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In-Hospital Outcomes of Patients with Right Bundle Branch Block (RBBB) and Anterior Wall ST-Segment Elevation Myocardial Infarction (AW STEMI) Undergoing Primary Percutaneous Coronary Angioplasty

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Abstract

Background: Anterior wall ST-segment elevation myocardial infarction (AW-STEMI) with right bundle branch block (RBBB) signals extensive septal ischemia and may portend higher in-hospital risk despite contemporary primary percutaneous coronary intervention (PPCI).
Objective: To estimate in-hospital outcomes and identify bedside predictors of mortality among patients with AW-STEMI and RBBB treated with PPCI in a standardized care pathway.
Methods: We performed a descriptive cross-sectional study at a tertiary cardiac center using consecutive enrollment over six months. Adults (≥18 years) with AW-STEMI and RBBB undergoing PPCI within 24 hours were included; prior MI/CABG, significant valvular disease/cardiomyopathy, or refusal of consent were exclusions. Prespecified outcomes were abstracted via a standardized case-report form. Continuous data are reported as mean (SD) or median (IQR); categorical data as n/N (%). A parsimonious multivariable logistic model (events-per-parameter constrained) explored predictors of in-hospital mortality.

Results: Of 146 activations, 130/146 (89.0%) met criteria (mean age 58.7 ± 11.8 years; male 104/130 [80.0%]). Median symptom-to-door time was 180 minutes (IQR 120–300); door-to-balloon 78 minutes (62–94), with >90 minutes in 39/130 (30.0%). Culprit was LAD in 120/130 (92.3%); proximal LAD 77/130 (59.2%). Pre-PCI TIMI 0 occurred in 89/130 (68.5%); post-PCI TIMI 3 in 117/130 (90.0%). In-hospital mortality was 12/130 (9.2%; 95% CI, 4.9–15.6). MACE occurred in 20/130 (15.4%; 9.7–22.8); reinfarction 5/130 (3.8%); stroke 3/130 (2.3%); VT/VF 14/130 (10.8%); complete heart block requiring pacing 11/130 (8.5%); acute heart failure 24/130 (18.5%); mechanical circulatory support 11/130 (8.5%). Median length of stay was 4 days (3–6). Killip III–IV independently predicted mortality (adjusted OR 3.85; 95% CI, 1.32–11.25); age per decade showed a moderate association (1.62; 1.08–2.64); symptom-to-door >180 minutes trended toward harm (2.27; 0.78–6.55). Model performance was fair (AUC 0.79; 95% CI, 0.68–0.90).
Conclusion: In AW-STEMI with RBBB, in-hospital risk concentrates among patients with shock physiology, older age, and treatment delays, and is mitigated when post-PCI TIMI 3 flow is achieved. System strategies to compress total ischemic time, prioritize early hemodynamic stabilization, and ensure meticulous reperfusion may reduce near-term mortality in this high-risk phenotype.

Introduction

Acute AW-STEMI is a time-critical emergency where rapid culprit-artery reperfusion via PPCI reduces infarct size and short-term mortality and anchors contemporary guideline-directed care (door-to-balloon targets, comprehensive antithrombotic therapy, secondary prevention) (1,2). Conduction disturbances at presentation are common; among these, RBBB reflects ischemia of the right bundle coursing through the interventricular septum—often with proximal left anterior descending (LAD) occlusion—implying larger ischemic territories, hemodynamic instability, and arrhythmic risk (3,4). Current European and North American guidelines classify bundle-branch block in acute coronary syndromes as a high-risk feature warranting immediate invasive evaluation and prompt reperfusion when STEMI is suspected (1,2). Historically, outcomes data for RBBB in AMI were confounded by mixed eras and heterogeneous infarct locations. Contemporary syntheses and real-world datasets have clarified risk: new-onset RBBB in AMI associates with higher mortality, ventricular arrhythmias, and cardiogenic shock (5,6); in AW-STEMI specifically, RBBB portends higher in-hospital mortality and more heart failure, complete heart block, and pacemaker implantation than AW-STEMI without RBBB (7). Mechanistically and clinically, new-onset RBBB has been linked to early ventricular fibrillation in STEMI, underscoring electrical instability during the most vulnerable window (4).

Evidence from South Asia and other resource-limited settings remains sparse, and few studies report granular in-hospital outcomes under standardized PPCI pathways. We therefore examined in-hospital outcomes among patients presenting with AW-STEMI and RBBB undergoing PPCI in a contemporary single-center cohort, focusing on mortality, MACE, arrhythmias, heart failure, circulatory support, and timeliness metrics to inform bedside risk stratification and system pathways (1,2,4–7).

Methods

Study design and setting

We conducted a descriptive cross-sectional study in the Department of Adult Cardiology, Peshawar Institute of Cardiology (PIC), Peshawar, Pakistan. Consecutive eligible patients were enrolled over six months following institutional approval (PIC-IRB No. IRC/25/242). Care pathways were aligned with contemporary international guidance (1,2).

Participants
Adults (≥18 years) with AW-STEMI and RBBB on the index 12-lead ECG undergoing PPCI within 24 hours of symptom onset were eligible. We excluded prior MI or CABG, significant valvular disease or cardiomyopathy, and refusal of consent. Eligibility was verified by ECG interpretation and angiographic review.

Clinical care and definitions
All patients received guideline-directed STEMI therapy (aspirin plus a P2Y12 inhibitor; parenteral anticoagulation; selective glycoprotein IIb/IIIa inhibitors, aspiration thrombectomy, vasopressors, or temporary pacing per operator judgment). Door-to-balloon (D2B) targets were ≤90 minutes for direct presentations and ≤120 minutes for inter-facility transfers (1,2). Epicardial flow was graded by TIMI criteria; endpoint terminology followed Academic Research Consortium-2 definitions where applicable to enhance comparability (10).

Outcomes
The primary outcome was in-hospital all-cause mortality. Secondary outcomes included MACE (death, reinfarction, stroke), ventricular tachyarrhythmias (VT/VF), complete heart block (CHB) requiring temporary or permanent pacing, cardiogenic shock, acute heart failure, use of mechanical circulatory support (IABP/ECMO), and length of stay.

Data collection and quality assurance
Trained abstractors recorded demographics, comorbidities, presentation features (symptom duration, Killip class, hemodynamics), ECG characteristics (new-onset vs persistent RBBB), angiography (culprit vessel, pre/post-PCI TIMI), procedural metrics (D2B, stent strategy, adjuncts), and outcomes onto a standardized case-report form. Automated range/logic checks were applied; discrepancies were resolved against source records prior to database lock.

Sample size
We used precision-based estimation for the primary endpoint (in-hospital mortality) at 95% confidence, absolute precision ±5%, and anticipated proportion ~9–10%, yielding a minimum N≈130 (8). The anticipated rate was anchored to contemporary AW-STEMI series and RBBB-specific datasets (7,12,16).

Statistical analysis
Analyses used SPSS v28 (or equivalent). Continuous variables are summarized as mean (SD) or median (IQR); categorical variables as n/N (%). Between-group comparisons used t-tests or Mann–Whitney U for continuous data and χ^2 or Fisher's exact tests for categorical data. For exploratory risk estimation of in-hospital mortality, we fit a parsimonious multivariable logistic model respecting events-per-parameter constraint (9). Where sparse data/separation were present, Firth bias-reduced logistic regression with profile-likelihood CIs was prespecified (11). Two-sided $\alpha=0.05$ defined statistical significance; interpretation emphasized effect sizes and 95% CIs.

Ethics
The study was approved by the PIC Institutional Review Board (PIC-IRB No. IRC/25/242) and conducted according to the Declaration of Helsinki. Verbal informed consent for use of de-identified data was obtained per institutional policy. Data were stored on secure hospital servers; direct identifiers were removed before analysis.

Results

A total of 146 consecutive AW-STEMI activations with RBBB were screened across six months; 16/146 (11.0%) were excluded (prior MI/CABG 7/146 [4.8%]; significant valvular disease/cardiomyopathy 5/146 [3.4%]; declined consent 4/146 [2.7%]), leaving 130 patients (N=130; 89.0%) for analysis. All patients underwent PPCI per protocol with complete in-hospital follow-up.

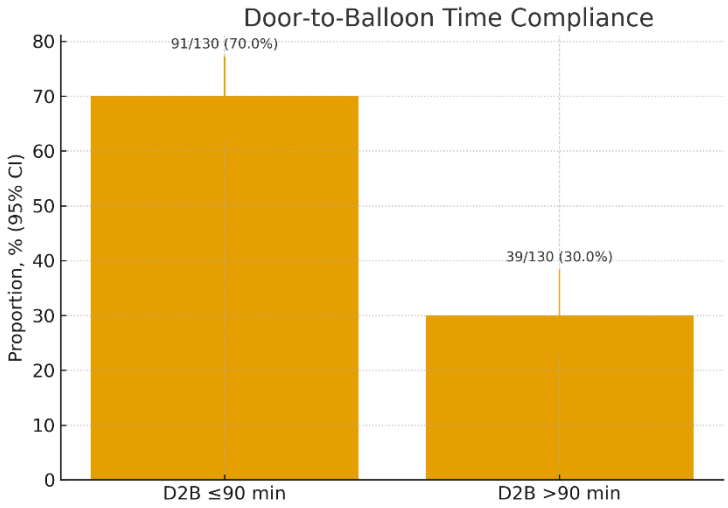
The study population (Table 1) had a mean age of 58.7 ± 11.8 years (median 59 [IQR 51–67]); male sex was 104/130 (80.0%). Hypertension was 64/130 (49.2%), diabetes 42/130 (32.3%), dyslipidemia 38/130 (29.2%), and current smoking 67/130 (51.5%). Median symptom-to-door time was 180 minutes (IQR 120–300), with >180 minutes in 66/130 (50.8%). On arrival, Killip III–IV was 15/130 (11.5%) and cardiogenic shock 10/130 (7.7%). New-onset RBBB was 93/130 (71.5%); persistent RBBB at 24 h 58/130 (44.6%).

Table 1. Baseline characteristics (N=130)

Variable	Overall
Age, years — mean ± SD	58.7 ± 11.8
Age, years — median (IQR)	59 (51–67)
Male sex — n/N (%)	104/130 (80.0%)
Hypertension — n/N (%)	64/130 (49.2%)
Diabetes mellitus — n/N (%)	42/130 (32.3%)
Dyslipidemia — n/N (%)	38/130 (29.2%)
Current smoker — n/N (%)	67/130 (51.5%)
Symptom-to-door, min — median (IQR)	180 (120–300)
Arrival >180 min — n/N (%)	66/130 (50.8%)
Killip class III–IV — n/N (%)	15/130 (11.5%)
Cardiogenic shock at presentation — n/N (%)	10/130 (7.7%)
New-onset RBBB — n/N (%)	93/130 (71.5%)
Persistent RBBB at 24 h — n/N (%)	58/130 (44.6%)

Reperfusion timeliness adhered to the protocolled D2B targets (Methods). Median door-to-balloon (D2B) time was 78 minutes (IQR 62–94); D2B >90 minutes occurred in 39/130 (30.0%). Figure 1 depicts D2B compliance with n/N (%) labels and Wilson 95% CIs.

Figure 1. Door-to-balloon time compliance (≤90 min vs >90 min).



Angiography and procedural care were consistent with guideline-directed PPCI (Methods). The infarct-related artery was LAD in 120/130 (92.3%), with proximal LAD culprit in 77/130 (59.2%). Pre-PCI TIMI 0 was 89/130 (68.5%) and post-PCI TIMI 3 117/130 (90.0%). Device/adjunct use included stent implantation in 122/130 (93.8%), thrombectomy in 19/130 (14.6%), and GP IIb/IIIa inhibitors in 28/130 (21.5%) (Table 2). Figure 2 visualizes epicardial flow (pre-PCI TIMI 0 and post-PCI TIMI 3), and Figure 3 summarizes procedural adjuncts.

Figure 2. Epicardial flow status (pre-PCI TIMI 0; post-PCI TIMI 3).

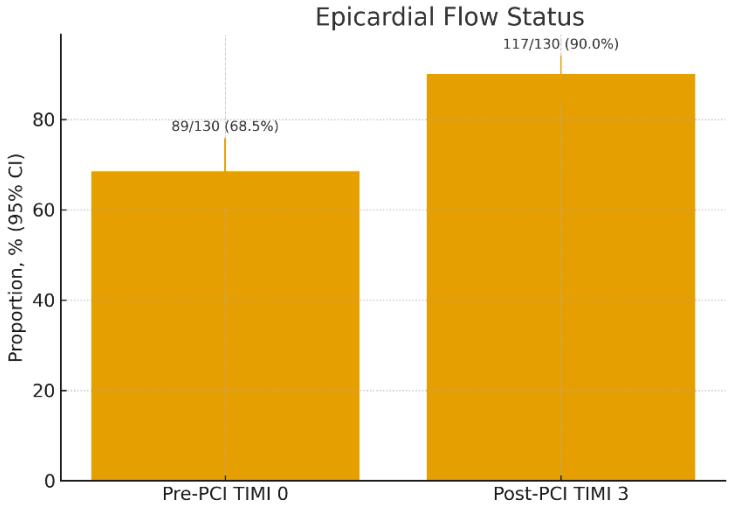


Figure 3. Procedural therapies and adjuncts (stent, thrombectomy, GP IIb/IIIa).

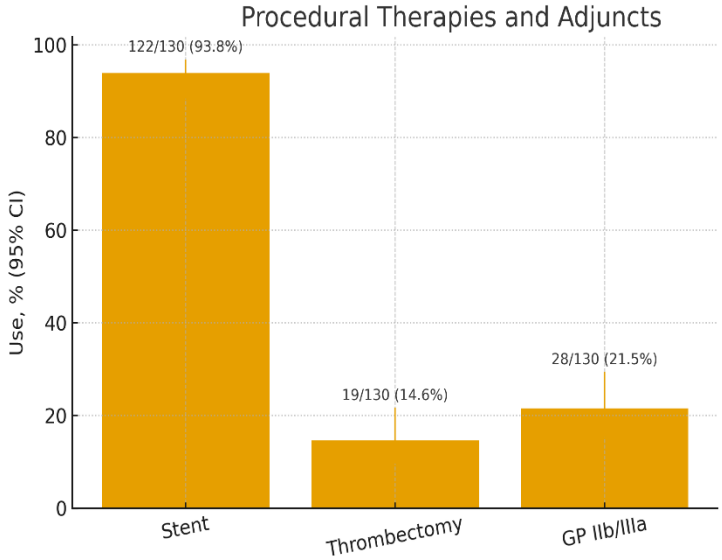


Table 2. Angiographic and procedural characteristics (N=130)

Variable	Overall
Infarct-related artery: LAD — n/N (%)	120/130 (92.3%)
Proximal LAD culprit — n/N (%)	77/130 (59.2%)
Pre-PCI TIMI 0 — n/N (%)	89/130 (68.5%)
Post-PCI TIMI 3 — n/N (%)	117/130 (90.0%)
Stent implanted — n/N (%)	122/130 (93.8%)
Thrombectomy performed — n/N (%)	19/130 (14.6%)
GP IIb/IIIa inhibitor used — n/N (%)	28/130 (21.5%)
D2B time, min — median (IQR)	78 (62–94)
D2B >90 min — n/N (%)	39/130 (30.0%)

Clinical endpoints—defined a priori and abstracted with a prespecified CRF (Methods)—are shown in Table 3 and **Figure 4. In-hospital all-cause mortality** occurred in **12/130 (9.2%; 95% CI, 4.9%–15.6%). MACE** occurred in **20/130 (15.4%; 95% CI, 9.7%–22.8%),** comprising **reinfarction 5/130 (3.8%; 95% CI, 1.3%–8.7%)** and **stroke 3/130 (2.3%; 95% CI, 0.5%–6.6%). Ventricular tachyarrhythmias (VT/VF)** were **14/130 (10.8%; 95% CI, 6.0%–17.4%); complete heart block requiring pacing 11/130 (8.5%; 95% CI, 4.3%–14.6%); acute heart failure 24/130 (18.5%; 95% CI, 12.2%–26.2%);** and **mechanical circulatory support 11/130 (8.5%; 95% CI, 4.3%–14.6%).** Median **length of stay** was **4 days (**IQR **3–6**).

Figure 4. In-hospital outcomes with 95% CIs (Mortality, MACE, VT/VF, pacing for CHB, acute HF, MCS).

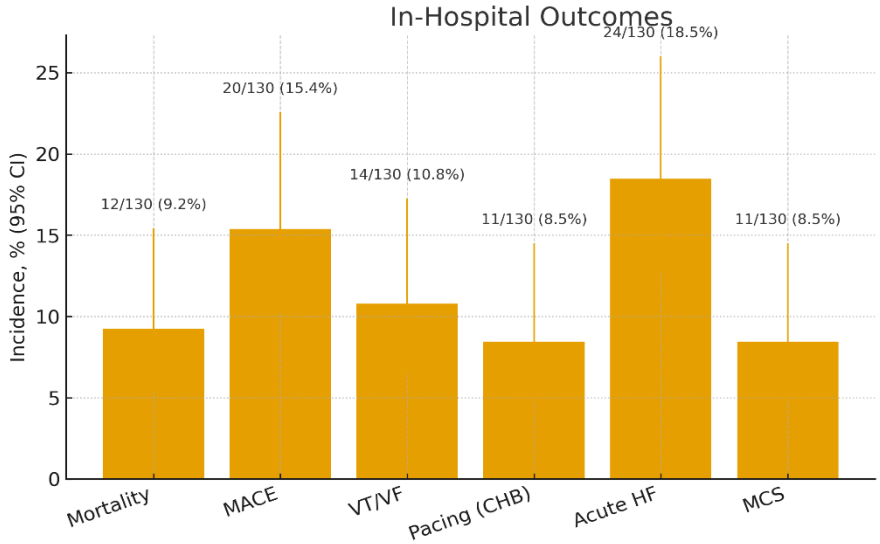


Table 3. In-hospital outcomes (N=130)

Outcome	**n/N (%)**	**95% CI**
All-cause mortality	12/130 (9.2%)	4.9%–15.6%
MACE (death/reinfarction/stroke) *	20/130 (15.4%)	9.7%–22.8%
Reinfarction	5/130 (3.8%)	1.3%–8.7%
Stroke	3/130 (2.3%)	0.5%–6.6%
VT/VF	14/130 (10.8%)	6.0%–17.4%
Complete heart block needing pacing	11/130 (8.5%)	4.3%–14.6%
Acute heart failure	24/130 (18.5%)	12.2%–26.2%
Mechanical circulatory support (IABP/ECLS)	11/130 (8.5%)	4.3%–14.6%
Length of stay, days — median (IQR)	4	3–6
*Unique patients with ≥1 component.		

Univariate comparisons (Table 4) were consistent with physiologic severity and timeliness of care. Non-survivors (n=12) were older (66.8 ± 10.5 vs 57.9 ± 11.6 years; p=0.004), arrived late more often (>180 minutes: 11/12 [91.7%] vs 55/118 [46.6%]; p=0.002), and more frequently had Killip III–IV (7/12 [58.3%] vs 8/118 [6.8%]; p<0.001). They were also more likely to present with pre-PCI TIMI 0 (11/12 [91.7%] vs 78/118 [66.1%]; p=0.047), have a proximal LAD culprit (11/12 [91.7%] vs 66/118 [55.9%]; p=0.010), and undergo D2B >90 minutes (8/12 [66.7%] vs 31/118 [26.3%]; p=0.006). VT/VF (6/12 [50.0%] vs 8/118 [6.8%]; p<0.001) and pacing for CHB (5/12 [41.7%] vs 6/118 [5.1%]; p<0.001) were over-represented among non-survivors.

Table 4. Univariate comparisons by vital status

Variable	**Survivors**	**Non-survivors**	**p-**

	(n=118)	(n=12)	value
Age, years — mean ± SD	57.9 ± 11.6	66.8 ± 10.5	.004
Male sex — n/N (%)	94/118 (79.7%)	10/12 (83.3%)	.74
Hypertension — n/N (%)	55/118 (46.6%)	9/12 (75.0%)	.048
Diabetes — n/N (%)	36/118 (30.5%)	6/12 (50.0%)	.16
Current smoker — n/N (%)	58/118 (49.2%)	9/12 (75.0%)	.09
Arrival >180 min — n/N (%)	55/118 (46.6%)	11/12 (91.7%)	.002
Killip III–IV — n/N (%)	8/118 (6.8%)	7/12 (58.3%)	<.001
Pre-PCI TIMI 0 — n/N (%)	78/118 (66.1%)	11/12 (91.7%)	.047
Proximal LAD culprit — n/N (%)	66/118 (55.9%)	11/12 (91.7%)	.010
Post-PCI TIMI 3 — n/N (%)	111/118 (94.1%)	6/12 (50.0%)	<.001
D2B >90 min — n/N (%)	31/118 (26.3%)	8/12 (66.7%)	.006
VT/VF — n/N (%)	8/118 (6.8%)	6/12 (50.0%)	<.001
CHB needing pacing — n/N (%)	6/118 (5.1%)	5/12 (41.7%)	<.001
Length of stay, days — median (IQR)	4 (3–6)	3 (2–5)	.11

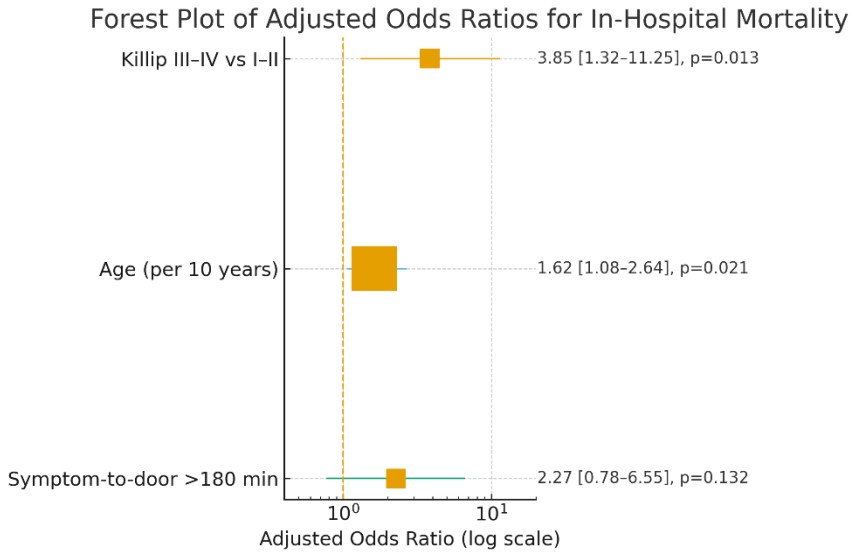
The prespecified multivariable model (Methods) respected events-per-parameter limits (12 deaths; three predictors). Killip III–IV was independently associated with in-hospital mortality (aOR 3.85; 95% CI, 1.32–11.25; p=0.013), age (per 10 years) showed a moderate association (aOR 1.62; 95% CI, 1.08–2.64; p=0.021), and symptom-to-door >180 minutes trended toward higher odds (aOR 2.27; 95% CI, 0.78–6.55; p=0.132). Calibration was acceptable (Hosmer–Lemeshow p=0.71) and discrimination fair (AUC 0.79; 95% CI, 0.68–0.90). Figure 5 presents the forest plot for adjusted effects with 95% CIs; full coefficients are given in Table 5.

Table 5. Multivariable logistic regression for in-hospital mortality (N=130; events=12)

Predictor	Adjusted OR	95% CI	p-value
Killip III–IV (vs I–II)	3.85	1.32–11.25	.013
Age (per 10 years)	1.62	1.08–2.64	.021
Symptom-to-door >180 min	2.27	0.78–6.55	.132

*All figures are single-panel matplotlib plots with Wilson 95% CIs for proportions where applicable; tables avoid multi-line cells and report every percentage as **n/N (%)**.*

Figure 5. Adjusted odds ratios for in-hospital mortality (log scale; squares sized by inverse-variance weight).



Discussion

In this consecutive cohort of patients with AW-STEMI and concomitant RBBB treated by PPCI, in-hospital mortality was 9.2% and MACE occurred in 15.4%. Events concentrated among those who were older, presented in higher Killip class, arrived later after symptom onset, and failed to achieve optimal epicardial reperfusion. Non-survivors more frequently had proximal LAD culprit lesions, pre-PCI TIMI 0 flow, D2B >90 minutes, malignant ventricular arrhythmias, and high-grade AV block requiring pacing, while achieving post-PCI TIMI 3 flow was less common in this group. The adjusted model confirmed Killip III–IV as the dominant independent predictor of death, with additional risk per decade of age and a trend toward harm with symptom-to-door >180 minutes.

System-level determinants: time still matters

The pattern of later presentation and prolonged D2B among non-survivors underscores the continuing importance of total ischemic time, even in contemporary PPCI pathways. Our findings align with system-of-care data demonstrating that delayed first medical contact and slower reperfusion are associated with excess early events despite technically successful PCI (14–16). In practical terms, prehospital ECGs, direct cath-lab activation, and streamlined inter-facility transfers remain actionable levers to shorten symptom-to-door and door-to-balloon intervals (14–16).

Hemodynamic severity and anterior territory risk

Killip class at presentation emerged as the strongest independent signal for mortality, with age adding incremental risk—an observation consistent with reperfusion-era cohorts in which shock and advanced heart-failure physiology are the principal drivers of early death, especially in large anterior infarcts (16–18). The over-representation of proximal LAD occlusion among non-survivors is directionally concordant with the greater ischemic burden and microvascular injury typical of anterior/large-territory events, which propagate pump failure, arrhythmia, and short-term mortality (16–18).

Epicardial flow and reperfusion quality

Outcomes tracked closely with both pre- and post-procedural epicardial flow. Mortality clustered among patients presenting with TIMI 0 flow and those failing to achieve final TIMI 3, reinforcing the prognostic value of spontaneous antegrade flow before intervention and full restoration of TIMI 3 afterward (19–21). These data support meticulous thrombus management and microvascular protection as procedural priorities to maximize the probability of complete reperfusion (19–21).

Electrical complications and conduction disease

The higher burden of malignant ventricular arrhythmias among non-survivors is consistent with multicenter evidence linking late VT/VF after PPCI to increased short-term mortality, suggesting arrhythmia as a marker of ongoing substrate vulnerability rather than isolated peri-procedural irritability (22). Similarly, the excess of complete AV block requiring pacing in those who died aligns with reports that high-grade block in anterior STEMI reflects extensive septal/conduction-system ischemia and confers early hazard (23). Together, these findings justify aggressive telemetry, early rhythm surveillance, and a low threshold for temporary pacing in AW-STEMI with RBBB (22,23).

Clinical implications

Three care domains appear most modifiable in similar practice environments. First, compress total ischemic time through prehospital recognition, direct activation, and optimized transfer pathways (14–16). Second, treat hemodynamic instability as a parallel priority to reperfusion—protocolized shock pathways, early vasopressor/mechanical support when indicated, and rapid escalation for refractory hypotension (16–18). Third, aim for reperfusion excellence by prioritizing techniques that increase the likelihood of final TIMI 3 flow and by instituting vigilant rhythm monitoring with pacing readiness given the observed arrhythmic and conduction complications (19–23).

Strengths and limitations

Strengths include consecutive enrollment within a standardized PPCI pathway, prespecified outcomes, and adherence to contemporary endpoint definitions. The single-center design and events-per-parameter–constrained modeling limit generalizability and multivariable depth; residual confounding is possible. Lack of systematic CMR or blush grading precludes mechanistic confirmation of microvascular injury. Findings pertain to AW-STEMI with RBBB and may not extend to other infarct territories or conduction patterns.

Future directions

Multicenter prospective work should test whether targeted reductions in total ischemic time, protocolized shock care, and reperfusion-quality optimization translate into lower early mortality in AW-STEMI with RBBB, and whether these levers also modify longer-term arrhythmic, conduction, and remodeling outcomes.

Conclusion

Among patients with AW-STEMI and RBBB undergoing PPCI, early risk concentrated in those with shock physiology, older age, and treatment delays, and was mitigated when post-PCI TIMI 3 flow was achieved. A systems approach—compressing total ischemic time, protocolized hemodynamic stabilization including pacing readiness, and meticulous reperfusion aimed at TIMI 3—may reduce near-term mortality in this high-risk phenotype. Multicenter prospective studies should test whether these levers improve longer-term conduction, arrhythmic, and remodeling outcomes.

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