

Complete versus incomplete coronary revascularisation in patients undergoing PCI after TAVI

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The results of this study were presented in the form of a poster at the 2026 EAPCI Congress, Munich, Germany.³⁴

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ABSTRACT

Background The optimal revascularisation strategy for patients undergoing percutaneous coronary intervention (PCI) after transcatheter aortic valve implantation (TAVI) remains undefined. In particular, the prognostic impact of complete revascularisation (CR) versus incomplete revascularisation (ICR) in this setting is unknown.

Aims To evaluate the long-term clinical outcomes of CR versus ICR in patients undergoing PCI after TAVI.

Methods We analysed 447 patients from the multicentre, international Revascularization After Transcatheter Aortic Valve Implantation (REVIVAL) registry who underwent PCI after TAVI between 2008 and 2023. Patients were classified as CR or ICR according to the presence of residual significant stenosis following PCI. The primary endpoint was the 4-year incidence of major adverse cardiovascular events (MACE), a composite of cardiovascular death, myocardial infarction or stroke. The Kaplan-Meier method was used to estimate cumulative event rates, and a weighted Cox regression model employing an entropy balance approach was used to adjust for possible confounders.

Results ICR was achieved in 132 patients (29.5%) and CR in 315 patients (70.5%). The mean follow-up after PCI was 1065±904 days (1213±938 in the ICR group; 1002±883 in the CR group). The crude 4-year incidence of MACE was 35.0% with ICR and 34.7% with CR (HR 0.93, 95% CI 0.62 to 1.39, p=0.710). Adjusted analysis confirmed no significant difference (32.5% vs 34.1%; HR 1.01, 95% CI 0.62 to 1.64, p=0.971).

Conclusion In this registry, CR after TAVI was common but not associated with improved outcomes compared with ICR. These results support a selective rather than systematic pursuit of CR in elderly, high-risk post-TAVI patients.

WHAT IS ALREADY KNOWN ON THIS TOPIC

- ⇒ Patients who undergo percutaneous coronary intervention (PCI) after previous transcatheter aortic valve implantation (TAVI) are at an increased risk for adverse cardiovascular events.
- ⇒ Complete revascularisation is generally the preferred strategy in patients undergoing PCI for CAD, but the optimal strategy in TAVI patients is unclear.

WHAT THIS STUDY ADDS

- ⇒ Complete revascularisation was commonly achieved in patients who previously underwent TAVI. However, the achievement of complete revascularisation was not associated with different clinical outcomes compared with incomplete revascularisation.

HOW THIS STUDY MIGHT AFFECT RESEARCH, PRACTICE OR POLICY

- ⇒ The results of this study underline the need for an individualised approach to coronary revascularisation in TAVI patients, and further research is warranted to inform clinicians on the indications for different revascularisation strategies.

INTRODUCTION

Coronary artery disease (CAD) and aortic stenosis (AS) share common pathogenic mechanisms and frequently coexist,¹ with CAD present in up to 75% of patients with AS and associated with worse clinical outcomes after aortic valve interventions.^{2,3}

Transcatheter aortic valve implantation (TAVI) has become the standard of care for symptomatic severe AS across a broad spectrum of surgical risk.^{4–7} As TAVI is increasingly performed in younger patients with longer

life expectancy, the incidence of unplanned percutaneous coronary intervention (PCI) after TAVI is rising, underscoring the importance of defining optimal revascularisation strategies in this expanding population.⁸

In the general CAD population, complete revascularisation (CR) is often associated with improved outcomes compared with incomplete revascularisation (ICR).⁹ In patients undergoing TAVI, most available evidence concerns revascularisation before the TAVI procedure. The NOTION-3 (Routine Revascularization With Percutaneous Coronary Intervention in Patients With Coronary Artery Disease Undergoing Transcatheter Aortic Valve Implantation - the Nordic Aortic Valve Intervention-3) trial demonstrated improved outcomes in patients randomised to PCI versus no PCI, with CR achieved in 89% of treated patients.¹⁰ In contrast, the randomised, non-inferiority ACTIVATION (Percutaneous Coronary Intervention Prior to Transcatheter Aortic Valve Implantation) trial showed that rates of death and rehospitalisation at 1 year were similar between PCI and no PCI prior to TAVI.¹¹ Also, observational studies have produced conflicting evidence.^{12–14}

As a result, no consensus has been reached on the optimal revascularisation strategy in patients undergoing TAVI, including timing and completeness of revascularisation.³ In particular, the post-TAVI setting remains unexplored, and it is unknown whether achieving complete revascularisation when performing PCI after TAVI would offer a clinical benefit. This is a clinically relevant gap, as achieving CR in this setting may be hindered by procedural challenges, including impaired coronary access due to the transcatheter valve frame, complex CAD anatomy and significant comorbidities.⁹

This study addresses this unmet need by evaluating the prognostic impact of complete versus incomplete revascularisation in patients with previous TAVI undergoing PCI.

METHODS

Study population

This study uses data collected in the Revascularization After Transcatheter Aortic Valve Implantation (REVIVAL; NCT03283501) registry, a multicentre, international, observational registry using pooled individual data of consecutive patients who underwent PCI after TAVI in 21 centres across Europe from 2008 to 2023 (online supplemental table 1). Completeness of revascularisation was defined anatomically on the final angiogram as the absence of any residual stenosis judged significant by the operator (generally $\geq 70\%$ in coronary vessels or segments ≥ 2.25 mm in diameter). ICR was defined by the persistence of at least one such lesion. Functional (physiology-guided) lesion assessment was not systematically performed, and revascularisation decisions were left to operator judgement without predefined criteria for deferring treatment of individual lesions.

The study was conducted according to the ethical principles of the Declaration of Helsinki, and each centre's institutional review board approved the corresponding study protocols. Informed written consent was obtained from all participants before enrolment in the study.

Standard techniques were used for TAVI and PCI in all centres, and the choice of the device was left to the operator's judgement.

All participant data, including baseline characteristics, data from the TAVI and PCI procedures, antithrombotic drug therapy and clinical outcomes, were collected by each centre in a prespecified data extraction sheet. The data were then anonymised and merged into a general core dataset. Patients were followed up using inpatient or outpatient visits or telephone interviews.

Endpoints

The primary endpoint of this study was the occurrence of a major adverse cardiovascular event (MACE), which was defined as the composite of cardiovascular (CV) death, myocardial infarction (MI) or stroke at 4-year follow-up. Secondary outcome parameters included the following: the individual components of the composite primary outcome, all-cause death, target vessel MI, periprocedural MI, target vessel revascularisation (TVR), target lesion revascularisation, target vessel failure (defined as a composite of TVR, MI and CV death), definite or probable stent thrombosis, bleeding (defined as Bleeding Academic Research Consortium type 2–5) and CV hospitalisation. All endpoints were assessed after a follow-up period of 4 years. Endpoint definitions can be found in online supplemental table 2.

Statistical analysis

Continuous variables are presented as mean $\pm$ SD or as median with IQR, whereas categorical variables are reported as absolute counts and corresponding percentages. Covariate balance between patients who underwent PCI with CR and those with ICR was evaluated using the standardised mean difference (SMD), with values below 0.1 considered indicative of good balance.

We applied entropy balancing to control for confounding and achieve covariate balance between the two groups at the time of PCI. This procedure reweights observations so that the covariate distributions in the two groups become as similar as possible. Weights were then truncated at the first and 99th percentiles to improve estimate stability and limit the influence of extreme weights.¹⁵ In our analysis, this trimming procedure did not introduce bias, as SMDs after truncation confirmed that covariates remained well balanced across groups.

We computed a weighted model adjusting for potential confounders, defined as pre-exposure variables associated with both the exposure and the outcome but not lying on the causal pathway (online supplemental figure 1). Online supplemental table 3 shows a complete list of all covariates included in the adjusted model. Online

supplemental figure 2 provides the pre-weighting and post-weighting balance diagnostics for the model.

To account for potential correlations introduced by the weighting process, we used a robust variance estimator to estimate 95% CIs.

Cox regression was then performed to estimate the effect of different revascularisation strategies on clinical outcomes, and the Kaplan-Meier method was used for the estimation of the crude and weighted cumulative incidences. These incidences were compared between the two groups by using the log-rank test for the time to first event.

We performed several sensitivity analyses to assess the statistical robustness of our findings. To test for the potential overestimation of risks by the Kaplan-Meier method, we applied an adjusted Fine-Gray hazard model as an alternative to the primary Cox model to estimate the subdistribution HRs while accounting for competing risks. The competing risks were defined as follows: non-cardiovascular death was treated as the competing risk for the composite endpoint of MACE and the individual endpoint of cardiovascular death, and all-cause death was treated as the competing risk for all other non-fatal endpoints. This way, we accounted for the nature of each of the different endpoints.

Because more than half of the cohort was enrolled at a single centre, we conducted a dedicated sensitivity analysis to assess the robustness of our findings to between-centre heterogeneity (online supplemental table 4). To reduce the statistical instability associated with very small clusters, we excluded centres contributing fewer than 10 patients. In the resulting restricted cohort, we re-estimated the weighted Cox regression using the same covariates as in the adjusted model, while additionally including country as a balancing variable in the entropy weighting procedure. Country, rather than individual centre, was used because most centres contributed few patients and balancing on centre-level indicators would have produced unstable weights; country captures the main expected sources of between-centre heterogeneity (healthcare system, referral patterns and local practice). Additionally, subgroup analyses with formal tests for interaction were conducted for the primary endpoint on the basis of clinical presentation (acute vs chronic coronary syndrome), sex, median age and timing of PCI after TAVI (early vs late groups using a 3-month cut-off). Lastly, to address the difference in follow-up duration between groups, we performed a Kaplan-Meier analysis of the various outcomes with follow-up truncated at 2 years.

The *p* values <0.05 were considered statistically significant. All analyses were performed using R, V.4.3.1 (R Foundation for Statistical Computing, Vienna, Austria).

RESULTS

Baseline clinical and procedural characteristics

Out of 464 patients included in the REVIVAL registry, 447 patients were included in the analysis, and 17 were

excluded due to missing data. ICR was achieved in 132 patients (29.5%) and CR in 315 patients (70.5%). The mean time from TAVI to PCI was 347 ± 489 days in the ICR group and 386 ± 568 days in the CR group.

Tables 1 and 2 present the baseline demographic characteristics as well as clinical and procedural characteristics in the ICR and CR cohorts before and after weighting for clinical confounders at the time of the PCI.

Before weighting, patients who received ICR were slightly older (81.4 ± 6.2 vs 80.8 ± 6.2 years, SMD=0.101), more likely to be men (68.2% vs 61.0%, SMD=0.152), to have a family history of CAD (17.6% vs 13.3%, SMD=0.121) and to be diabetic (36.4% vs 28.9%, SMD=0.160). Patients with ICR furthermore had more previous revascularisations (53.8% vs 45.1%, SMD=0.175). These patients also had higher rates of procedural complexity (40.7% vs 30.1%, SMD=0.221), and PCI was less likely to be deemed successful (89.4% vs 99.7%, SMD=0.465). The two groups were similar regarding hypertension (90.2% vs 89.5%, SMD=0.021), body mass index (26.9 ± 4.3 vs 27.3 ± 5.1 , SMD=0.078) and left ventricular ejection fraction (54.0 ± 11.7 vs 54.9 ± 12.0 , SMD=0.083).

An acute coronary syndrome (ACS) as index presentation was more common in the ICR cohort compared with the CR cohort (36.4% vs 32.9%; SMD=0.171). When broken down by clinical subtype, unstable angina occurred in 10.6% versus 7.1%, non-ST-elevation myocardial infarction in 22.0% versus 19.7%, and ST-elevation myocardial infarction in 3.8% versus 6.1% of patients with ICR and CR, respectively.

After adjusting for several demographic, clinical and TAVI-related confounders, balance was achieved for most of the included variables (table 1, online supplemental figure 2), while some differences in procedural characteristics remained (table 2).

Outcomes before weighting

Mean follow-up after PCI was 1065 ± 904 days overall, 1213 ± 938 days in the ICR group and 1002 ± 883 days in the CR group. 282 patients (63.1%) completed a 4-year follow-up.

The 4-year crude incidence of MACE was 35.0% in the ICR group and 34.7% in the CR group (HR 0.93, 95% CI 0.62 to 1.39, *p*=0.710; table 3 and figure 1A). Similarly, no significant differences were found in all-cause death (34.6% vs 40.9%, HR 1.16, 95% CI 0.78 to 1.71, *p*=0.459; table 3), CV death (21.3% in the ICR group, 25.5% in the CR group; HR 1.10, 95% CI 0.66 to 1.84, *p*=0.709; table 3 and figure 1B), MI (13.7% vs 11.4%, HR 0.84, 95% CI 0.43 to 1.65, *p*=0.609; table 3 and figure 1C), stroke (5.2% vs 8.4%, HR 1.41, 95% CI 0.51 to 3.89, *p*=0.504; table 3 and figure 1D) or any other of the secondary endpoints (table 3).

Outcomes after weighting

In the adjusted model, the 4-year adjusted incidence of MACE was 32.5% in the ICR group and 34.1% in the CR group (HR 1.01, 95% CI 0.62 to 1.64, *p*=0.971;

Table 1 Baseline clinical characteristics of the population with no adjustment and after adjustment

	Unadjusted			Adjusted		
	ICR	CR	SMD	ICR	CR	SMD
Number of patients	132	315		117.4	266.0	
Clinical characteristics						
Age, mean (SD)	81.4 (6.2)	80.8 (6.2)	0.101	81.0 (6.0)	80.9 (6.1)	0.013
Females, n (%)	42 (31.8)	123 (39.0)	0.152	44.5 (37.9)	101.8 (38.3)	0.008
Logistic Euroscore, mean (SD)	15.5 (12.9)	13.2 (11.3)	0.187	14.2 (11.5)	13.4 (11.5)	0.068
Euroscore 2, mean (SD)	7.0 (7.5)	5.0 (5.6)	0.299	5.9 (5.7)	5.3 (6.3)	0.107
Body mass index, mean (SD)	26.9 (4.3)	27.3 (5.1)	0.078	27.2 (4.6)	27.2 (4.9)	0.002
Hypertension, n (%)	119 (90.2)	282 (89.5)	0.021	105.6 (89.9)	238.3 (89.6)	0.011
Diabetes, n (%)	48 (36.4)	91 (28.9)	0.160	35.9 (30.6)	81.0 (30.5)	0.003
Dyslipidaemia, n (%)	99 (75.0)	224 (71.1)	0.088	86.1 (73.3)	195.3 (73.4)	0.002
Family history of coronary artery disease, n (%)	12 (17.6)	19 (13.3)	0.121	7.4 (12.9)	14.5 (13.2)	0.008
Previous PCI, n (%)	58 (43.9)	133 (42.2)	0.035	48.1 (41.0)	110.1 (41.4)	0.008
Previous CABG, n (%)	31 (23.5)	32 (10.2)	0.362	17.1 (14.5)	38.1 (14.3)	0.006
Previous PCI or CABG, n (%)	71 (53.8)	142 (45.1)	0.175	55.3 (47.1)	122.7 (46.1)	0.020
Peripheral arterial disease, n (%)	20 (15.3)	62 (19.7)	0.116	21.5 (18.3)	48.5 (18.2)	0.002
Estimated glomerular filtration rate, mean (SD)	60.6 (23.5)	62.6 (21.1)	0.090	62.6 (23.1)	62.8 (21.3)	0.007
Left ventricular ejection fraction, mean (SD)	54.0 (11.7)	54.9 (12.0)	0.083	55.1 (12.2)	55.0 (11.9)	0.004
TAVI characteristics						
Year of TAVI procedure, n (%)			0.173			0.006
2008–2012	24 (18.2)	40 (12.7)		14.5 (12.3)	32.6 (12.2)	
2012–2017	69 (52.3)	163 (51.9)		62.6 (53.3)	141.3 (53.1)	
2018–2023	39 (29.5)	111 (35.4)		40.4 (34.4)	92.1 (34.6)	
Transapical approach, n (%)	10 (7.6)	26 (8.3)	0.025	8.6 (7.3)	19.4 (7.3)	0.001
Valve size, mean (SD)	26.6 (2.2)	26.4 (2.4)	0.087	26.37 (2.2)	26.36 (2.3)	0.004
Valve-in-valve, n (%)	2 (1.5)	3 (1.0)	0.051	1.5 (1.3)	3.5 (1.3)	0.001
Postdilatation, n (%)	38 (28.8)	86 (27.3)	0.033	35.9 (30.6)	81.0 (30.5)	0.003

Variables included in the adjusted model are reported in online supplemental table 3.
CABG, coronary artery bypass graft; CR, complete revascularisation; ICR, incomplete revascularisation; PCI, percutaneous coronary intervention; SMD, standardised mean difference; TAVI, transcatheter aortic valve implantation.

table 3 and figure 2A). Similarly, no statistically significant differences were found regarding all-cause death (35.4% vs 40.9%; HR 1.18, 95% CI 0.75 to 1.86, $p=0.466$; table 3), CV death (22.1% vs 25.2%; HR 1.09, 95% CI 0.59 to 1.99, $p=0.787$; table 3 and figure 2B), MI (10.4% vs 11.6%; HR 1.05, 95% CI 0.42 to 2.60, $p=0.919$; table 3 and figure 2C), stroke (5.8% vs 7.3%; HR 1.20, 95% CI 0.41 to 3.48, $p=0.744$; table 3 and figure 2D) or any other of the secondary endpoints (table 3).

Sensitivity analyses

The statistical robustness of our findings from the adjusted model was confirmed across multiple sensitivity analyses. In the country-level analysis, in which centres and countries with statistical instability were excluded, the adjusted HR for 4-year MACE was consistent with the primary results (online supplemental table 4). A formal competing risk analysis was performed to evaluate

a possible overestimation by the Kaplan-Meier method and showed results which were consistent with the main analysis (online supplemental table 5). Furthermore, formal testing for interaction showed a consistent effect of revascularisation strategies on MACE at 4 years across all subgroups of interest (all p -interaction >0.10 ; online supplemental figure 3). In the sensitivity analysis truncating follow-up at 2 years, the MACE rate was 19.8% in the ICR group and 18.8% in the CR group (HR 0.87, 95% CI 0.49 to 1.56, $p=0.64$), with no statistically significant differences in the secondary outcomes (online supplemental figure 4).

DISCUSSION

To our knowledge, this is the first dedicated investigation assessing the prognostic impact of complete versus incomplete revascularisation in patients undergoing PCI

Table 2 PCI characteristics of the population with no adjustment and after adjustment

	Unadjusted			Adjusted		
	ICR	CR	SMD	ICR	CR	SMD
Number of patients	132	315		117.4	266.0	
PCI characteristics						
Days from TAVI to PCI, mean (SD)	346.9 (488.7)	385.9 (568.5)	0.074	357.4 (481.7)	356.8 (536.0)	0.001
PCI planned at time of TAVI, n (%)	44 (33.3)	65 (20.6)	0.289	32.1 (27.4)	72.0 (27.1)	0.006
ACS as index presentation, n (%)	48 (36.4)	102 (32.9)	0.171	38.4 (32.7)	86.6 (32.6)	0.004
Radial access, n (%)	50 (39.4)	153 (50.2)	0.218	45.2 (39.1)	125.5 (48.7)	0.194
Number of diseased vessels, mean (SD)	1.3 (0.5)	1.2 (0.5)	0.069	1.3 (0.5)	1.3 (0.5)	0.083
ACC/AHA 2B or C, n (%)	79 (81.4)	233 (89.6)	0.234	74.4 (84.2)	197.7 (89.5)	0.159
Severe calcifications, n (%)	27 (20.5)	86 (27.3)	0.161	26.0 (22.2)	66.6 (25.0)	0.067
Chronic total occlusion, n (%)	4 (3.0)	7 (2.2)	0.051	4.4 (3.7)	5.5 (2.1)	0.100
Evidence of thrombus, n (%)	8 (6.6)	9 (3.0)	0.167	7.5 (6.8)	9.3 (3.7)	0.142
Intravascular imaging, n (%)	9 (6.8)	23 (7.3)	0.019	7.3 (6.2)	20.8 (7.8)	0.064
Bifurcation, n (%)	37 (28.0)	77 (24.4)	0.082	30.5 (26.0)	68.3 (25.7)	0.008
In-stent restenosis, n (%)	16 (12.1)	40 (12.7)	0.018	16.4 (14.0)	34.7 (13.0)	0.027
Thrombus aspiration, n (%)	2 (1.6)	0 (0.0)	0.181	1.4 (1.3)	0.0 (0.0)	0.159
Use of plaque modification device, n (%)	4 (3.4)	15 (5.1)	0.087	5.1 (4.7)	13.9 (5.6)	0.040
PCI on left Main (coronary artery), n (%)	21 (16.7)	48 (15.8)	0.024	17.9 (15.7)	42.9 (16.6)	0.026
PCI on left anterior descending (coronary artery), n (%)	51 (40.5)	146 (48.0)	0.152	49.9 (43.8)	124.9 (48.5)	0.093
PCI with drug-eluting stent, n (%)	112 (90.3)	279 (93.3)	0.109	101.7 (91.4)	238.3 (93.0)	0.059
PCI with drug-eluting balloon, n (%)	12 (9.7)	24 (8.0)	0.058	11.8 (10.6)	20.7 (8.1)	0.087
Number of lesions per patient, mean (SD)	1.39 (0.8)	1.4 (0.7)	0.042	1.4 (0.8)	1.4 (0.7)	0.060
Number of stents per lesion, mean (SD)	1.67 (1.21)	1.62 (1.02)	0.043	1.7 (1.2)	1.7 (1.1)	0.061
Total stent length per lesion, mean (SD)	37.29 (28.6)	38.1 (25.2)	0.028	39.2 (28.1)	39.0 (26.6)	0.008
Minimum stent diameter, mean (SD)	2.96 (0.62)	2.9 (0.6)	0.037	2.95 (0.61)	3.0 (0.6)	0.002
Total number of stents per patient, mean (SD)	1.7 (1.3)	1.53 (1.01)	0.144	1.8 (1.2)	1.59 (1.00)	0.139
Total stent length per patient, mean (SD)	36.6 (29.9)	31.1 (26.3)	0.195	39.0 (30.0)	33.1 (27.5)	0.206
Complex PCI, n (%)	50 (40.7)	88 (30.1)	0.221	41.5 (37.4)	79.3 (31.4)	0.126
Successful PCI, n (%)	118 (89.4)	314 (99.7)	0.465	105.9 (90.2)	266.0 (100.0)	0.465
Angiographic success, n (%)	121 (100.0)	298 (98.0)	0.201	110.5 (100.0)	255.4 (99.1)	0.136
Coronary occlusion n (%)	0 (0.0)	4 (1.3)	0.160	0.0 (0.0)	2.6 (1.0)	0.141
Post-PCI antiplatelet therapy						
Oral anticoagulation, n (%)	31 (24.0)	78 (25.4)	0.032	29.8 (25.4)	67.2 (25.3)	0.003
Short dual antiplatelet therapy, n (%)	30 (26.3)	74 (25.0)	0.030	27.2 (25.9)	66.6 (26.3)	0.010

Variables included in the adjusted model are reported in online supplemental table 3.

ACS was defined as unstable angina, non-ST-elevation myocardial infarction or ST-elevation myocardial infarction. Complex PCI was defined as at least one among ≥ 3 vessels treated, ≥ 3 lesions treated, or bifurcation lesions treated.

ACS, acute coronary syndrome; CR, complete revascularisation; ICR, incomplete revascularisation; PCI, percutaneous coronary intervention; SMD, standardised mean difference; TAVI, transcatheter aortic valve implantation.

after TAVI. The main findings can be summarised as follows:

1. In this elderly, high-risk cohort, CR was achieved in most cases (70.5%) yet was not associated with differences in 4-year MACE or other secondary endpoints.
2. This result was consistent across crude, adjusted and sensitivity analyses.

3. ICR was more frequent in the setting of greater procedural complexity and lower procedural success but did not translate into worse clinical outcomes.

In the general PCI population, CR is typically preferred when technically feasible, with more robust evidence supporting its benefit in ACS than in stable CAD.^{9 16 17} Evidence in the TAVI setting is limited and

Table 3 4-year clinical outcomes in the unadjusted population and after adjustment

Endpoint	Unadjusted				Adjusted for clinical variables			
	KM incidences at 4 years				KM incidences at 4 years			
	ICR (132 pts)	CR (315 pts)	HR (95% CI)	P value	ICR (117.4 pts)	CR (266.0 pts)	HR (95% CI)	P value
Major adverse cardiovascular events	35.0	34.7	0.93 (0.62 to 1.39)	0.710	32.5	34.1	1.01 (0.62 to 1.64)	0.971
Death	34.6	40.9	1.16 (0.78 to 1.71)	0.459	35.4	40.9	1.18 (0.75 to 1.86)	0.466
MI	13.7	11.4	0.84 (0.43 to 1.65)	0.609	10.4	11.6	1.05 (0.42 to 2.60)	0.919
Stroke	5.2	8.4	1.41 (0.51 to 3.89)	0.504	5.8	7.3	1.20 (0.41 to 3.48)	0.744
Cardiovascular death	21.3	25.5	1.10 (0.66 to 1.84)	0.709	22.1	25.2	1.09 (0.59 to 1.99)	0.787
Stent thrombosis	2.5	1.1	0.43 (0.09 to 2.11)	0.295	3.8	1.2	0.31 (0.06 to 1.67)	0.174
Periprocedural MI	9.6	8.1	0.53 (0.21 to 1.30)	0.164	9.7	7.1	0.43 (0.15 to 1.20)	0.105
Target vessel MI	9.6	5.4	0.55 (0.23 to 1.32)	0.181	8.8	5.9	0.62 (0.20 to 1.91)	0.408
Target lesion revascularisation	13.6	8.9	0.68 (0.34 to 1.37)	0.286	15.4	10.3	0.69 (0.31 to 1.54)	0.367
Target vessel revascularisation	21.6	13.1	0.69 (0.39 to 1.24)	0.215	22.2	15.2	0.80 (0.41 to 1.55)	0.504
Target vessel failure	34.9	36.4	1.00 (0.67 to 1.49)	0.990	33.5	38.5	1.14 (0.71 to 1.84)	0.581
Bleeding	3.2	5.7	1.26 (0.40 to 3.97)	0.690	4.3	4.4	1.14 (0.44 to 2.98)	0.790

Variables included in the adjusted model are reported in online supplemental table 3.

CR, complete revascularisation; ICR, incomplete revascularisation; KM, Kaplan-Meier; MI, myocardial infarction; PCI, percutaneous coronary intervention.

existing studies almost exclusively address PCI performed before or during TAVI, often as part of preprocedural CAD management.^{11 18} The clinical context of PCI after TAVI is different, frequently involving unplanned PCI for CAD progression or ACS, and thus prior findings may not be directly applicable.

The REVIVAL registry population was characterised by advanced age (median age of 81 years) and high surgical risk by both Logistic EuroSCORE and EuroSCORE II. In younger surgical cohorts, the prognostic importance of residual CAD is often greater, likely due to longer life expectancy and fewer comorbidities. This may explain why surgical guidelines advocate concomitant coronary artery bypass grafting in patients undergoing surgical aortic valve replacement.^{19 20} Such recommendations cannot be directly extrapolated to elderly, frail post-TAVI patients.²¹ The CR rate in this study (~70%) was considerably high, probably due to the low angiographic disease burden in our cohort (mean 1.25±0.49 diseased vessels). This reflects real-world practice, in which TAVI patients with significant CAD often undergo revascularisation

before or concomitantly with the valve procedure,²¹ as supported by the similarly high rate of prior PCI in both groups (43.9% in the ICR group and 42.2% in the CR group, SMD=0.035). This introduces a degree of selection bias, limiting the generalisability of the findings to patients presenting with complex multivessel disease after TAVI.

Interpretation of ICR depends heavily on its definition, which in this study was based on operator angiographic assessment without systematic functional evaluation. ICR can range from leaving untreated high-risk lesions due to technical or anatomical constraints to deferring intervention on minor, non-flow-limiting lesions.^{22 23} Previous work in TAVI populations has shown that only 'unreasonable' ICR, defined by leaving untreated high-risk lesions, is associated with increased mortality.^{12 24 25} Without functional lesion assessment, our analysis cannot distinguish between these categories, which may dilute any signal for prognostic difference. A procedural success rate of 89.4% in the ICR group further indicates that this cohort is heterogeneous, including both patients in whom

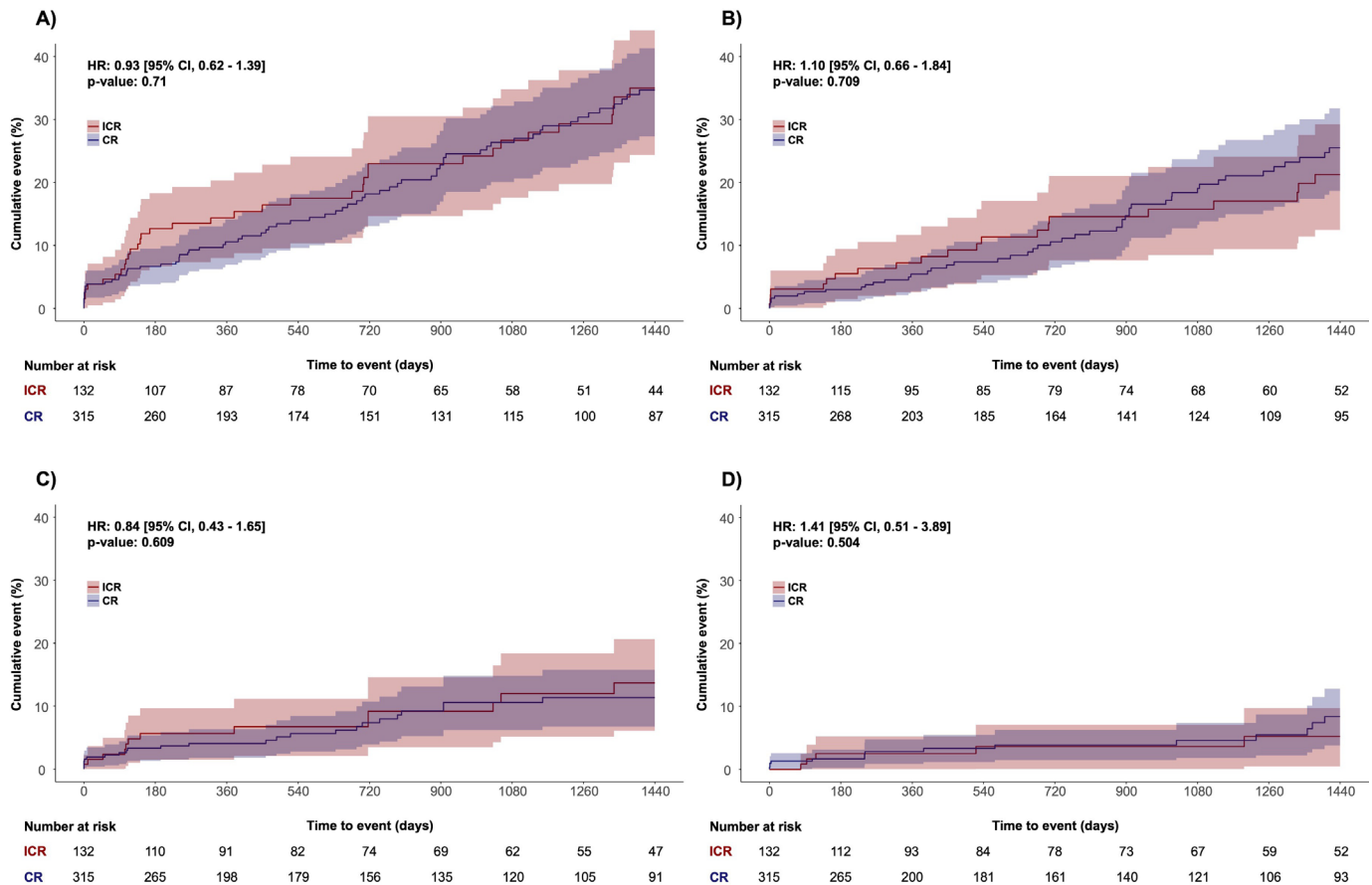


Figure 1 Kaplan-Meier estimates of 4-year clinical outcomes in the unadjusted population (A: major adverse cardiovascular event, B: cardiovascular death, C: myocardial infarction and D: stroke). CR, complete revascularisation; ICR, incomplete revascularisation.

revascularisation was intentionally deferred on clinical or anatomical grounds and, less frequently, patients in whom CR was not achieved because of procedural complications. Notably, part of what is anatomically classified as ICR may reflect reasonable ICR, that is, anatomical ICR but functional CR. Rather than undermining our findings, this reinforces that systematically pursuing anatomical CR may not be warranted in this population.

Non-randomised data in stable CAD have not conclusively shown that anatomical CR improves outcomes over ICR, and confounding remains a major limitation.¹³ Indeed, similarly to our study, ICR is often unintentional, arising from complications or technical failure, both of which could themselves worsen prognosis. Conversely, aggressive pursuit of CR may increase procedural risk without guaranteeing resolution of ischaemia, especially in the presence of small-vessel or microvascular disease.²⁶

In our study, ICR was associated with more complex anatomy, less radial access and lower procedural success, yet these factors did not result in worse outcomes. The absence of a clear crude or adjusted advantage for CR argues against aggressively pursuing CR in this frail cohort and rather supports an individually tailored revascularisation strategy.^{27 28} A selective ICR strategy may allow shorter procedures, reduced contrast use, lower intraprocedural risk, and potentially shorter durations

of intensified antiplatelet therapy, without compromising long-term outcomes.^{29 30}

Limitations

Several important limitations of this study must be acknowledged. First, due to the observational design of this study, the results should be considered hypothesis-generating. Randomised trials would be necessary to account for confounders and provide solid evidence on the preferable revascularisation strategy in TAVI patients. However, conducting such trials currently poses great challenges because the incidence of PCI after TAVI is still relatively low. Additionally, patient selection for PCI may be biased by comorbidities and frailty, potentially influencing clinical outcomes within the groups. Importantly, the differentiation between anatomical and functional ICR was not possible in this study. Residual CAD was assessed angiographically, and completeness was classified according to an operator-based anatomical definition, consistent with the pivotal randomised trials on revascularisation completeness,^{16 31-33} while functional lesion assessment was not systematically performed. Furthermore, the 15-year study period encompasses substantial advances in PCI techniques, and CR may not have been achievable in earlier enrolments because of technical limitations that would no longer apply with

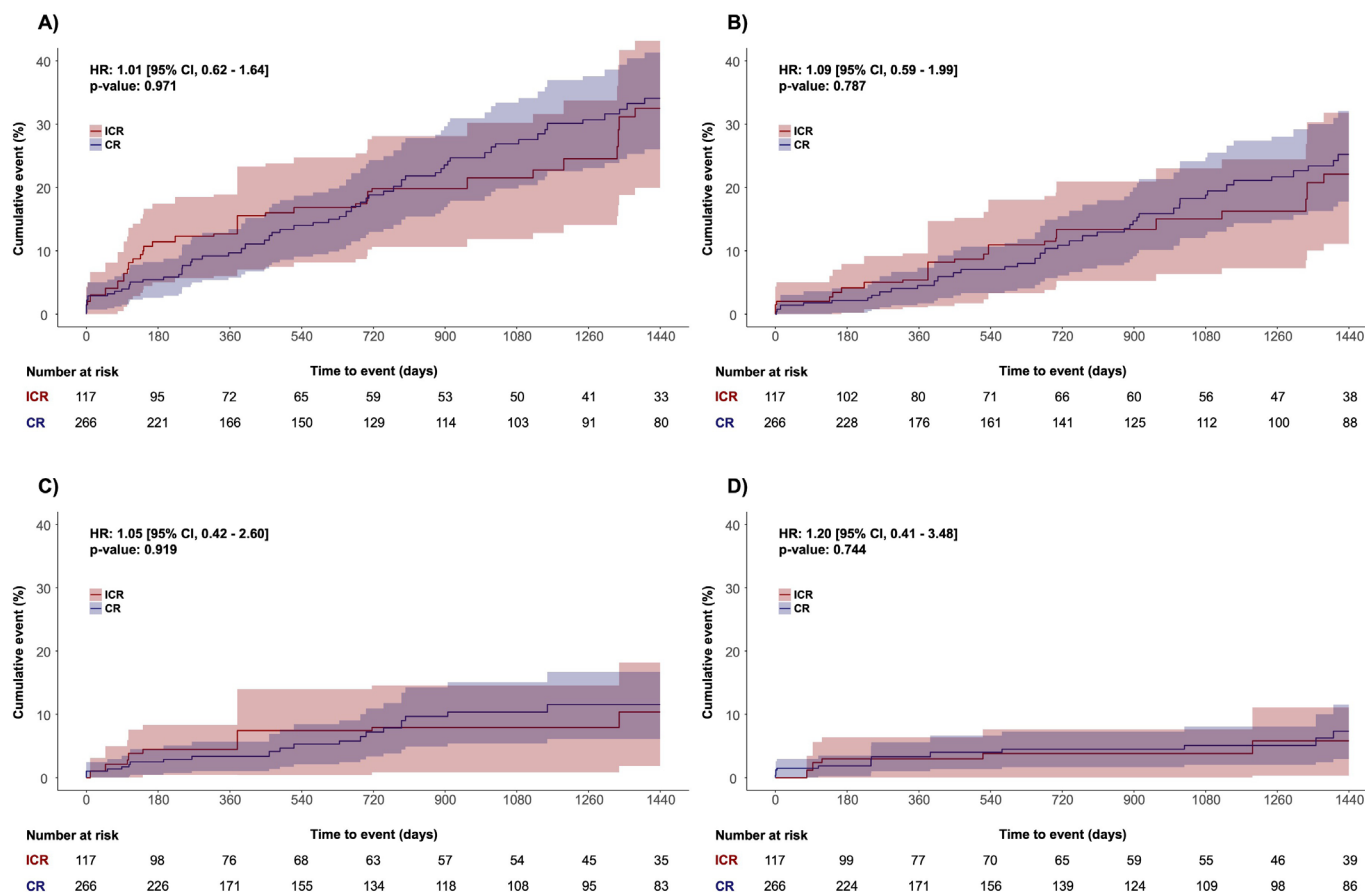


Figure 2 Kaplan-Meier estimates of 4-year clinical outcomes after adjustment (A: major adverse cardiovascular event, B: cardiovascular death, C: myocardial infarction and D: stroke). CR, complete revascularisation; ICR, incomplete revascularisation.

contemporary techniques. Finally, the modest sample size and event rate limit the power to detect more subtle differences in endpoints.

CONCLUSIONS

In this multicentre registry of elderly, high-risk patients undergoing PCI after TAVI, complete revascularisation was common but not associated with improved long-term outcomes compared with incomplete revascularisation. These findings support a selective rather than systematic pursuit of complete revascularisation in this population, balancing procedural feasibility and risk against uncertain prognostic benefit. Larger, prospective studies, ideally incorporating functional lesion assessment, are needed to define the optimal revascularisation strategy post-TAVI.

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