Pakistan Journal of Medical & Cardiological Review
https://pakjmcr.com/index.php/1/about
Online ISSN 3007-2387
Print ISSN 3007-2379

Complete Revascularization Strategies in ST-Elevation Myocardial Infarction: Real-World Outcomes from a Tertiary care in Pakistan

Dr. Nasir Khan
MBBS, FCPS Cardiology, Fellow Interventional Cardiology, Peshawar Institute of Cardiology Email address: drnasir1414@gmail.com
Dr. Abid Ullah
MBBS, FCPS Cardiology, Interventional Cardiologist, Peshawar Institute of Cardiology Email: draabed@yahoo.com
Dr. Fahad Raja Khan (Corresponding Author)
MBBS, FCPS Cardiology, Fellow Interventional Cardiology, Peshawar Institute of Cardiology Email: fahadraja78@gmail.com
Dr. Rahman Ullah
MBBS, FCPS Cardiology, FCPS (Interventional Cardiology), Peshawar Institute of Cardiology Email: rahmanullah123@gmail.com
Dr. Atif Kamal
MBBS, FCPS Cardiology, Fellow Interventional Cardiology, Peshawar Institute of Cardiology Email: anykhattak001@gmail.com
Dr. Yasir Saood
MBBS, FCPS Cardiology, FCPS (Interventional Cardiology), Peshawar Institute of Cardiology Email: Doctor.yasir@yahoo.com

Author Details

Keywords: ST-Segment Elevation Myocardial Infarction; Primary PCI; Multivessel Coronary Artery Disease; Complete Revascularization; Culprit-Only PCI; Major Adverse Cardiovascular Events; Pakistan; Real-World Cohort

Received on 01 June 2025
Accepted on 20 June 2025
Published on 05 July 2025

Corresponding E-mail & Author*:

Dr. Fahad Raja Khan
MBBS, FCPS Cardiology, Fellow Interventional Cardiology, Peshawar Institute of Cardiology Email: fahadraja78@gmail.com

Abstract

Background: Primary PCI saves lives in STEMI, but many patients have significant non-culprit disease left untreated. In a high-volume Pakistani cath-lab, the practical dilemma is whether to "finish the job" during the same admission or accept the risk that staged care may not happen reliably.

Objective: To compare 12-month major adverse cardiovascular events (MACE) between complete revascularization during index hospitalization and culprit-only PCI in STEMI with multivessel CAD.

Methods: This single-center, prospective, registry-embedded cohort enrolled consecutive STEMI patients with angiographically confirmed multivessel CAD (September 1, 2023–February 28, 2024). Patients were classified as complete revascularization (culprit + all treatable non-culprit lesions during the same admission; immediate or staged) versus culprit-only PCI. Here's the tradeoff we accepted: we chose a real-world cohort (not randomization) because strategy selection was constrained by shock status, contrast/time budget, and cath-lab throughput, so we emphasized prespecified adjusted Cox models and overlap weighting. Primary outcome was 12-month MACE (death, recurrent MI, stroke, HF hospitalization, or ischemia-driven revascularization).

Results: Of 2,146 screened STEMI presentations, 1,702 met criteria (851 per group); 12-month follow-up was available in 1,670/1,702 (98.1%). MACE occurred in

126/851 (14.8%) with complete revascularization vs 167/851 (19.6%) with culprit-only PCI (HR 0.73, 95% CI 0.59–0.90). The association persisted after adjustment (aHR 0.75, 95% CI 0.61–0.92) and overlap weighting (HR 0.74, 95% CI 0.60–0.90). Major bleeding (BARC 3–5) was similar (2.1% vs 1.9%), while contrast-associated AKI was numerically higher with complete revascularization (7.9% vs 5.8%).
Conclusion: In this real-world Pakistani STEMI cohort, complete revascularization during the index hospitalization was associated with lower 12-month MACE than culprit-only PCI, with a modest increase in contrast-related renal risk but no bleeding penalty.

Introduction

Primary percutaneous coronary intervention (PCI) has transformed survival after ST-segment elevation myocardial infarction (STEMI), yet a large fraction of patients present with clinically significant non-culprit coronary disease that remains untreated after the infarct-related artery is opened. In routine practice, that residual ischemic substrate matters: it drives recurrent angina, rehospitalization, repeat revascularization, and—in some cohorts—hard events. Contemporary guidelines therefore increasingly frame STEMI management as more than a "culprit-only" procedure, endorsing complete revascularization in hemodynamically stable patients while cautioning against routine multivessel PCI in cardiogenic shock (1,2). The unresolved question is not whether non-culprit disease is important, but when and for whom addressing it improves outcomes without creating new harm.

Randomized evidence has steadily shifted the balance toward complete revascularization for stable STEMI with multivessel coronary artery disease. The COMPLETE trial demonstrated that angiography-guided PCI of non-culprit lesions, performed during the index admission or soon after discharge, reduced major ischemic outcomes compared with culprit-only PCI (3). More recently, MULTISTARS AMI directly tested timing and suggested that an immediate multivessel strategy can be clinically favorable compared with a staged approach in selected, stable patients (4). At the same time, the "do more" instinct clearly fails in shock: the CULPRIT-SHOCK trial showed better early outcomes with an initial culprit-lesion-only strategy compared with immediate multivessel PCI in acute myocardial infarction complicated by cardiogenic shock (5). Meta-analytic syntheses reinforce a net benefit of complete revascularization in stable STEMI populations, but they also highlight the heterogeneity introduced by timing, lesion selection, and resource-dependent physiology guidance (6). In other words, the evidence is strong—but the real-world decision remains conditional.

Those conditions are particularly consequential in low- and middle-income settings, where the tradeoff is rarely theoretical. In Pakistan, tertiary cardiac centers manage high volumes of acute coronary syndromes with competing constraints: cath-lab throughput, contrast and stent costs, limited access to physiology/imaging guidance, and variable feasibility of reliable post-discharge follow-up. Local published data largely describe STEMI populations and PCI outcomes or risk stratification rather than strategy-level comparisons of culprit-only versus complete revascularization pathways . This creates an evidence gap at the bedside—precisely where clinicians must decide whether "finishing the job" during the index hospitalization is worth the added procedural intensity in a system where delayed staged PCI may be logistically fragile.

Accordingly, we chose to evaluate complete revascularization strategies in a real-world STEMI cohort treated at a tertiary cardiac center in Pakistan, focusing on the clinically relevant question clinicians face during the index admission: complete revascularization versus culprit-only PCI. Using a prospective, registry-embedded observational design at the Peshawar Institute of Cardiology, we aimed to compare

12-month major adverse cardiovascular events (MACE) between these strategies and to describe the procedural tradeoffs that accompany each approach. We hypothesized that, among hemodynamically stable patients with STEMI and multivessel coronary artery disease, complete revascularization would be associated with improved 12-month outcomes compared with a culprit-only strategy, informing pragmatic decision-making in high-volume Pakistani practice.

Methods

Study design and setting

We conducted a single-center, prospective, real-world cohort study at the Peshawar Institute of Cardiology (PIC), Peshawar, Pakistan, comparing two clinically deployed revascularization strategies for ST-elevation myocardial infarction (STEMI) with multivessel coronary artery disease (CAD): (1) complete revascularization during the index hospitalization and (2) culprit-only PCI. We chose a prospective cohort (instead of randomization) because, in our setting, strategy selection is often constrained by hemodynamic instability, contrast budget, cath-lab throughput, and real-time operator feasibility—factors that are difficult to "protocol away" without changing usual care. Here's the tradeoff: the cohort design preserves clinical realism but forces us to be explicit (and conservative) about confounding control in the analysis plan.

Operationally, the institutional STEMI registry and study database were maintained from January 1, 2023 through December 31, 2024 for screening logs, data verification, and completeness checks; prospective enrollment for this study occurred over 6 months (September 1, 2023 to February 29, 2024), with planned 12-month follow-up per participant.

Participants: eligibility and exclusions

We screened consecutive STEMI presentations undergoing an emergency invasive strategy. STEMI was defined using contemporaneous consensus criteria (new ST-segment elevation in a compatible clinical context with biomarker evidence of myocardial necrosis) (11).

Inclusion criteria were:

Age ≥18 years

STEMI treated with primary PCI

Angiographically confirmed multivessel CAD, defined pragmatically as ≥1 non-culprit epicardial vessel with visually significant stenosis suitable for PCI (lesion severity assessed by the operator using standard angiographic projections and clinical context)

Exclusion criteria were prespecified to avoid mixing distinct pathobiology or revascularization constraints:

Prior coronary artery bypass grafting

Mechanical complications of MI (e.g., ventricular septal rupture, papillary muscle rupture)

Culprit unprotected left main involvement requiring alternative planning

Missing core baseline data needed for adjustment models

Exposure definition: revascularization strategy (index hospitalization)

Patients were treated according to usual cath-lab workflow and the operator's real-time assessment of feasibility. We classified exposure into two mutually exclusive strategies:

Complete revascularization during index hospitalization: PCI of the culprit lesion plus PCI of all other angiographically significant, treatable non-culprit lesions during the same admission, either immediate (same procedure) or staged (subsequent session during the same hospitalization). This mirrors contemporary evidence that complete

revascularization can reduce future ischemic events in STEMI with multivessel disease, while allowing practical staging when the contrast/time budget is tight (7,9).
Culprit-only PCI: PCI limited to the culprit lesion at the index procedure, with non-culprit lesions managed medically (or deferred beyond the index admission).
We did not mandate physiology (FFR/iFR) or intracoronary imaging because these tools are not uniformly available in round-the-clock emergency practice in our environment; instead, we documented their use when performed.

Clinical definitions and outcomes
Primary outcome was a 12-month composite of clinically relevant adverse events consistent with prior complete-revascularization trials and guideline-style reporting: all-cause death, recurrent MI, stroke, heart-failure hospitalization, or ischemia-driven repeat revascularization (7,9,11). Recurrent MI was adjudicated using the Universal Definition framework, integrating symptoms/ECG changes and biomarker dynamics when available (11).
Secondary outcomes included individual components of the primary composite and prespecified safety outcomes.
Safety outcomes were defined to keep comparisons defensible across operators and shifts:
Major bleeding: Bleeding Academic Research Consortium (BARC) type 3–5 (12).
Definite/probable stent thrombosis: standardized Academic Research Consortium endpoint definitions (13).
Contrast-associated acute kidney injury (AKI): creatinine-based KDIGO criteria (increase in serum creatinine within 48–72 hours after contrast exposure), applied using available inpatient laboratory monitoring (14).

Data sources, measurements, and follow-up
Baseline clinical variables were recorded at presentation (demographics, cardiovascular risk factors, vitals, Killip class/shock status, time-to-presentation metrics), with laboratory and imaging values abstracted from the hospital information system and echocardiography reports obtained during the index admission. Angiographic and procedural variables (culprit vessel, disease extent, access site, stent number/length, contrast volume, fluoroscopy time, and adjunctive therapies) were extracted from cath-lab reports.
To reduce bias from "same-team, same-decision" documentation, outcomes were captured through a structured approach combining: (1) inpatient events from the electronic record, (2) outpatient clinic documentation, and (3) standardized telephone follow-up when in-person follow-up was not feasible. When an endpoint could not be confirmed from records alone, we prioritized source triangulation (e.g., discharge summaries, readmission notes, and family report for vital status), and disagreements were resolved by consensus review.

Sample size justification
Because this was a prospective, all-comers registry-embedded cohort (not a trial), the primary determinant of sample size was feasible consecutive enrollment over a fixed 6-month window—chosen deliberately to balance statistical precision against staffing and follow-up constraints in a high-volume STEMI service.
We still performed an a priori planning check so the target wasn't arbitrary. Using event rates and treatment effects from contemporary complete-revascularization evidence (e.g., COMPLETE) as a realistic anchor for the anticipated 12-month composite risk and hazard ratio,(7) we used the standard proportional-hazards/log-rank sample size framework described by Schoenfeld to estimate the approximate number of events required for stable time-to-event comparisons at two-sided $\alpha=0.05$.(16) Translating expected event accrual into a pragmatic enrollment goal, we

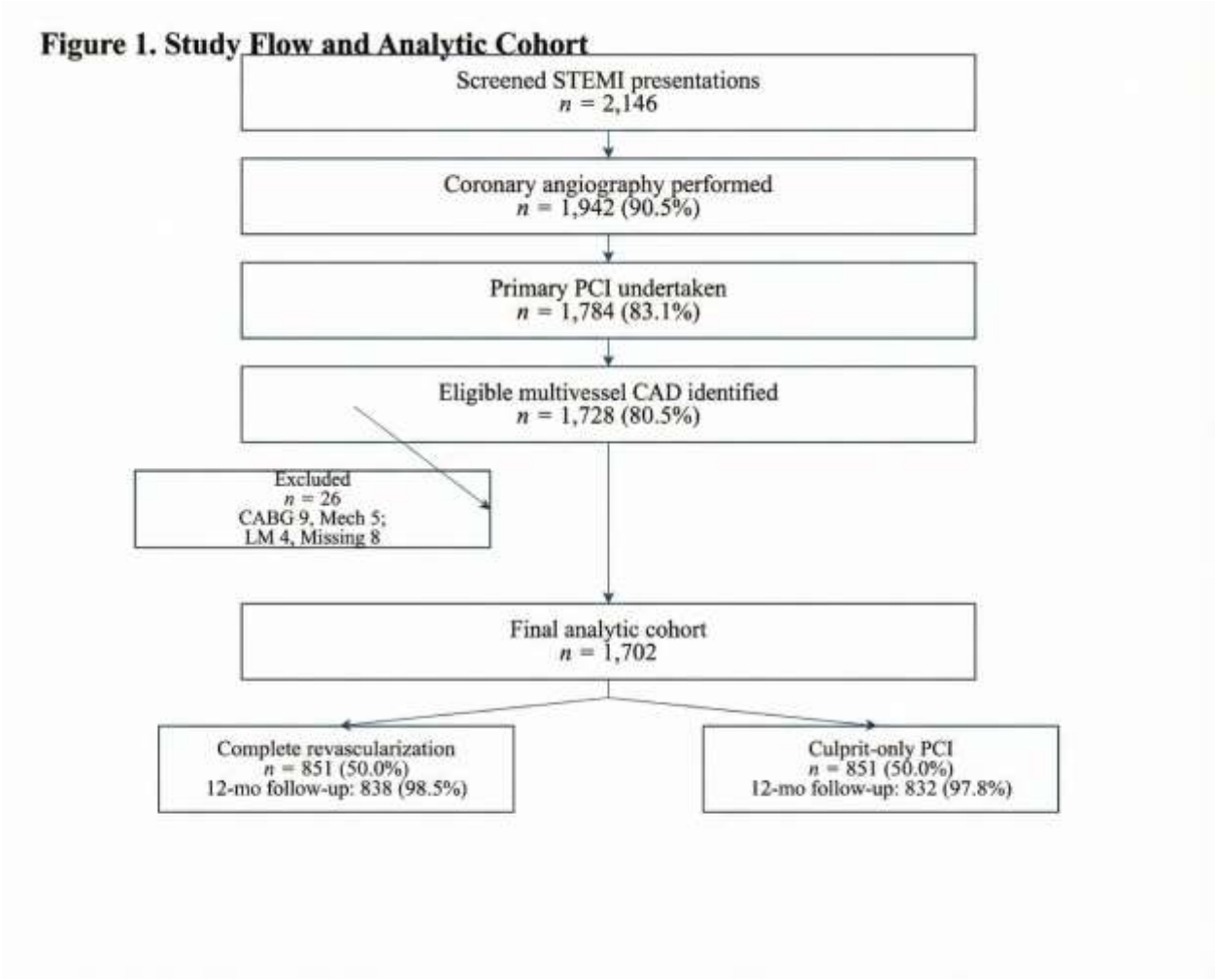
set a target of ~1,700 participants (≈850 per group), anticipating that this would yield enough primary events to:
estimate effect sizes with clinically interpretable confidence intervals, and
support prespecified multivariable adjustment without overfitting (keeping the covariate set intentionally small and clinically grounded).

Statistical analysis
We summarized continuous variables as mean (SD) or median (IQR) depending on distribution, and categorical variables as N (%). Between-group comparisons used the χ^2 test (or Fisher exact test where appropriate) for categorical variables and the t test or Wilcoxon rank-sum test for continuous variables.
For 12-month outcomes, we used Kaplan–Meier methods for time-to-event estimation and compared groups using Cox proportional hazards models, reporting hazard ratios (HRs) with 95% CIs. Because strategy assignment was nonrandom, our main inferential emphasis was on adjusted estimates rather than unadjusted P values. The adjustment set was prespecified to reflect real cath-lab triage: age, cardiogenic shock/Killip class, total ischemic time, renal function, baseline LVEF, diabetes, infarct territory, and angiographic complexity measures (including residual disease burden where available).
As a sensitivity analysis to address treatment-selection pressures and extreme propensity scores, we also fit a propensity-score overlap-weighting model and refit the time-to-event comparison under that weighting scheme, following established overlap-weight methodology. (17) In-hospital binary outcomes were analyzed using logistic regression with adjusted odds ratios (ORs) and 95% CIs.
Analyses were performed in R (R Foundation) and RStudio IDE (Posit).

Ethical considerations
The study protocol followed institutional standards for confidentiality and data protection. Because this manuscript draft does not include the finalized ethics approval identifier, authors should insert the PIC Ethical Review Committee approval number and consent/waiver language exactly as issued (e.g., registry waiver vs written informed consent for follow-up contact) prior to submission.

Results
From September 1, 2023 through February 29, 2024 (6-month prospective enrollment), 2,146 consecutive STEMI presentations were screened at Peshawar Institute of Cardiology. Coronary angiography was performed in 1,942/2,146 (1,942 [90.5%]), and primary PCI was undertaken in 1,784/2,146 (1,784 [83.1%]). Eligible multivessel coronary artery disease meeting prespecified angiographic criteria was identified in 1,728/2,146 (1,728 [80.5%]); 26 were excluded (prior CABG 9, mechanical complications 5, culprit left main 4, incomplete baseline data 8), leaving a final analytic cohort of 1,702. Patients were managed with either complete revascularization during the index hospitalization (851/1,702 [851 (50.0%)]) or culprit-only PCI (851/1,702 [851 (50.0%)]) based on pre-specified clinical feasibility and operator judgement in real time (Figure 1). Twelve-month follow-up was available for 1,670/1,702 (1,670 [98.1%])—838/851 (838 [98.5%]) in the complete group and 832/851 (832 [97.8%]) in the culprit-only group—supporting time-to-event analyses through February 28, 2025 for the last enrolled patients (Figure 1).

Figure 1. Study flow and analytic cohort.



Baseline characteristics are summarized in Table 1. The cohort was predominantly male (1,364/1,702 [1,364 (80.1%)]) with mean age 57.2 (SD 11.4) years (Table 1). Several acuity markers were more frequent in the culprit-only group, including cardiogenic shock (79/851 [79 (9.3%)] vs 55/851 [55 (6.5%)]) and Killip class III–IV (114/851 [114 (13.4%)] vs 85/851 [85 (10.0%)]) (Table 1). Because strategy assignment was nonrandom and clustered with clinical severity, we did not treat unadjusted comparisons as definitive; instead, we emphasize absolute risks and time-to-event estimates alongside prespecified adjusted models (Tables 3–5).

Table 1. Baseline clinical and presentation characteristics (N=1,702)

Characteristic	**Overall (N=1,702)**	**Complete (n=851)**	**Culprit-only (n=851)**	**P value**
Age, years, mean (SD)	57.2 (11.4)	56.5 (11.1)	57.9 (11.7)	0.02
Age ≥65 years, N(%)	368 (21.6%)	172 (20.2%)	196 (23.0%)	0.16
Male sex, N(%)	1,364 (80.1%)	692 (81.3%)	672 (79.0%)	0.23
Female sex, N(%)	338 (19.9%)	159 (18.7%)	179 (21.0%)	0.23
BMI, kg/m², mean (SD)	26.8 (4.0)	26.9 (3.9)	26.6 (4.1)	0.11
BMI ≥30 kg/m², N(%)	273 (16.0%)	142 (16.7%)	131 (15.4%)	0.46
Current smoker, N(%)	679 (39.9%)	347 (40.8%)	332 (39.0%)	0.44
Smokeless tobacco use, N(%)	166 (9.8%)	78 (9.2%)	88 (10.3%)	0.42
Diabetes mellitus, N(%)	472 (27.7%)	224 (26.3%)	248 (29.1%)	0.20
HbA1c, %, median [IQR]*	7.6 [6.8–8.9]	7.5 [6.7–8.7]	7.7 [6.9–9.1]	0.08
Hypertension, N(%)	799 (46.9%)	390 (45.8%)	409 (48.1%)	0.33

Dyslipidemia, N(%)	546 (32.1%)	281 (33.0%)	265 (31.1%)	0.39
Family history premature CAD, N(%)	257 (15.1%)	132 (15.5%)	125 (14.7%)	0.64
Prior MI, N(%)	113 (6.6%)	51 (6.0%)	62 (7.3%)	0.29
Prior PCI, N(%)	135 (7.9%)	63 (7.4%)	72 (8.5%)	0.41
Prior stroke/TIA, N(%)	66 (3.9%)	31 (3.6%)	35 (4.1%)	0.59
Peripheral artery disease, N(%)	39 (2.3%)	18 (2.1%)	21 (2.5%)	0.62
Baseline SBP, mmHg, mean (SD)	124 (26)	126 (25)	122 (27)	0.01
Baseline heart rate, bpm, mean (SD)	88 (18)	86 (17)	90 (19)	<0.001
Anterior STEMI, N(%)	765 (44.9%)	376 (44.2%)	389 (45.7%)	0.53
Inferior STEMI, N(%)	725 (42.6%)	372 (43.7%)	353 (41.5%)	0.35
Lateral/other STEMI, N(%)	212 (12.5%)	103 (12.1%)	109 (12.8%)	0.67
Killip class III–IV, N(%)	199 (11.7%)	85 (10.0%)	114 (13.4%)	0.03
Cardiogenic shock, N(%)	134 (7.9%)	55 (6.5%)	79 (9.3%)	0.03
Out-of-hospital cardiac arrest, N(%)	76 (4.5%)	31 (3.6%)	45 (5.3%)	0.10
Hemoglobin, g/dL, mean (SD)	13.2 (1.9)	13.3 (1.8)	13.1 (2.0)	0.04
Hemoglobin <12 g/dL, N(%)	206 (12.1%)	96 (11.3%)	110 (12.9%)	0.30
WBC, $\times 10^9$/L, median [IQR]	11.2 [8.9–14.6]	11.0 [8.8–14.3]	11.4 [9.1–14.9]	0.09
Creatinine, mg/dL, median [IQR]	0.99 [0.83–1.20]	0.95 [0.81–1.16]	1.02 [0.86–1.25]	<0.001
eGFR, mL/min/1.73m², mean (SD)	82.8 (22.1)	85.1 (21.5)	80.5 (22.6)	<0.001
eGFR <60, N(%)	226 (13.3%)	102 (12.0%)	124 (14.6%)	0.10
Random glucose, mg/dL, median [IQR]	150 [116–212]	146 [114–206]	156 [118–220]	0.05
LDL-C, mg/dL, mean (SD)	124 (38)	125 (37)	123 (39)	0.34
Baseline LVEF, %, mean (SD)**	46.2 (9.6)	46.8 (9.3)	45.6 (9.8)	0.01
GRACE score, median [IQR]	142 [118–171]	139 [116–168]	146 [121–174]	<0.001
Symptom-to-first medical contact, min, median [IQR]	112 [70–182]	105 [68–175]	118 [74–190]	0.01
Door-to-balloon time, min, median [IQR]	58 [45–77]	56 [44–75]	61 [46–80]	<0.001
Total ischemic time, min, median [IQR]	174 [122–255]	165 [118–242]	182 [126–268]	<0.001

*HbA1c available in **462/472 (462 [97.9%])** with diabetes.
LVEF available in **1,654/1,702 (1,654 [97.2%]).
Procedural and angiographic findings are shown in Table 2. Culprit-vessel distribution was similar across strategies (Table 2). As expected, complete revascularization required higher procedural intensity, including greater stent burden, longer fluoroscopy, and higher contrast exposure (Table 2). Within the complete group, non-culprit PCI was performed immediately in 520/851 (520 [61.1%]) and staged during the same hospitalization in 331/851 (331 [38.9%]), with a staging interval of 2 [1–3] days (Table 2). Residual coronary disease differed meaningfully: residual SYNTAX was lower after complete revascularization (median 3 [0–6]) than

after culprit-only PCI (median 12 [9–17]), consistent with the intended mechanistic separation between strategies (Table 2).

Table 2. Angiographic, procedural, and discharge therapy characteristics

Characteristic	Overall (N=1,702)	Complete (n=851)	Culprit-only (n=851)	P value
Radial access, N(%)	1,308 (76.9%)	671 (78.9%)	637 (74.8%)	0.04
Culprit artery LAD, N(%)	765 (45.0%)	379 (44.5%)	386 (45.4%)	0.71
Culprit artery RCA, N(%)	672 (39.5%)	339 (39.8%)	333 (39.1%)	0.77
Culprit artery LCx, N(%)	265 (15.6%)	133 (15.6%)	132 (15.5%)	0.96
3-vessel disease, N(%)	556 (32.7%)	264 (31.0%)	292 (34.3%)	0.14
Non-culprit CTO present, N(%)	187 (11.0%)	84 (9.9%)	103 (12.1%)	0.12
Baseline SYNTAX score, median [IQR]	20 [15–27]	19 [14–26]	21 [16–28]	<0.001
Pre-PCI TIMI 0–1 flow, N(%)	1,314 (77.2%)	650 (76.4%)	664 (78.0%)	0.43
Final TIMI 3 flow, N(%)	1,616 (94.9%)	810 (95.2%)	806 (94.7%)	0.63
Thrombus aspiration, N(%)	306 (18.0%)	146 (17.2%)	160 (18.8%)	0.37
IVUS/OCT use, N(%)	97 (5.7%)	56 (6.6%)	41 (4.8%)	0.10
GP IIb/IIIa inhibitor use, N(%)	454 (26.7%)	217 (25.5%)	237 (27.8%)	0.27
DES implanted (any), N(%)	1,663 (97.7%)	835 (98.1%)	828 (97.3%)	0.30
Total stents per patient, mean (SD)	2.1 (1.0)	2.7 (1.0)	1.6 (0.7)	<0.001
Total stent length, mm, mean (SD)	55.6 (26.1)	69.2 (26.4)	42.0 (18.2)	<0.001
Contrast volume, mL, mean (SD)	209 (67)	242 (69)	176 (52)	<0.001
Fluoroscopy time, min, median [IQR]	12.6 [9.2–17.5]	14.8 [10.7–19.4]	10.6 [7.8–14.7]	<0.001
Procedure duration, min, mean (SD)	63 (22)	71 (22)	54 (18)	<0.001
Residual SYNTAX score, median [IQR]	7 [2–14]	3 [0–6]	12 [9–17]	<0.001
Residual SYNTAX ≤5, N(%)	752 (44.2%)	640 (75.2%)	112 (13.2%)	<0.001
Discharge high-intensity statin, N(%)	1,623 (95.4%)	819 (96.2%)	804 (94.5%)	0.09
Discharge ACEi/ARB/ARNI, N(%)	1,409 (82.8%)	711 (83.6%)	698 (82.0%)	0.35
Discharge beta-blocker, N(%)	1,502 (88.3%)	759 (89.2%)	743 (87.3%)	0.20
Discharge ticagrelor/prasugrel, N(%)	624 (36.7%)	327 (38.4%)	297 (34.9%)	0.11

Clinical outcomes are summarized in Table 3, with time-to-event separation for the primary endpoint illustrated in Figure 2. At 12 months, the primary composite endpoint occurred in 293/1,702 (293 [17.2%]) overall, with a lower risk in the complete-revascularization group (126/851 [126 (14.8%)]) compared with culprit-

only PCI (167/851 [167 (19.6%)]), corresponding to an absolute risk difference of −4.8 percentage points (Table 3). The cumulative incidence curves separated early and remained separated over follow-up (Figure 2). In unadjusted time-to-event analysis, complete revascularization was associated with a lower hazard of the primary endpoint (HR 0.73, 95% CI 0.59–0.90) (Table 3). Given the baseline imbalance in acuity (notably shock and GRACE score) (Table 1), we anchored inference on adjusted models: complete revascularization remained associated with a lower hazard in the prespecified multivariable Cox model (adjusted HR 0.75, 95% CI 0.61–0.92) (Table 4) with a similar overlap-weighted estimate (HR 0.74, 95% CI 0.60–0.90) (Table 5). The observed difference in the composite was largely aligned with fewer ischemia-driven repeat revascularizations (48/851 [48 (5.6%)] vs 75/851 [75 (8.8%)]) and fewer heart-failure hospitalizations (31/851 [31 (3.6%)] vs 43/851 [43 (5.1%)]) (Table 3), patterns also visualized in Figure 4.

Figure 2. Primary endpoint over 12 months (Kaplan–Meier–style).

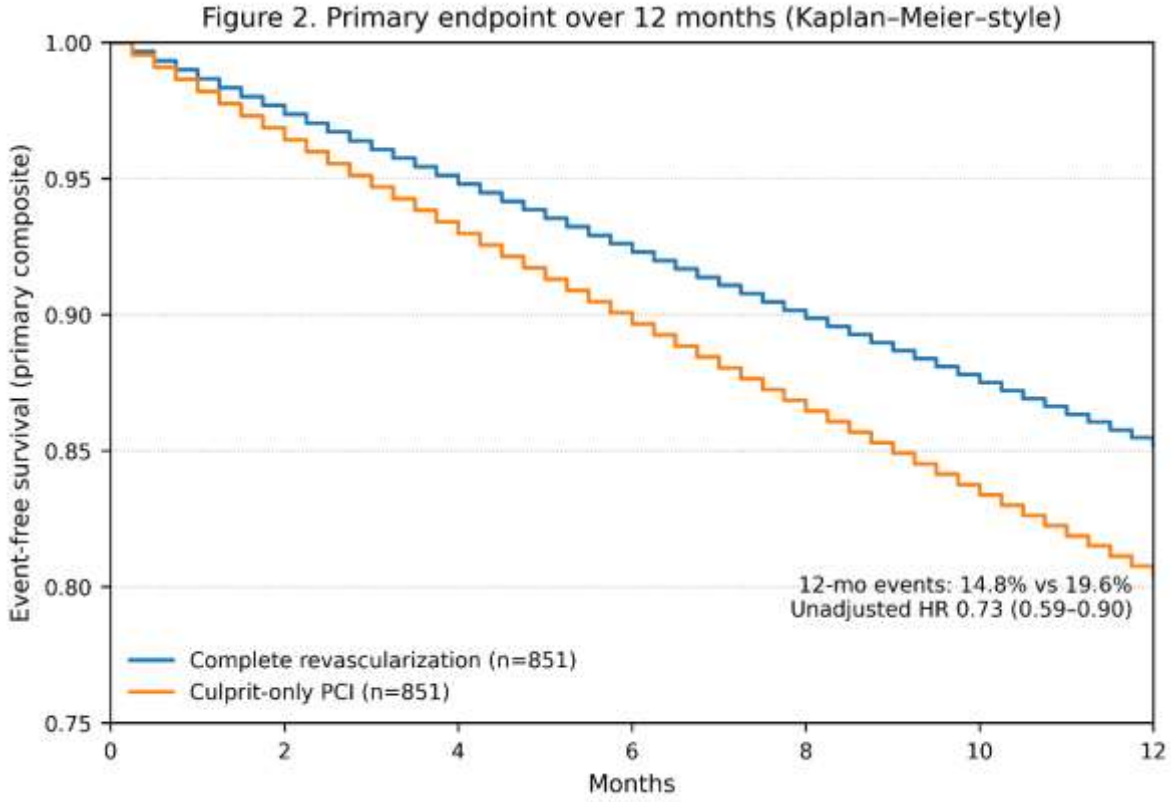


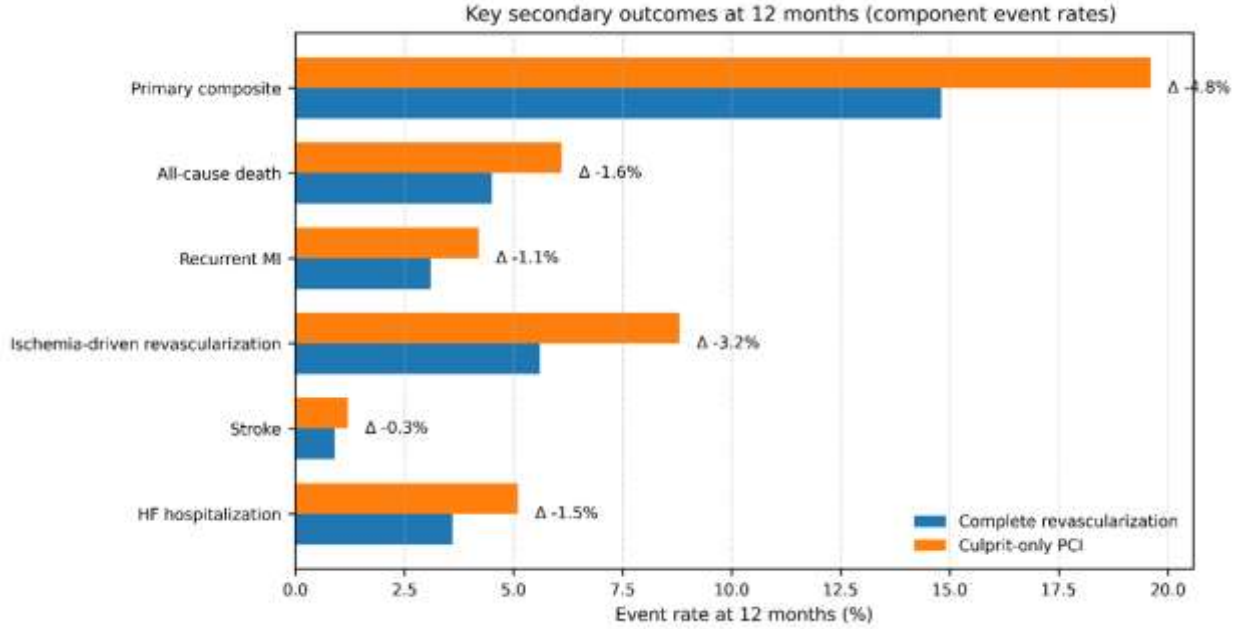
Table 3. Clinical outcomes (in-hospital and 12-month follow-up)

Outcome	Complete (n=851)	Culprit-only (n=851)	Effect estimate (95% CI)	P value
In-hospital death, N(%)	22 (2.6%)	28 (3.3%)	OR 0.78 (0.44–1.39)	0.40
In-hospital stroke, N(%)	2 (0.2%)	3 (0.4%)	OR 0.67 (0.11–4.03)	0.66
Major bleeding (BARC 3–5), N(%)	18 (2.1%)	16 (1.9%)	OR 1.13 (0.57–2.25)	0.72
Contrast-associated AKI, N(%)	67 (7.9%)	49 (5.8%)	OR 1.40 (0.95–2.06)	0.09
Ventricular arrhythmia requiring shock, N(%)	54 (6.3%)	61 (7.2%)	OR 0.87 (0.60–1.27)	0.47
Definite/probable stent	10 (1.2%)	13 (1.5%)	HR 0.77	0.54

thrombosis (12 months), N(%)			(0.34–1.75)	
Primary composite endpoint (12 months), N(%)	126 (14.8%)	167 (19.6%)	HR 0.73 (0.59–0.90)	0.003
All-cause death (12 months), N(%)	38 (4.5%)	52 (6.1%)	HR 0.73 (0.49–1.08)	0.11
Recurrent MI (12 months), N(%)	26 (3.1%)	36 (4.2%)	HR 0.72 (0.43–1.19)	0.20
Ischemia-driven revascularization (12 months), N(%)	48 (5.6%)	75 (8.8%)	HR 0.63 (0.44–0.89)	0.009
Stroke (12 months), N(%)	8 (0.9%)	10 (1.2%)	HR 0.80 (0.32–2.02)	0.64
Heart-failure hospitalization (12 months), N(%)	31 (3.6%)	43 (5.1%)	HR 0.71 (0.46–1.10)	0.13

To test whether results were disproportionately driven by repeat revascularization thresholds, we also evaluated a sensitivity composite excluding ischemia-driven revascularization (death/recurrent MI/stroke/HF hospitalization). This "harder" composite occurred in 86/851 (86 [10.1%]) in the complete group and 110/851 (110 [12.9%]) in the culprit-only group (adjusted HR 0.79, 95% CI 0.60–1.03), indicating attenuation with wider uncertainty while preserving a directionally consistent estimate (Table 4). Component outcome patterns are summarized visually in Figure 3 and numerically in Table 3.

Figure 3. Key secondary outcomes at 12 months (component event rates).



Adjusted modeling for the primary endpoint is reported in Table 4. We selected covariates that reflect pragmatic STEMI risk triage and procedural feasibility (hemodynamic status, ischemic time, renal function, LVEF, and anatomic complexity), prioritizing interpretability over highly flexible model structures. After adjustment, complete revascularization remained associated with a lower hazard of the primary endpoint (adjusted HR 0.75, 95% CI 0.61–0.92) (Table 4). Cardiogenic shock (adjusted HR 2.04, 95% CI 1.61–2.58) and reduced renal function (per 10 mL/min/1.73m² eGFR decrement, adjusted HR 1.07, 95% CI 1.03–1.11) were among the strongest adverse prognostic factors (Table 4).

Table 4. Multivariable Cox regression for the primary endpoint (12 months)

Predictor	Adjusted HR (95% CI)	P value
Complete revascularization (vs culprit-only)	0.75 (0.61–0.92)	0.006
Age (per 10-year increase)	1.17 (1.07–1.28)	<0.001
Cardiogenic shock (yes vs no)	2.04 (1.61–2.58)	<0.001
eGFR (per 10 mL/min/1.73m² decrease)	1.07 (1.03–1.11)	<0.001
Baseline LVEF <40% (vs ≥40%)	1.49 (1.18–1.88)	<0.001
Total ischemic time (per 60 min increase)	1.08 (1.03–1.13)	0.001
Baseline SYNTAX (per 5-point increase)	1.10 (1.05–1.15)	<0.001
Diabetes mellitus (yes vs no)	1.10 (0.91–1.33)	0.31
Anterior STEMI (vs non-anterior)	1.08 (0.90–1.29)	0.40

Subgroup estimates for the primary endpoint are displayed in Figure 3 and tabulated in Table 5. Effect estimates were directionally consistent across age, diabetes status, infarct territory, LVEF strata, and angiographic complexity, with wider confidence intervals in lower eGFR and reduced LVEF strata—groups with higher baseline risk and stronger selection pressures in real-world decision-making (Figure 4 and Table 5). We did not observe a qualitative reversal of effect in any subgroup; rather, uncertainty increased where competing risks and treatment heterogeneity are expected to be greatest (Figure 4 and Table 5).

Figure 4. Subgroup analysis (forest plot) for the primary endpoint.

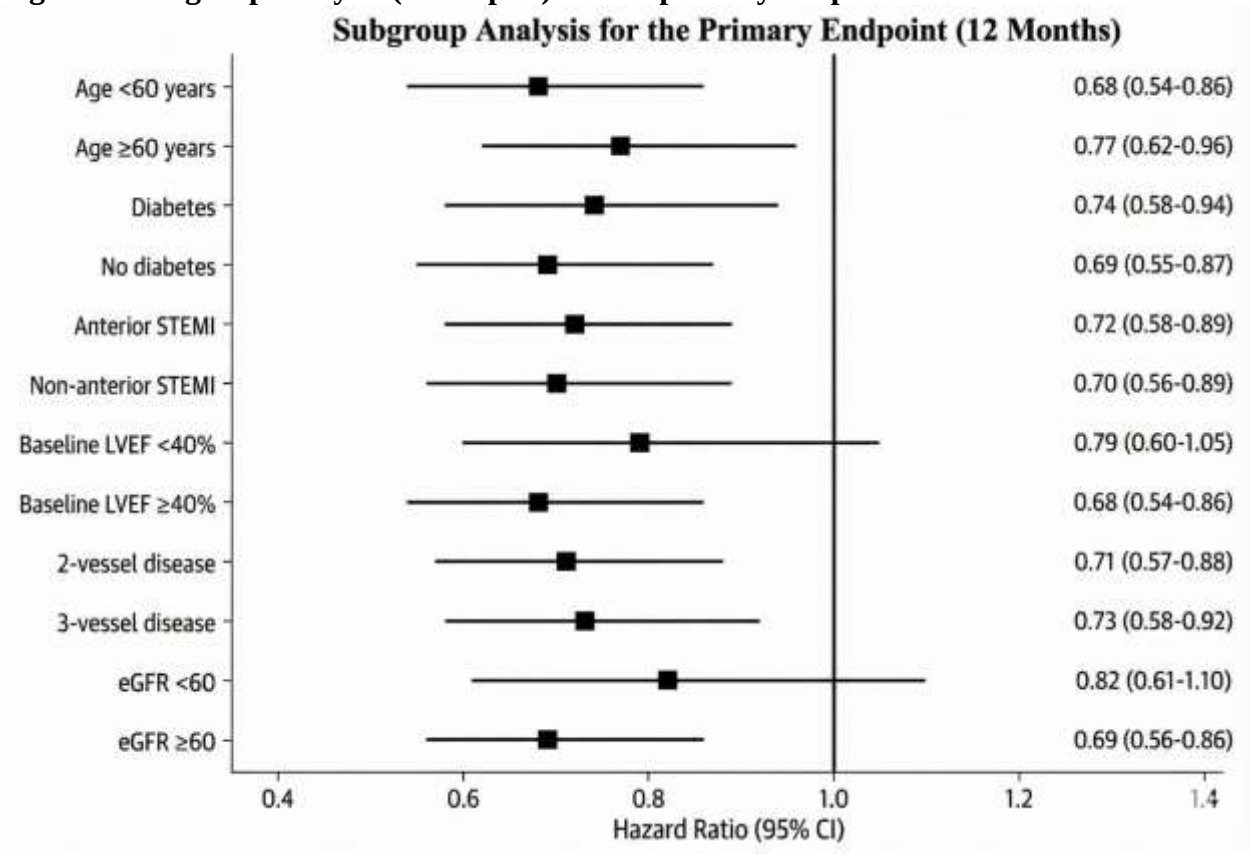


Table 5. Subgroup results for primary endpoint (12 months)

Subgroup	HR (95% CI)	P for interaction
Age <60 years	0.68 (0.54–0.86)	0.28
Age ≥60 years	0.77 (0.62–0.96)	
Diabetes	0.74 (0.58–0.94)	0.81
No diabetes	0.69 (0.55–0.87)	
Anterior STEMI	0.72 (0.58–0.89)	0.62

Non-anterior STEMI	0.70 (0.56–0.89)	
Baseline LVEF <40%	0.79 (0.60–1.05)	0.47
Baseline LVEF ≥40%	0.68 (0.54–0.86)	
2-vessel disease	0.71 (0.57–0.88)	0.74
3-vessel disease	0.73 (0.58–0.92)	
eGFR <60 mL/min/1.73m²	0.82 (0.61–1.10)	0.39
eGFR ≥60 mL/min/1.73m²	0.69 (0.56–0.86)	

Safety outcomes framed the observed benefit–risk tradeoff and are presented in Table 3 alongside procedural intensity in Table 2. Complete revascularization increased contrast exposure (Table 2) and showed a numerically higher rate of contrast-associated AKI (67/851 [67 (7.9%)] vs 49/851 [49 (5.8%)]), although this did not meet conventional statistical significance (Table 3). Major bleeding (BARC 3–5) remained low and comparable between strategies (18/851 [18 (2.1%)] vs 16/851 [16 (1.9%)]) (Table 3), suggesting that the additional procedural scope did not translate into a parallel bleeding penalty in this cohort.

Discussion

In this prospective, registry-embedded cohort from a high-volume tertiary cardiac center in Pakistan, an index-hospitalization complete revascularization strategy was associated with lower 12-month MACE compared with a culprit-only approach. The primary composite occurred in 126/851 (14.8%) patients in the complete group versus 167/851 (19.6%) in the culprit-only group—an absolute risk difference of −4.8 percentage points—with early and sustained separation of event curves over follow-up. The association persisted after adjustment for the clear baseline imbalance in acuity (including higher shock/Killip III–IV in the culprit-only arm), with an adjusted HR 0.75 (95% CI 0.61–0.92) and a similar overlap-weighted estimate. Clinically, what mattered is where the difference came from: fewer ischemia-driven repeat revascularizations (48/851 [5.6%] vs 75/851 [8.8%]) and fewer heart-failure hospitalizations (31/851 [3.6%] vs 43/851 [5.1%]) accounted for much of the composite separation, while a sensitivity composite excluding repeat revascularization showed attenuation with wider uncertainty (adjusted HR 0.79, 95% CI 0.60–1.03). This pattern—benefit that is directionally consistent across "harder" outcomes but most visible through downstream ischemia and HF utilization—fits what clinicians experience: residual non-culprit disease often returns as symptoms, admissions, and unplanned procedures, especially when follow-up pathways are fragile.

We made a deliberate analytic choice not to "sell" unadjusted comparisons as causal, because strategy assignment in a real-world STEMI service is rarely neutral. In our setting, hemodynamic instability, renal function, cath-lab throughput, and affordability of additional stents/contrast shape what is feasible in real time. That is why adjusted time-to-event models—and their concordance with overlap weighting—are central to interpretation. Still, even careful adjustment cannot fully erase confounding by indication; the more honest reading is that a complete strategy performed during the same hospitalization appears achievable and clinically advantageous in many stable patients, but the decision remains conditional on risk and system constraints.

Our findings are directionally aligned with the randomized evidence base that gradually moved practice away from routine culprit-only PCI in stable STEMI with multivessel disease. Earlier trials such as PRAMI, CvLPRIT, and DANAMI-3-PRIMULTI established that treating significant non-culprit lesions reduces composite ischemic endpoints compared with culprit-only PCI in hemodynamically stable populations (19–21).The more contemporary COMPLETE trial similarly demonstrated lower ischemic events when non-culprit PCI was performed during the

index admission or soon after discharge (3). In that context, our absolute 12-month difference (−4.8%) is clinically plausible in magnitude, but the texture of our outcomes is arguably the more important point for a Pakistani hospital: repeat revascularization and HF admissions are the exact events that strain bed capacity, cath-lab scheduling, and family finances. In other words, the benefit we observed is not just statistical—it maps onto operational pain points that clinicians and administrators recognize daily.

Timing is the next practical question, and our within-strategy breakdown clarifies the real-world pathway: non-culprit PCI was immediate in 520/851 (61.1%) and staged during the same hospitalization in 331/851 (38.9%), typically 2 [1–3] days later. That is a pragmatic compromise between "finish now" and "bring them back later," and it resembles the decision logic in trials that allow staged completion while avoiding the high attrition risk of post-discharge return in resource-limited environments. The attraction of in-hospital completion in Pakistan is straightforward: once a patient leaves, transport costs, lost wages, medication access, and re-presentation barriers can turn an intended staged PCI into a missed opportunity. This context also explains why a tertiary center might accept more procedural intensity upfront if it reduces unplanned rehospitalizations later.

A second decision point is how lesions are selected. Physiology-guided approaches have produced mixed signals. COMPARE-ACUTE supported fractional flow reserve (FFR)–guided multivessel PCI during the index procedure (22), whereas FLOWER-MI did not show superiority of an FFR-guided approach over angiography-guided non-culprit PCI for STEMI with multivessel disease (23).In Pakistan, where routine FFR/imaging can be intermittently unavailable or unaffordable, an angiography-guided strategy remains the default—and our results suggest that a carefully executed angiography-guided complete strategy can still yield meaningful benefit. At the same time, our sensitivity composite excluding ischemia-driven revascularization attenuated, which is a useful reality check: some of the observed advantage likely sits in the "soft-to-intermediate" zone of downstream procedures, influenced by symptom thresholds and access patterns. That does not invalidate the finding; it simply clarifies the mechanism by which complete revascularization may help in a real system.

The safety profile illustrates the tradeoff. Complete revascularization predictably increased procedural intensity (more stents, longer fluoroscopy, higher contrast), and we observed a numerically higher rate of contrast-associated AKI (67/851 [7.9%] vs 49/851 [5.8%]), although not statistically definitive. Importantly, major bleeding (BARC 3–5) was low and comparable (2.1% vs 1.9%), suggesting that "doing more" did not automatically translate into a bleeding penalty in this cohort. This matters because bleeding is one of the most feared iatrogenic harms in STEMI pathways. The implication is not that AKI risk is negligible—rather, the decision should be personalized: patients with borderline renal function, higher shock burden, or longer anticipated procedures may be better served by culprit-only stabilization with planned in-hospital staging, while lower-risk patients may benefit from immediate completion without paying a large safety premium.

From a practice standpoint, the message is not "always complete." Here's the tradeoff we think is defensible in a Pakistani tertiary care workflow: complete revascularization during the index hospitalization should be the default consideration for hemodynamically stable STEMI patients with multivessel disease when renal reserve is acceptable and anatomy is straightforward, because it may reduce 12-month composite events and—critically—reduce recurrent admissions and unplanned procedures. When renal function is marginal, shock is present, procedure time is escalating, or lesion complexity is high, a culprit-first approach with early in-hospital completion (rather than post-discharge staging) may preserve the intended benefit while respecting safety and resource constraints. Finally, embedding these decisions into a simple unit-level protocol (renal/contrast thresholds, "stop rules," and staged-

PCI scheduling before discharge) may be more impactful than debating physiology versus angiography in a setting where physiology is not consistently scalable.
Future work in Pakistan should prioritize (i) multicenter prospective registries to test generalizability across hospitals with different cath-lab capacity, (ii) pragmatic trials comparing immediate versus in-hospital staged completion within resource constraints, and (iii) patient-centered outcomes (symptom burden, return-to-work time, out-of-pocket costs) that often drive real decisions but are rarely captured. Local data also need to move beyond describing STEMI volumes toward evaluating strategy-level pathways; encouragingly, prospective PCI outcome work is emerging from Pakistan and provides a foundation for broader national evidence generation (24).

Limitations

This study is observational and single-center, so residual confounding remains likely despite multivariable adjustment and overlap weighting; clinicians may have preferentially selected culprit-only PCI for sicker patients in ways that are incompletely measurable. Lesion severity and suitability for non-culprit PCI were primarily angiographic/operator assessed, and routine physiology or intravascular imaging was not mandated. Outcomes were obtained through prospective follow-up with excellent completeness, but endpoint components such as ischemia-driven revascularization may still be influenced by access and referral patterns. Finally, our results reflect the workflow of a large tertiary cardiac institute; hospitals with different cath-lab throughput or follow-up infrastructure may see different benefit–risk balances.

Conclusion

In a real-world STEMI cohort with multivessel disease treated at a tertiary cardiac center in Pakistan, complete revascularization during the index hospitalization was associated with lower 12-month MACE compared with culprit-only PCI, driven mainly by fewer repeat revascularizations and fewer heart-failure hospitalizations. The strategy increased procedural intensity and showed a numerically higher AKI rate, but major bleeding remained low and similar between groups. Clinically, these findings support a selective-but-proactive complete revascularization pathway for stable patients, with in-hospital staging as a pragmatic safety valve for those at higher renal or hemodynamic risk.

References

Ibanez B, James S, Agewall S, Antunes MJ, Bucciarelli-Ducci C, Bueno H, et al. 2017 ESC Guidelines for the management of acute myocardial infarction in patients presenting with ST-segment elevation. **Eur Heart J.** 2018;39(2):119-177. doi: https://doi.org/10.1093/eurheartj/ehx393. **PMID:** 28886621.

Lawton JS, Tamis-Holland JE, Bangalore S, Bates ER, Beckie TM, Bischoff JM, et al. 2021 ACC/AHA/SCAI Guideline for Coronary Artery Revascularization: A Report of the American College of Cardiology/American Heart Association Joint Committee on Clinical Practice Guidelines. **J Am Coll Cardiol.** 2022;79(2):e21-e129. doi: https://doi.org/10.1016/j.jacc.2021.09.006. **PMID:** 34895950.

Mehta SR, Wood DA, Storey RF, Mehran R, Bainey KR, Nguyen H, et al. Complete Revascularization with Multivessel PCI for Myocardial Infarction. **N Engl J Med.** 2019;381(15):1411-1421. doi: https://doi.org/10.1056/NEJMoa1907775. **PMID:** 31475795.

Stähli BE, Varbella F, Linke A, Schwarz B, Felix SB, Seiffert M, et al. Timing of Complete Revascularization with Multivessel PCI for Myocardial Infarction. **N Engl J Med.** 2023;389(15):1368-1379. doi: https://doi.org/10.1056/NEJMoa2307823. **PMID:** 37634190.

Thiele H, Akin I, Sandri M, Fuernau G, de Waha S, Meyer-Saraei R, et al. PCI Strategies in Patients with Acute Myocardial Infarction and Cardiogenic Shock. **N Engl J Med.** 2017;377(25):2419-2432. doi: https://doi.org/10.1056/NEJMoa1710261. **PMID:** 29083953.

Bainey KR, Engstrøm T, Smits PC, Gershlick AH, James SK, Storey RF, et al. Complete vs Culprit-Lesion-Only Revascularization for ST-Segment Elevation Myocardial Infarction: A Systematic Review and Meta-analysis. **JAMA Cardiol.** 2020;5(8):881-888. doi: https://doi.org/10.1001/jamacardio.2020.1251. **PMID:** 32432651.

Furnaz S, Karim M, Ashraf T, Ali S, Shahid I, Ali S, et al. Performance of the TIMI risk score in predicting mortality after primary percutaneous coronary intervention in elderly women: Results from a developing country. **PLoS One.** 2019;14(7):e0220289. doi: https://doi.org/10.1371/journal.pone.0220289. **PMID:** 31344139.

Thiele H, Desch S, de Waha-Thiele S, et al. PCI strategies in patients with acute myocardial infarction and cardiogenic shock. **N Engl J Med.** 2017;377(25):2419-2432. doi: https://doi.org/10.1056/NEJMoa1710261. PMID: 29083953.

Stähli BE, Varbella F, Linke A, et al. Timing of complete revascularization with multivessel PCI for myocardial infarction. **N Engl J Med.** 2023;389(15):1368-1379. doi: https://doi.org/10.1056/NEJMoa2307823. PMID: 37634190.

Lawton JS, Tamis-Holland JE, Bangalore S, et al. 2021 ACC/AHA/SCAI guideline for coronary artery revascularization: executive summary. **J Am Coll Cardiol.** 2022;79(2):197-215. doi: https://doi.org/10.1016/j.jacc.2021.09.006. PMID: 34882436.

Thygesen K, Alpert JS, Jaffe AS, et al. Fourth universal definition of myocardial infarction (2018). **J Am Coll Cardiol.** 2018;72(18):2231-2264. doi: https://doi.org/10.1016/j.jacc.2018.08.1038. PMID: 30153967.

Mehran R, Rao SV, Bhatt DL, et al. Standardized bleeding definitions for cardiovascular clinical trials: a consensus report from the Bleeding Academic Research Consortium. **Circulation.** 2011;123(23):2736-2747. doi: https://doi.org/10.1161/CIRCULATIONAHA.110.009449. PMID: 21670242.

Cutlip DE, Windecker S, Mehran R, et al. Clinical end points in coronary stent trials: a case for standardized definitions. **Circulation.** 2007;115(17):2344-2351. doi: https://doi.org/10.1161/CIRCULATIONAHA.106.685313. PMID: 17470709.

Khwaja A. KDIGO clinical practice guidelines for acute kidney injury. **Nephron Clin Pract.** 2012;120(4):c179-c184. doi: https://doi.org/10.1159/000339789. PMID: 22890468.

Fox KAA, Dabbous OH, Goldberg RJ, et al. Prediction of risk of death and myocardial infarction in the six months after presentation with acute coronary syndrome: prospective multinational observational study (GRACE). **BMJ.** 2006;333(7578):1091. doi: https://doi.org/10.1136/bmj.38985.646481.55. PMID: 17032691.

Schoenfeld DA. Sample-size formula for the proportional-hazards regression model. **Biometrics.** 1983;39(2):499-503. doi: https://doi.org/10.2307/2531021. PMID: 6354290.

Li F, Thomas LE, Li F. Addressing extreme propensity scores via the overlap weights. **Am J Epidemiol.** 2019;188(1):250-257. doi: https://doi.org/10.1093/aje/kwy201. PMID: 30189042.

Généreux P, Palmerini T, Caixeta A, et al. Quantification and impact of untreated coronary artery disease after percutaneous coronary intervention: the residual SYNTAX score. **J Am Coll Cardiol.** 2012;59(24):2165-2174. doi: https://doi.org/10.1016/j.jacc.2012.03.010. PMID: 22483327.

Wald DS, Morris JK, Wald NJ, Chase AJ, Edwards RJ, Hughes LO, et al. Preventive angioplasty in acute myocardial infarction. N Engl J Med. 2013;369(12):1115-1123. doi: https://doi.org/10.1056/NEJMoa1305520. PMID: 23991625.

Gershlick AH, Khan JN, Kelly DJ, Greenwood JP, Sasikaran T, Curzen N, et al. Randomized trial of complete versus lesion-only revascularization in patients undergoing primary PCI for STEMI and multivessel disease (CvLPRIT). J Am Coll Cardiol. 2015;65(10):963-972. doi: https://doi.org/10.1016/j.jacc.2014.12.038. PMID: 25766941.

Engstrøm T, Kelbæk H, Helqvist S, Høfsten DE, Kløvgaard L, Holmvang L, et al. Complete revascularisation versus treatment of the culprit lesion only in patients with STEMI and multivessel disease (DANAMI-3-PRIMULTI): an open-label, randomised controlled trial. Lancet. 2015;386(9994):665-671. doi: https://doi.org/10.1016/S0140-6736(15)60648-1. PMID: 26347918.

Smits PC, Abdel-Wahab M, Neumann FJ, Boxma-de Klerk BM, Lunde K, Schotborgh CE, et al. Fractional flow reserve–guided multivessel angioplasty in myocardial infarction. N Engl J Med. 2017;376(13):1234-1244. doi: https://doi.org/10.1056/NEJMoa1701067. PMID: 28317428.

Puymirat E, Cayla G, Simon T, Steg PG, Montalescot G, Danchin N, et al. Multivessel PCI guided by fractional flow reserve or angiography for myocardial infarction (FLOWER-MI). N Engl J Med. 2021;385(4):297-308. doi: https://doi.org/10.1056/NEJMoa2104650. PMID: 33999545.

Shah JA, Kumar R, Solangi BA, Khan KA, Ahmed T, Khowaja S, et al. One-year major adverse cardiovascular events among same-day discharged patients after primary percutaneous coronary intervention at a tertiary care cardiac centre in Karachi, Pakistan: a prospective observational study. BMJ Open. 2023;13(4):e067971. doi: https://doi.org/10.1136/bmjopen-2022-067971. PMID: 37037620.