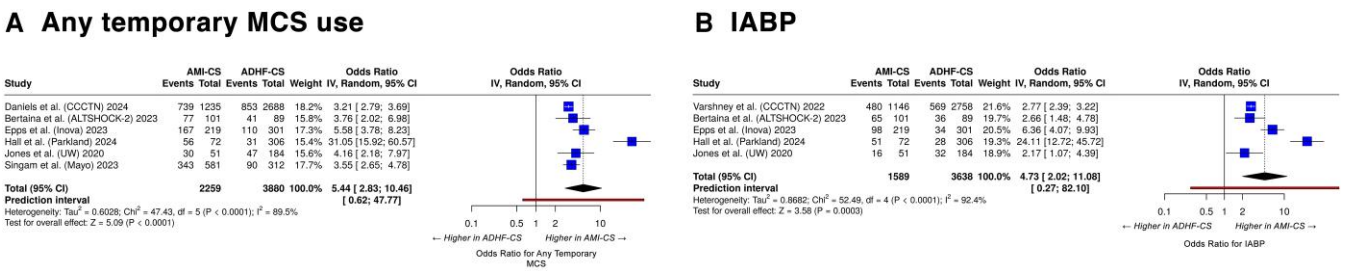

Figure 5 Temporary mechanical circulatory support exposure in acute myocardial infarction-driven cardiogenic shock vs acutely decompensated heart failure-driven cardiogenic shock, pooled odds ratios: (*A*) any temporary mechanical circulatory support, (*B*) intra-aortic balloon pump, (*C*) percutaneous ventricular assist device (Impella), (*D*) veno-arterial extracorporeal membrane oxygenation.

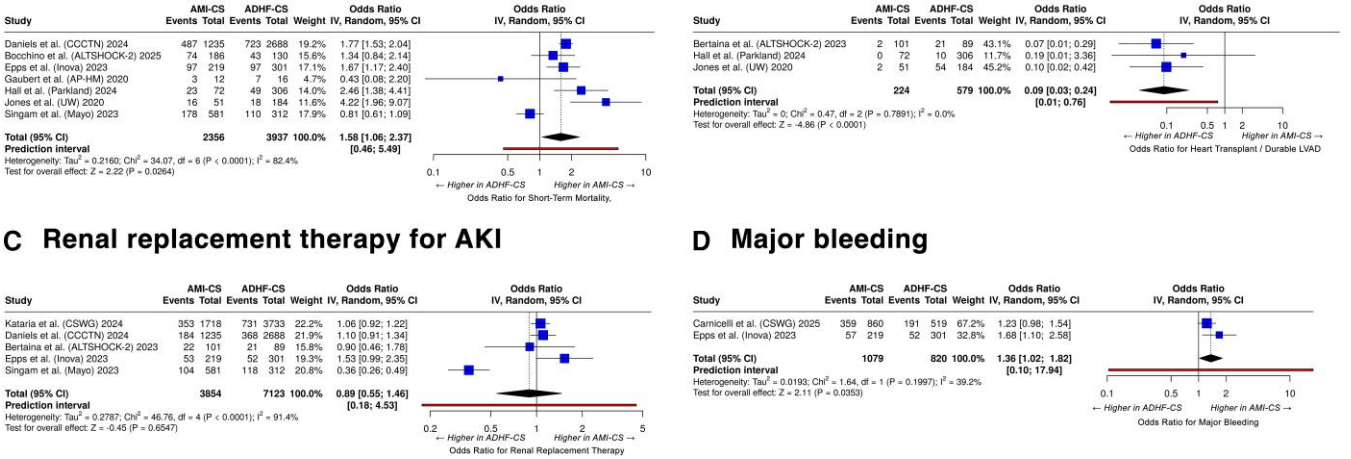

Figure 6 Early outcomes and resource utilization in acute myocardial infarction-driven cardiogenic shock vs acutely decompensated heart failure-driven cardiogenic shock, pooled odds ratios: (*A*) short-term mortality (in-hospital/30-day), (*B*) heart transplant or durable LVAD during index admission, (*C*) renal replacement therapy for acute kidney injury, (*D*) major bleeding.

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Table 1 Baseline characteristics of included studies

Study	Study design and region	Total patients (*N*)	N (AMI-CS/ ADHF-CS)	Age (years)[a]	Male (%)[a]	LVEF (%)[a]	SCAI Stage D/E (%)[a]
Alhuneafat *et al.* (2025)[17]	National database/USA	329 280	204 660/124 620	68.2/64.5	65.4/69.5	NR/NR	NR/NR
Bertaina *et al.* (2023)[23]	Registry/Europe	190	101/89	67.0/63.0	77.2/8.9	25.0/20	43.6/25.8
Bocchino *et al.* (2025)[24]	Registry/Europe	316	186/130	67.0/61.0	80.0/8.8	25.0/20	39.0/24.6
Carnicelli *et al.* (2026)[25]	Registry/USA	1379	860/519	64.0/60	76.0/73.6	NR/NR	97.7/99.2
Choi *et al.* (2024)[26]	National database/Asia	136 092	48 704/87 388	NR/72.6	67.5/53.7	NR/NR	NR/NR
Daniels *et al.* (2024)[27]	Registry/N. America	3923	1235/2688	NR/NR	68.9/67.7	NR/NR	67.5/58.5
Epps *et al.* (2023)[28]	Single-centre/USA	520	219/301	NR/NR	71.7/7.4	30.0/20	NR/NR
Fried *et al.* (2024)[29]	Registry/USA	694	210/484	63.0/56.6	81.4/84.5	22.3/17.8	86.7/68.1
Garan *et al.* (2020)[32]	Registry/USA	1206	494/712	NR/NR	NR/NR	NR/NR	NR/NR
Garan *et al.* (2024)[31]	Registry/USA	1648	593/1055	65.2/6.3	70.8/69.8	28.4/23.7	90.7/82.5
Garan *et al.* (2025)[30]	Registry/USA	791	384/407	66.1/58.6	71.1/73.7	NR/NR	49.7/69.1
Gaubert *et al.* (2020)[33]	Single-centre/Europe	28	12/16	72.0/78.0	75.0/81.2	30.0/23.0	NR/NR
Guglin *et al.* (2024)[42]	Registry/USA	2993	969/2024	NR/NR	NR/NR	NR/NR	NR/NR
Hall *et al.* (2025)[34]	Single-centre/USA	378	72/306	60.6/55.7	68.1/77.8	28.0/20	26.4/14.7
Hong *et al.* (2024)[35]	Single-centre/USA	133	47/86	64.0/59.0	83.0/75.6	NR/NR	NR/NR
Jones *et al.* (2020)[36]	Single-centre/USA	235	51/184	52.0/55.0	74.5/76.6	34.0/24.0	NR/NR
Kanwar *et al.* (2025)[37]	Registry/USA	873	246/627	NR/NR	NR/NR	NR/NR	NR/NR
Kataria *et al.* (2025)[38]	Registry/USA	5451	1718/3733	NR/NR	72.5/70	NR/NR	62.3/47.7
Khalife *et al.* (2024)[39]	Registry/USA	1990	604/1386	67.0/62.0	70.2/72.5	25.0/20	53.6/35.0
Kochar *et al.* (2024)[40]	Registry/USA	5922	1951/3971	NR/NR	NR/NR	NR/NR	NR/NR
Malick *et al.* (2019)[41]	Single-centre/USA	205	73/132	71.8/61.2	50.7/84.1	30.2/18.0	NR/NR
Modi *et al.* (2023)[43]	Single-centre/USA	44	20/24	54.5/49.6	65.0/58.3	NR/NR	80.0/83.3
Sicke *et al.* (2024)[44]	Single-centre/USA	67	23/44	64.0/54.0	78.0/77.0	21.0/17.0	82.6/50
Singam *et al.* (2023)[45]	Single-centre/USA	893	581/312	71.0/65.0	64.7/6.9	35.0/28.0	NR/NR
Sinha *et al.* (2022)[46]	Single-centre/USA	520	219/301	65.6/58.5	71.7/7.4	32.3/24.9	55.3/48.1
Thayer *et al.* (2020)[47]	Registry/USA	1206	494/712	64.9/57.9	69.0/76.3	30.9/2.2	74.0/51.7
Ton *et al.* (2023)[48]	Registry/USA	4267	1492/2775	65.5/6.1	69.4/71.8	NR/NR	NR/NR
Varshney *et al.* (2025)[49]	Registry/N. America	3904	1146/2758	67.0/NR	69.3/67.7	NR/NR	27.7/19.0
Zweck *et al.* (2023)[50]	Registry/USA	631	286/345	NR/NR	72.0/7.7	NR/NR	NR/NR

AMI-CS, acute myocardial infarction-driven cardiogenic shock; ADHF-CS, acute decompensated heart failure-driven cardiogenic shock; LVEF, left ventricular ejection fraction; *N*, number of patients; NR, not reported; SCAI, Society for Cardiovascular Angiography and Interventions.
[a]Data are presented as mean for continuous variables or percentage for categorical variables, in the following format: (value for AMI-CS group/value for ADHF-CS group). Standard deviations are omitted for brevity.

These findings inform whether management should be guided primarily by aetiology or by universal severity staging and support a dual-axis framework in which severity defines the immediate strategy and aetiology defines the therapeutic endpoint. SCAI stage classification appears an important mediator of outcomes, although the limited direct contribution of SCAI-staged patients (≈1% of the cohort) tempers this inference.[46,51–57] AMI-CS patients consistently presented with higher rates of cardiac arrest, greater prevalence of SCAI stage D/E, and more frequent rescue MCS, raising the possibility that worse outcomes are driven primarily by advanced shock severity rather than aetiology *per se*; the severity gradient persisted in device-incorporated cohorts, implying a genuine phenotypic distinction rather than a selection artefact. Conversely, isolated invasive filling pressures did not discriminate phenotypes, a uniform null across general and mechanical support cohorts that probably reflects biventricular interaction, variable resuscitation, and timing of measurement rather than physiological equivalence.[33] The dual-axis framework is therefore proposed as an organizing principle for prospective validation rather than as a clinical prescription.

Therapeutically, AMI-CS typically follows a bridge-to-recovery logic centred on rapid revascularization and short-term mechanical support.[58] ADHF-CS, in contrast, demands early systematic identification of the underlying precipitant of decompensation, including ischaemia in chronic cardiomyopathy, uncontrolled arrhythmia, acute valvular lesion, or systemic precipitants, such as infection, thyrotoxicosis, severe anaemia, or non-adherence; the corrective strategy and candidacy assessment for advanced therapies depend on this identification.[59] Destination therapy with durable LVAD or transplantation was substantially less frequent in AMI-CS, reflecting myocardial recovery potential after revascularization vs end-stage disease in ADHF-CS.[60] Higher tMCS use in AMI-CS carries a high risk of confounding by indication, since MCS is deployed predominantly in patients with cardiac arrest and at advanced shock stages; mortality amplification in device-incorporated cohorts (OR 2.13 vs 1.58) likely reflects severity selection alongside the procedural risks of urgent mechanical support, including bleeding, vascular access complications, and limb ischaemia.

Phenotype-specific valvular considerations further shape MCS decision-making and likely contribute to the divergent utilization observed. In accordance with the Heart Failure Association (HFA)/Association for Acute CardioVascular Care (ACVC)/European Association of Percutaneous Cardiovascular Interventions (EAPCI)

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Table 2 Comparative summary of phenotype, management, and outcomes in AMI-CS vs ADHF-CS

Domain	AMI-CS	ADHF-CS	Clinical interpretation
Phenotype			
Onset	Acute ischaemic insult	Acute-on-chronic decompensation	Aetiology defines the biological substrate
Age	Older (+4.76 years) (7 studies; .64% of pooled *N*)	Younger	Higher frailty burden in AMI-CS
Cardiac arrest	Higher (OR 2.03) (5 studies; 28.5% of pooled *N*)	Lower	Ischaemic aetiology drives arrhythmic instability
SCAI Stage D/E	Higher prevalence (OR 1.50) (4 studies; 1.03% of pooled *N*)	Lower prevalence	AMI-CS presents more fulminantly; estimate derived from a small sub-cohort
LVEF	Higher (+6.04%) (7 studies; .88% of pooled *N*)	Lower (chronic remodelling)	Higher LVEF in AMI-CS observed in a small sub-cohort; does not imply preserved pump reserve—systolic reserve is acutely exhausted
Comorbidity burden	Lower	Higher (CKD, AF)	ADHF-CS complexity driven by chronic organ dysfunction
Haemodynamics			
RAP/CVP	No significant difference (4 studies; .17% of pooled *N*)	No significant difference	Static filling pressures do not discriminate phenotypes; limited data
PCWP	No significant difference (3 studies; .49% of pooled *N*)	No significant difference	Biventricular dysfunction and resuscitation timing confound values
Management			
Strategy	Bridge-to-recovery	Bridge-to-decision/advanced therapy (preceded by early identification of the underlying cause)	Severity defines the strategy; aetiology defines the endpoint
Any tMCS	Markedly higher (OR 5.44)	Lower	AMI-CS often requires rescue MCS
IABP	Higher (OR 4.73)	Lower	Traditional support in ischaemic shock
pVAD/Impella	Higher (OR 2.67)	Lower	Active unloading strategy; contraindicated in significant aortic regurgitation (more common in ADHF-CS)[a]
VA-ECMO	Higher (OR 3.27)	Lower	Rescue for refractory shock
Destination therapy	Rare (OR .09) (3 studies; .16% of pooled *N*)	Frequent	AMI-CS: recovery potential; ADHF-CS: end-stage disease. In mixed/registry cohorts; claims-based data show the same direction with high heterogeneity
Outcomes			
Short-term mortality	Higher (OR 1.58) (7 studies; 1.27% of pooled *N*)	Lower	Driven by interaction of shock severity and ischaemic injury
Mortality in MCS cohorts	Amplified (OR 2.13)	Lower	Reflects selection into device-program registries; not generalizable to all AMI-CS
Major bleeding	Higher (OR 1.36)	Lower	Aggressive antithrombotics + large-bore access
RRT requirement	Setting-dependent	Setting-dependent	Direction reversal across data sources (registries OR 1.53, AMI-CS higher; claims OR .60, ADHF-CS higher)

ACVC, Association for Acute CardioVascular Care; ADHF-CS, acutely decompensated heart failure-driven cardiogenic shock; AF, atrial fibrillation; AMI-CS, acute myocardial infarction-driven cardiogenic shock; CKD, chronic kidney disease; CVP, central venous pressure; EAPCI, European Association of Percutaneous Cardiovascular Interventions; HFA, Heart Failure Association; IABP, intra-aortic balloon pump; LVEF, left ventricular ejection fraction; MCS, mechanical circulatory support; MD, mean difference; *N*, number of patients; OR, odds ratio; PCWP, pulmonary capillary wedge pressure; pp, percentage points; pVAD, percutaneous ventricular assist device (e.g. Impella); RAP, right atrial pressure; RRT, renal replacement therapy; SCAI, Society for Cardiovascular Angiography and Interventions; tMCS, temporary mechanical circulatory support; VA-ECMO, veno-arterial extracorporeal membrane oxygenation.

[a]Per the HFA/ACVC/EAPCI scientific statement on acute heart failure and valvular heart disease (Chioncel O, Adamo M, Nikolaou M, et al. Eur J Heart Fail 2023;25(7):1025–1048; doi:10.1002/ejhf.2918), temporary MCS devices that rely on antegrade left ventricular unloading are contraindicated in moderate-to-severe aortic regurgitation, which is more prevalent in ADHF-CS.

scientific statement on acute heart failure and valvular heart disease,[61] devices reliant on antegrade left ventricular unloading (IABP, axial-flow pVAD) are contraindicated or unfavourable in moderate-to-severe aortic regurgitation, a lesion disproportionately encountered in ADHF-CS rather than AMI-CS. These considerations support routine early echocardiographic phenotyping before MCS escalation.

Clinical implications

Several practical inferences, all hypothesis generating, follow. In AMI-CS, the combination of hyperacute presentation and intensive mechanical support strategies is associated with consistently higher rates of major bleeding, persisting in device-incorporated cohorts; haemorrhagic complications likely reflect aggressive antithrombotic

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therapy, higher procedural burden, and large-bore vascular access alongside acute kidney injury.[62–64] Setting-dependent RRT signals support institution-level harm-reduction protocols, including meticulous large-bore access management, limb-perfusion surveillance, device-tailored anticoagulation, and contrast-sparing workflows.[65] Although granular pharmacological data were not pooled, aetiology likely dictates vasoactive strategy, with vasopressors often favoured in the vasodilatory phase of AMI-CS vs inotropes in the congested phenotype of ADHF-CS.[13] In ADHF-CS, lower short-term mortality should not justify therapeutic de-escalation, as observational trajectories indicate possible late risk after the first month, supporting early advanced HF involvement, guideline-directed therapy optimization, and structured post-discharge surveillance.[46,66,67] Across aetiologies, reliance on isolated haemodynamic thresholds is insufficient; integrated indices (organ perfusion pressure, cardiac power output trajectory, pulmonary artery pulsatility) provide more actionable prognostic information than static values.[32,68,69]

Limitations

With 19 of 29 included studies judged at serious risk of bias, predominantly from confounding and selection, findings should be regarded as hypothesis generating rather than definitive. Phenotype-defining variables were available in only a small proportion of the pooled cohort: of 497 368 patients, LVEF was extractable in .88%, SCAI stage in 1.03%, right atrial pressure in .17%, and pulmonary capillary wedge pressure in .49%. Administrative and claims datasets, which contributed >93% of the pooled sample, did not report these variables. Pooled estimates for LVEF, SCAI stage, and invasive haemodynamics were therefore derived from device-programme registries and single-centre studies rather than from the overall population (Supplementary Table S5). The evidence is observational, with attendant risks of selection bias and residual confounding. Substantial between-study heterogeneity reflects variation in populations, era, device availability, practice patterns, and outcome definitions; variable timing of SCAI stage assessment may further affect severity comparability. Cohort overlap was managed through dynamic per-outcome selection at the expense of variable study counts by endpoint. Study-level data preclude patient-level risk adjustment and finer phenotyping (cardiorenal vs cardiometabolic shock subtypes), and incomplete reporting constrained specific analyses (stroke, limb ischaemia). Administrative and claims-based datasets, analysed separately due to disparities in case capture and phenotype classification, produced distinct signals (particularly for renal outcomes) reflecting residual confounding that aggregation cannot resolve. These factors collectively support cautious interpretation and prospective standardized data collection, with individual patient-data meta-analysis where feasible.[70]

Conclusion

AMI-CS and ADHF-CS represent clinically distinct presentations of CS. AMI-CS is associated with more fulminant features at presentation, greater reliance on tMCS, and higher short-term mortality, whereas ADHF-CS is characterized by greater chronic comorbidity burden and more frequent need for durable advanced therapies. Effect magnitudes vary across data sources, and several phenotype-defining estimates derive from sub-cohorts representing a small fraction of the pooled population. These findings are hypothesis generating and support, but do not establish, a dual-axis framework integrating severity with aetiology, requiring validation in prospective phenotype-rich cohorts before informing individualized clinical decision-making.

Author contributions

M.B.Y. and M.E.K. led conceptualization, data curation, formal analysis, investigation, and drafting. M.B.Y. provided supervision and validation. M.E.K. led visualization. J.B., R.C., O.G., E.O., M.P., and M.M. contributed to formal analysis, investigation, drafting, and critical revision for important intellectual content. All authors reviewed, edited, and approved the final manuscript and agreed to be accountable for all aspects of the work.

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Large language models (Claude Sonnet, Google Gemini, and ChatGPT) were used as supportive tools for eligibility screening, structured data extraction, and generation of R analytical code, as detailed in the Methods and in Supplementary Methods (S-Methods S3) and Table S2. All outputs were verified by human reviewers, and final decisions on study inclusion, data interpretation, and manuscript content remained under the investigators' control.

Supplementary data

Supplementary data are available at *European Journal of Heart Failure* online.

Declarations

Disclosure of Interest

M.B.Y. has received institutional fees from Novartis, Bayer, Amgen, Novo Nordisk, AstraZeneca, and Boehringer Ingelheim. M.E.K. declares no conflicts of interest. J.B. has received honoraria from Bayer, Boehringer Ingelheim, and AstraZeneca for lectures and from Alleviant Medical, Reprieve Cardiovascular, and WhiteSwell for participation in clinical trial advisory boards. R.C. declares no conflicts of interest. O.G. has received personal fees from AstraZeneca, Bayer, Servier, Boehringer Ingelheim, Novartis, Novo Nordisk, and Vifor Pharma. E.O. declares no conflicts of interest. M.P. has received personal fees from Abbott Vascular, AstraZeneca, Bayer, Boehringer Ingelheim, Eli Lilly, Novartis, Novo Nordisk, Roche Diagnostics, Viatris, and Vifor Pharma. M.M. has received consulting fees from Abbott, AstraZeneca, and Bayer.

Data Availability

This systematic review and meta-analysis was conducted on aggregate data extracted from previously published studies; no new individual patient data were generated. All primary data sources are cited in the reference list and detailed in Supplementary Table S1. The review protocol is prospectively registered with PROSPERO (CRD420251242655).

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Ethical Approval

Ethical approval was not required.

Pre-registered Clinical Trial Number

None supplied.

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