

Original Article

Prognostic factors after FFR-guided PCI in diffuse long coronary lesions: the value of post-PCI fractional flow reserve

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Received March 26, 2026; Accepted July 13, 2026; Epub October 15, 2026; Published October 30, 2026

Abstract: Objectives: To identify prognostic factors influencing clinical outcomes after fractional flow reserve (FFR)-guided percutaneous coronary intervention (PCI) in patients with diffuse long coronary lesions, and to determine the prognostic value of post-PCI FFR. Methods: This retrospective study enrolled 327 patients with diffuse coronary lesions (≥ 20 mm) who underwent FFR-guided PCI between January 2018 and December 2022. FFR was measured using a 0.014-inch pressure wire during maximal adenosine-induced hyperemia, calculated as the ratio of mean distal coronary pressure to mean aortic pressure. Post-PCI FFR was recorded with the sensor positioned ≥ 10 mm distal to the stented segment. The primary endpoint was major adverse cardiovascular events (MACE), comprising target lesion revascularization, target vessel myocardial infarction, and cardiac death. Independent predictors were identified using multivariate Cox regression. Results: During a median follow-up of 24 months, 57 patients (17.4%) experienced MACE. Five independent predictors were identified: suboptimal post-PCI FFR < 0.90 (hazard ratio [HR], 2.68; 95% confidence interval [CI], 1.78-4.02; $P < 0.001$), lesion length ≥ 30 mm (HR, 1.95; $P = 0.002$), diabetes mellitus (HR, 1.84; $P = 0.003$), severe calcification requiring plaque modification (HR, 1.78; $P = 0.004$), and baseline FFR ≤ 0.60 (HR, 1.64; $P = 0.012$). Achieving optimal post-PCI FFR (≥ 0.90) reduced MACE risk by 63%. Conclusions: Post-PCI FFR is the strongest predictor of long-term outcomes after FFR-guided PCI for diffuse long lesions. Achieving post-PCI FFR ≥ 0.90 should be a procedural target, which supports a physiology-guided optimization strategy to improve prognosis.

Keywords: Fractional flow reserve, diffuse coronary disease, long lesions, percutaneous coronary intervention, prognostic factors

Introduction

Coronary heart disease (CHD) affects approximately 126 million people worldwide and remains a leading cause of mortality. Percutaneous coronary intervention (PCI) has transformed the management of CHD; however, treatment of diffuse long coronary lesions remains a major clinical challenge. Diffuse atherosclerotic disease, defined as continuous atherosclerotic involvement of a coronary segment ≥ 20 mm, is present in up to 40% of patients undergoing diagnostic angiography and is associated with greater procedural complexity, higher periprocedural risk, and worse

long-term outcomes compared with focal stenoses [1-3].

The technical challenges of treating diffuse lesions include extensive calcium deposits, vascular tortuosity, and overlapping atherosclerotic plaques, each of which compromise procedural success [4]. Geographic miss (incomplete coverage of the diseased segment by stent edges) predisposes to future ischemic events [5]. Furthermore, vessel reconstruction frequently requires multiple or extended stents, increasing the risk of stent thrombosis, target lesion failure, and in-stent restenosis [6]. Historically, these limitations have favored sur-

gical revascularization through coronary artery bypass grafting over percutaneous approaches in such patients.

Recent advances in device technology, including new-generation drug-eluting stents (DES), specialized plaque modification tools, and intravascular imaging, have expanded the role of PCI in this setting [7]. Nevertheless, consensus regarding optimal management of diffuse coronary disease remains lacking, and treatment strategies vary considerably across centers. The introduction of physiological lesion assessment using fractional flow reserve (FFR) has refined diagnostic algorithms by quantifying the ratio of hyperemic distal coronary pressure to aortic pressure, providing an objective measure of ischemic burden [8]. The landmark FAME trials demonstrated that limiting stenting to physiologically significant lesions improve clinical outcomes compared with angiography-guided strategies [9]. However, the role of FFR in diffuse long lesions is less well established. Diffuse disease produces a gradual, continuous decline in perfusion pressure rather than the abrupt pressure drop seen in focal stenoses, which complicates functional interpretation [10]. It remains uncertain whether the conventional ischemic threshold of $\text{FFR} \leq 0.80$ is applicable in this context. Moreover, post-PCI FFR frequently remains abnormal in diffuse vessels even after angiographically successful stenting, due to residual untreated plaque or microvascular dysfunction [11].

Previous prognostic studies have identified clinical and anatomical risk factors for adverse outcomes after complex PCI, including advanced age, renal insufficiency, diabetes mellitus, small vessel diameter, calcification, and total stent length [12]. However, few studies have specifically focused on FFR-guided PCI in diffuse long lesions or systematically integrated physiological with clinical and anatomical parameters. We therefore conducted this study to comprehensively identify prognostic factors determining clinical outcomes after FFR-guided PCI in diffuse long coronary lesions, with the aim of developing a clinically applicable risk stratification model.

Methods

Cohort formation and research architecture

This retrospective observational study was conducted at the Department of Cardiovascular Medicine, Sichuan Provincial People's Hospital, University of Electronic Science and Technology of China. We consecutively enrolled patients who underwent FFR-guided PCI for diffuse long coronary lesions between January 2018 and December 2022. The study protocol was approved by the Deyang People's Hospital Ethics Committee (approval number: CARD-2023-0456), and the requirement for informed consent was waived due to the retrospective and de-identified nature of the data. The study was conducted in accordance with the Declaration of Helsinki. The ethics approval document is provided as a supplementary file.

Inclusion criteria: (1) Age ≥ 18 years; (2) Presence of at least one diffuse long coronary lesion, defined by quantitative coronary angiography (QCA) as a continuous atherosclerotic segment ≥ 20 mm in length; (3) Availability of baseline FFR measurement; (4) Implantation of drug-eluting stent(s); and (5) Documented post-PCI FFR measurement. Exclusion criteria: (1) Cardiogenic shock or ST-segment elevation myocardial infarction; (2) Protected left main coronary artery lesions; (3) Prior coronary artery bypass grafting; (4) In-stent restenosis; (5) Chronic total occlusion; (6) Severe left ventricular dysfunction (ejection fraction $< 30\%$); (7) Terminal non-cardiac illness with life expectancy < 1 year; and (8) Known intolerance to adenosine. After application of these criteria, 327 patients were included in the final analysis.

Definitions and classification of endpoints

Diffuse lesions were classified by QCA as moderate (20-29.9 mm) or very long (≥ 30 mm). Hemodynamic significance at baseline was defined as $\text{FFR} \leq 0.80$. Post-PCI FFR results were categorized as suboptimal (≤ 0.80), intermediate (0.81-0.89), or optimal (≥ 0.90), based on emerging evidence on post-procedural physiological targets. The primary endpoint was MACE, a composite of target lesion revascularization, target vessel myocardial infarction, and cardiac death. Secondary endpoints

included definite or probable stent thrombosis (defined according to Academic Research Consortium criteria), all-cause mortality, and non-target vessel revascularization. The median follow-up duration was 24 months (interquartile range: 18-36 months).

Cardiac death was defined as death due to heart failure, fatal arrhythmia, procedural complication, or myocardial infarction. Target vessel myocardial infarction required ischemic symptoms or electrocardiographic changes accompanied by elevation of cardiac biomarkers exceeding five times the 99th percentile upper reference limit in patients with normal baseline values. Repeat revascularization was defined as any subsequent percutaneous or surgical intervention performed on the treated segment or within 5 mm of the stent edges.

Angiographic data processing and assessment of coronary artery quality

Coronary angiography was performed via standard transradial or transfemoral access. After intracoronary administration of nitroglycerin (100-200 µg), multiple orthogonal projections were obtained. Angiograms were analyzed offline by independent analysts blinded to clinical outcomes and FFR data, using validated QCA software (QAngio XA, Medis Medical Imaging Systems). QCA parameters included percent diameter stenosis, lesion length, minimum lumen diameter, and reference vessel diameter (RVD). Vessels with RVD <2.75 mm were classified as small vessels.

Lesion complexity was graded according to the American College of Cardiology/American Heart Association classification, with type B2 and C lesions considered highly complex. Calcification, bifurcation involvement, and vessel tortuosity were systematically recorded. Severe calcification was defined as dense radiopacities visible on both sides of the arterial lumen before contrast injection, as determined by two independent operators, with disagreements resolved by consensus. Extensive diffuse coronary plaques were defined as continuous atherosclerotic involvement ≥20 mm in length on QCA, with a diffuse disease index (calculated as the ratio of total diseased segment length to total target vessel length) used to quantify overall atherosclerotic burden.

Hemodynamic assessment and intervention protocol

FFR was measured using a 0.014-inch pressure wire (Verrata, Philips Volcano; or PressureWire X, Abbott Vascular). After equalization of pressure signals, the sensor was advanced distal to the target stenosis. Maximal hyperemia was induced either by intracoronary adenosine (80-150 µg) or by continuous intravenous infusion (140 µg/kg/min for ≥2 minutes). FFR was calculated as the ratio of mean distal coronary pressure to mean aortic pressure during stable hyperemia, with the lowest stable value recorded as the baseline FFR. FFR measurements were supervised by experienced interventional cardiologists, with all tracings reviewed for technical adequacy (signal stability, absence of pressure drift, and adequate hyperemia) before being accepted. Pressure drift exceeding 0.02 required repeat equalization and remeasurement.

Because diffuse disease rarely produces an isolated pressure drop, motorized or manual pullback during sustained hyperemia was frequently performed to map the pressure gradient along the vessel. When multiple discrete pressure step-ups were detected within a diffuse segment, resting indices such as instantaneous wave-free ratio (iFR) or resting full-cycle ratio (RFR) were used adjunctively to localize the dominant ischemic zone.

Procedural decisions, including access route, guide catheter selection, predilation, and stent sizing, were left to the operator's discretion. Stenting strategy - spot stenting versus full metal jacket - was determined based on combined angiographic and physiological findings. In heavily calcified lesions, plaque modification with cutting balloons, scoring balloons, or rotational atherectomy was performed prior to stent deployment. New-generation drug-eluting stents were used in more than 95% of procedures. After stent deployment and repeat nitroglycerin administration, the pressure sensor was repositioned at least 10 mm distal to the stented segment to record post-PCI FFR. Suboptimal results often prompted further post-dilation or additional stenting. All patients received dual antiplatelet therapy in accordance with contemporary clinical guidelines.

Follow-up scheme and data collection

Follow-up was conducted through structured clinical visits or telephone interviews at 1, 6, 12, and 24 months, and annually thereafter. Repeat angiography was performed only when clinically indicated. Medical records were obtained from external hospitals when patients reported events at outside institutions. Data collected included baseline demographics, metabolic profile (glycated hemoglobin [HbA1c], lipid panel, high-sensitivity C-reactive protein [hs-CRP]), procedural details, and physiological parameters. An independent clinical events committee blinded to angiographic and physiological data adjudicated all endpoints.

Statistical architecture

Continuous variables were expressed as mean $\pm$ standard deviation or median (interquartile range), depending on distribution assessed by the Shapiro-Wilk test. Between-group comparisons were performed using Student's t-test or Mann-Whitney U test for continuous variables, and chi-square or Fisher's exact test for categorical variables. χ^2 and t values are presented in the tables where applicable.

Survival was estimated using the Kaplan-Meier method, and between-group differences were assessed using the log-rank test. Independent predictors of MACE were identified using Cox proportional hazards regression. Variables with $P < 0.10$ in univariate analysis were entered into the multivariate model using stepwise variable selection. Results were reported as hazard ratios (HRs) with 95% confidence intervals (CIs). Multicollinearity was assessed using variance inflation factors (threshold > 5), and the proportional hazards assumption was verified using Schoenfeld residuals. Model discrimination was quantified using Harrell's C-statistic, with internal validation using 1,000 bootstrap replicates. A risk score was developed using β -coefficients from the final Cox model, and Kaplan-Meier curves were generated for risk-stratified subgroups. Subgroup analyses, interaction testing, and sensitivity analyses excluding patients lost to follow-up were performed. All analyses were performed using R version 4.0.3 and SPSS version 25.0; a two-sided P -value < 0.05 was considered statistically significant.

Results

Baseline clinical characteristics

The final analytical cohort comprised 327 patients with 364 diffuse long coronary lesions. During a median follow-up of 24 months, 57 patients (17.4%) experienced the primary MACE endpoint. Baseline demographic and metabolic characteristics are summarized in **Table 1**. Although demographic features such as age, sex, and body mass index were comparable between groups, metabolic abnormalities were significantly more prevalent in the MACE group. Diabetes mellitus was substantially more common among patients who experienced events (56.1% vs 31.9%, $P < 0.001$). Systemic inflammation (hs-CRP: 3.4 vs 1.9 mg/L, $P = 0.004$), suboptimal glycemic control (HbA1c: 6.9% vs 6.3%, $P = 0.002$), and mild renal impairment were also more prominent in the MACE group.

Angiographic and procedural characteristics

Anatomical complexity was strongly associated with clinical outcome (**Table 2**). Patients who experienced MACE had longer lesions (mean 34.8 vs 28.3 mm, $P < 0.001$) and a higher proportion of very long lesions (≥ 30 mm: 68.4% vs 44.8%, $P = 0.001$). Vessel caliber was smaller in the MACE group (RVD 2.62 vs 2.83 mm, $P = 0.002$), with a higher prevalence of small-vessel disease (56.1% vs 35.6%, $P = 0.004$). Severe calcification was nearly twice as common in the MACE group (38.6% vs 21.1%, $P = 0.007$), and aggressive plaque modification techniques, including cutting balloons and rotational atherectomy, were more frequently employed in these high-risk patients. Consequently, the MACE group required longer total stent length (45.6 vs 36.6 mm, $P < 0.001$), with longer procedure times and higher contrast volumes.

FFR measurements and clinical outcomes

Hemodynamic parameters demonstrated marked between-group differences (**Table 3**). Baseline ischemic burden was significantly more severe in the MACE group (pre-PCI FFR: 0.62 vs 0.69, $P < 0.001$). Although stent implantation increased FFR overall, the absolute post-procedural FFR achieved differed substantially between groups, with the MACE group

Post-PCI FFR in diffuse long coronary lesions

Table 1. Baseline clinical characteristics

Characteristic	All patients (n=327)	MACE group (n=57)	Non-MACE group (n=270)	χ^2/t	P
Age, years	64.7 ± 10.3	66.8 ± 9.7	64.2 ± 10.4	t=1.73	0.086
Male	231 (70.6)	39 (68.4)	192 (71.1)	$\chi^2=0.16$	0.687
BMI, kg/m ²	26.3 ± 4.2	26.8 ± 4.5	26.2 ± 4.1	t=0.99	0.325
Cardiovascular risk factors					
Hypertension	225 (68.8)	43 (75.4)	182 (67.4)	$\chi^2=1.41$	0.236
Diabetes mellitus	118 (36.1)	32 (56.1)	86 (31.9)	$\chi^2=12.04$	<0.001
Dyslipidemia	193 (59.0)	37 (64.9)	156 (57.8)	$\chi^2=0.99$	0.320
Current smoking	94 (28.7)	19 (33.3)	75 (27.8)	$\chi^2=0.71$	0.413
Family history of CHD	71 (21.7)	13 (22.8)	58 (21.5)	$\chi^2=0.05$	0.826
Medical history					
Previous MI	63 (19.3)	15 (26.3)	48 (17.8)	$\chi^2=2.21$	0.139
Previous PCI	83 (25.4)	18 (31.6)	65 (24.1)	$\chi^2=1.40$	0.238
Stroke/TIA	31 (9.5)	8 (14.0)	23 (8.5)	$\chi^2=1.67$	0.194
Chronic kidney disease (eGFR <60 mL/min/1.73 m ²)	58 (17.7)	16 (28.1)	42 (15.6)	$\chi^2=5.05$	0.024
COPD	39 (11.9)	9 (15.8)	30 (11.1)	$\chi^2=0.98$	0.323
Clinical presentation				$\chi^2=0.65$	0.721
(1) Stable angina	182 (55.7)	29 (50.9)	153 (56.7)		
(2) Unstable angina	97 (29.7)	19 (33.3)	78 (28.9)		
(3) NSTEMI	48 (14.7)	9 (15.8)	39 (14.4)		
Baseline laboratory values					
Total cholesterol, mg/dL	173.5 ± 42.1	179.2 ± 44.8	172.3 ± 41.4	t=1.13	0.254
LDL-C, mg/dL	97.6 ± 36.2	103.5 ± 38.7	96.3 ± 35.6	t=1.37	0.172
HDL-C, mg/dL	44.3 ± 11.6	42.1 ± 10.2	44.8 ± 11.9	t=1.59	0.103
Triglycerides, mg/dL	138 (98-192)	147 (104-213)	136 (96-189)	Z=1.45	0.148
hs-CRP, mg/L	2.1 (0.9-5.4)	3.4 (1.3-7.8)	1.9 (0.8-4.9)	Z=2.88	0.004
HbA1c, %	6.4 ± 1.3	6.9 ± 1.5	6.3 ± 1.2	t=3.28	0.002
Creatinine, mg/dL	1.05 ± 0.39	1.15 ± 0.47	1.03 ± 0.36	t=2.16	0.032
Medications at discharge					
Aspirin	327 (100)	57 (100)	270 (100)	-	1.000
P2Y12 inhibitor				$\chi^2=0.45$	0.797
(1) Clopidogrel	173 (52.9)	32 (56.1)	141 (52.2)		
(2) Ticagrelor	139 (42.5)	22 (38.6)	117 (43.3)		
(3) Prasugrel	15 (4.6)	3 (5.3)	12 (4.4)		
Statins	318 (97.2)	55 (96.5)	263 (97.4)	$\chi^2=0.15$	0.705
Beta-blocker	248 (75.8)	46 (80.7)	202 (74.8)	$\chi^2=0.89$	0.346
ACEi/ARB	217 (66.4)	40 (70.2)	177 (65.6)	$\chi^2=0.45$	0.505

Values are mean ± standard deviation, median (interquartile range), or n (%). Continuous variables were compared using Student's t-test (t value reported) or the Mann-Whitney U test (Z value reported); categorical variables were compared using the χ^2 test (χ^2 value reported). Aspirin was not testable (100% in both groups). ACEi, angiotensin-converting enzyme inhibitor; ARB, angiotensin receptor blocker; BMI, body mass index; CHD, coronary heart disease; CI, confidence interval; COPD, chronic obstructive pulmonary disease; eGFR, estimated glomerular filtration rate; HbA1c, glycated hemoglobin; HDL-C, high-density lipoprotein cholesterol; hs-CRP, high-sensitivity C-reactive protein; LDL-C, low-density lipoprotein cholesterol; MACE, major adverse cardiovascular events; MI, myocardial infarction; NSTEMI, non-ST-segment elevation myocardial infarction; PCI, percutaneous coronary intervention; TIA, transient ischemic attack (Newly defined in this revision: CI).

exhibiting significantly worse post-PCI physiological results (post-PCI FFR: 0.83 vs 0.90, $P<0.001$). Notably, nearly 30% of patients in the MACE group had persistently abnormal FFR (≤ 0.80) at the end of the procedure - approximately four times the rate in the non-MACE group. The clinical consequences were severe: in addition to 57 MACE events, the

MACE group had a high all-cause mortality rate (21.1%) and a striking incidence of stent thrombosis (14.0%), with no thrombotic events occurring in the non-MACE group.

Survival analysis revealed a clear gradient of risk across post-PCI FFR categories (**Figure 1**). Patients achieving post-PCI FFR ≥ 0.90 had

Post-PCI FFR in diffuse long coronary lesions

Table 2. Angiographic and procedural characteristics

Characteristic	All patients (n=327)	MACE group (n=57)	Non-MACE group (n=270)	χ^2/t	P
Target vessel				$\chi^2=3.05$	0.217
(1) Left anterior descending	178 (54.4)	34 (59.6)	144 (53.3)		
(2) Left circumflex artery	68 (20.8)	7 (12.3)	61 (22.6)		
(3) Right coronary artery	81 (24.8)	16 (28.1)	65 (24.1)		
QCA measurements					
Lesion length, mm	29.5 ± 10.2	34.8 ± 12.1	28.3 ± 9.4	t=4.50	<0.001
Reference vessel diameter, mm	2.79 ± 0.47	2.62 ± 0.43	2.83 ± 0.48	t=3.05	0.002
Minimum lumen diameter, mm	0.92 ± 0.38	0.84 ± 0.35	0.94 ± 0.39	t=1.79	0.063
Diameter stenosis, %	67.2 ± 12.3	69.8 ± 11.1	66.7 ± 12.5	t=1.73	0.092
Lesion characteristics					
Lesion classification				$\chi^2=4.99$	0.025
(1) Type B2	147 (45.0)	18 (31.6)	129 (47.8)		
(2) Type C	180 (55.0)	39 (68.4)	141 (52.2)		
Very long lesion (≥30 mm)	160 (48.9)	39 (68.4)	121 (44.8)	$\chi^2=10.50$	0.001
Small vessel (RVD <2.75 mm)	127 (38.8)	32 (56.1)	95 (35.2)	$\chi^2=8.70$	0.004
Calcification				$\chi^2=10.63$	0.005
(1) Absent/mild	148 (45.3)	16 (28.1)	132 (48.9)		
(2) Moderate	100 (30.6)	19 (33.3)	81 (30.0)		
(3) Severe	79 (24.2)	22 (38.6)	57 (21.1)		
Moderate/severe tortuosity	73 (22.3)	17 (29.8)	56 (20.7)	$\chi^2=2.24$	0.136
Bifurcation involvement	92 (28.1)	19 (33.3)	73 (27.0)	$\chi^2=0.92$	0.338
Diffuse disease index	0.38 ± 0.14	0.44 ± 0.15	0.37 ± 0.13	t=3.59	0.001
Procedural characteristics					
Pre-dilation	315 (96.3)	56 (98.2)	259 (95.9)	$\chi^2=0.72$	0.395
Specialized plaque modification	79 (24.2)	22 (38.6)	57 (21.1)	$\chi^2=7.85$	0.007
(1) Cutting/scoring balloon	51 (15.6)	14 (24.6)	37 (13.7)	$\chi^2=4.21$	0.046
(2) Rotational atherectomy	28 (8.6)	8 (14.0)	20 (7.4)	$\chi^2=2.64$	0.108
Total stent length, mm	38.2 ± 15.3	45.6 ± 17.8	36.6 ± 14.3	t=4.13	<0.001
Number of stents per lesion	1.53 ± 0.67	1.81 ± 0.75	1.47 ± 0.64	t=3.53	0.002
Average stent diameter, mm	2.94 ± 0.42	2.79 ± 0.38	2.97 ± 0.42	t=2.99	0.007
Stent type				$\chi^2=0.54$	0.763
(1) Everolimus-eluting	178 (54.4)	33 (57.9)	145 (53.7)		
(2) Zotarolimus-eluting	93 (28.4)	16 (28.1)	77 (28.5)		
(3) Sirolimus-eluting	56 (17.1)	8 (14.0)	48 (17.8)		
Post-dilatation	287 (87.8)	52 (91.2)	235 (87.0)	$\chi^2=0.77$	0.379
Maximum balloon pressure, atm	18.3 ± 3.6	18.9 ± 3.8	18.2 ± 3.5	t=1.35	0.214
Procedural success	317 (96.9)	54 (94.7)	263 (97.4)	$\chi^2=1.13$	0.274
Procedure time, min	68.4 ± 26.5	79.2 ± 30.7	66.0 ± 25.1	t=3.46	0.002
Contrast volume, mL	194.7 ± 72.3	218.2 ± 83.6	189.7 ± 69.0	t=2.73	0.014
Periprocedural complications	26 (8.0)	7 (12.3)	19 (7.0)	$\chi^2=1.77$	0.185

Values are mean ± standard deviation or n (%). Continuous variables were compared using Student's t-test (t value reported); categorical variables were compared using the χ^2 test (χ^2 value reported). MACE, major adverse cardiovascular events; MI, myocardial infarction; PCI, percutaneous coronary intervention; QCA, quantitative coronary angiography; RVD, reference vessel diameter (No further abbreviations require definition; all present abbreviations are now defined).

excellent event-free survival, with a MACE rate of only 6.4%, whereas those with suboptimal

results (<0.80) had a markedly higher event rate approaching 45% (log-rank P<0.001).

Post-PCI FFR in diffuse long coronary lesions

Table 3. FFR measurements and clinical outcomes

Variable	All patients (n=327)	MACE group (n=57)	Non-MACE group (n=270)	χ^2/t	P
FFR measurement					
Baseline FFR	0.68 ± 0.12	0.62 ± 0.13	0.69 ± 0.11	t=4.22	<0.001
Baseline FFR ≤0.60	87 (26.6)	25 (43.9)	62 (23.0)	$\chi^2=10.52$	0.001
Post-PCI FFR	0.89 ± 0.07	0.83 ± 0.08	0.90 ± 0.06	t=7.52	<0.001
Post-PCI FFR category				$\chi^2=35.28$	<0.001
(1) Optimal (≥0.90)	156 (47.7)	10 (17.5)	146 (54.1)		
(2) Intermediate (0.81-0.89)	133 (40.7)	30 (52.6)	103 (38.1)		
(3) Suboptimal (≤0.80)	38 (11.6)	17 (29.8)	21 (7.8)		
Absolute FFR improvement	0.21 ± 0.13	0.21 ± 0.14	0.21 ± 0.13	t=0.05	0.961
Clinical outcomes					
MACE	57 (17.4)	57 (100)	0 (0)	-†	-†
(1) Cardiac death	11 (3.4)	11 (19.3)	0 (0)	-†	-†
(2) Target vessel MI	19 (5.8)	19 (33.3)	0 (0)	-†	-†
(3) Target lesion revascularization	37 (11.3)	37 (64.9)	0 (0)	-†	-†
All-cause mortality	19 (5.8)	12 (21.1)	7 (2.6)	$\chi^2=29.31$	<0.001
Definite/probable stent thrombosis	8 (2.4)	8 (14.0)	0 (0)	-‡	<0.001‡
Non-target vessel revascularization	42 (12.8)	14 (24.6)	28 (10.4)	$\chi^2=8.47$	0.004
Medication adherence at last follow-up					
Dual antiplatelet therapy	282 (86.2)	50 (87.7)	232 (85.9)	$\chi^2=0.13$	0.723
Statins	308 (94.2)	53 (93.0)	255 (94.4)	$\chi^2=0.18$	0.668
Beta-blocker	229 (70.0)	43 (75.4)	186 (68.9)	$\chi^2=0.96$	0.326
ACEi/ARB	205 (62.7)	38 (66.7)	167 (61.9)	$\chi^2=0.47$	0.494

Values are mean ± standard deviation or n (%). Continuous variables were compared using Student's t-test (t value reported); categorical variables were compared using the χ^2 test (χ^2 value reported). †MACE and its individual components (cardiac death, target vessel MI, target lesion revascularization) define group allocation, so between-group testing is not applicable. ‡Compared by Fisher's exact test owing to zero events in the non-MACE group. ACEi, angiotensin-converting enzyme inhibitor; ARB, angiotensin receptor blocker; FFR, fractional flow reserve; MACE, major adverse cardiovascular events; MI, myocardial infarction; PCI, percutaneous coronary intervention (No further abbreviations require definition; all present abbreviations are now defined).

Although a moderate correlation existed between baseline and post-PCI FFR ($r=0.39$, $P<0.001$; **Figure 2**), severely depressed baseline FFR did not preclude achieving optimal post-PCI results, underscoring the importance of post-procedural physiological assessment.

Multivariate predictors of MACE

After multivariate adjustment (**Table 4**), Cox regression analysis identified five independent predictors of MACE. The strongest predictor was suboptimal post-PCI FFR <0.90 (HR, 2.68 95% CI, 1.78-4.02; $P<0.001$). Additional independent predictors included lesion length ≥ 30 mm (HR, 1.95; $P=0.002$), diabetes mellitus (HR, 1.84; $P=0.003$), severe calcification requiring plaque modification (HR, 1.78; $P=$

0.004), and baseline FFR ≤ 0.60 (HR, 1.64; $P=0.012$). Combining these five factors enabled robust risk stratification (**Figure 3**). Patients with four or five risk factors had a 2-year MACE rate of 42.3%, compared with only 5.9% in those with 0-1 risk factors (log-rank $P<0.001$). The model demonstrated good discrimination, with a C-statistic of 0.78.

Subgroup consistency

The protective effect of optimal post-PCI FFR (≥ 0.90) was consistent across all examined subgroups, including those defined by vessel size, diabetic status, and baseline ischemic severity (all interaction P -values >0.05). Even heavily calcified, very long lesions had favorable outcomes when optimal final hemodynamic results were achieved. Sensitivity analysis

Post-PCI FFR in diffuse long coronary lesions

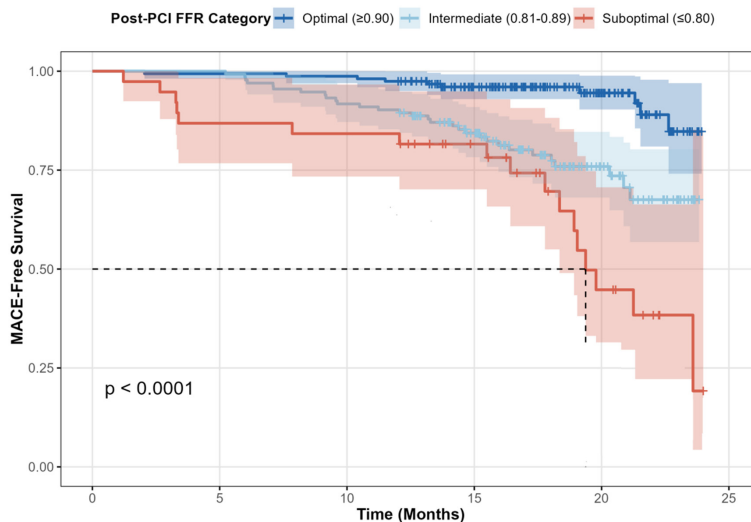


Figure 1. Cumulative incidence of major adverse cardiovascular events (MACE) stratified by post-percutaneous coronary intervention (PCI) fractional flow reserve (FFR) category. The vertical axis shows MACE-free survival and the horizontal axis shows follow-up time in months. Curves are shown for the optimal (FFR ≥ 0.90), intermediate (FFR 0.81-0.89), and suboptimal (FFR ≤ 0.80) categories, with between-group differences assessed by the log-rank test ($P < 0.0001$). Shaded bands represent 95% confidence intervals. FFR, fractional flow reserve; MACE, major adverse cardiovascular events; PCI, percutaneous coronary intervention.

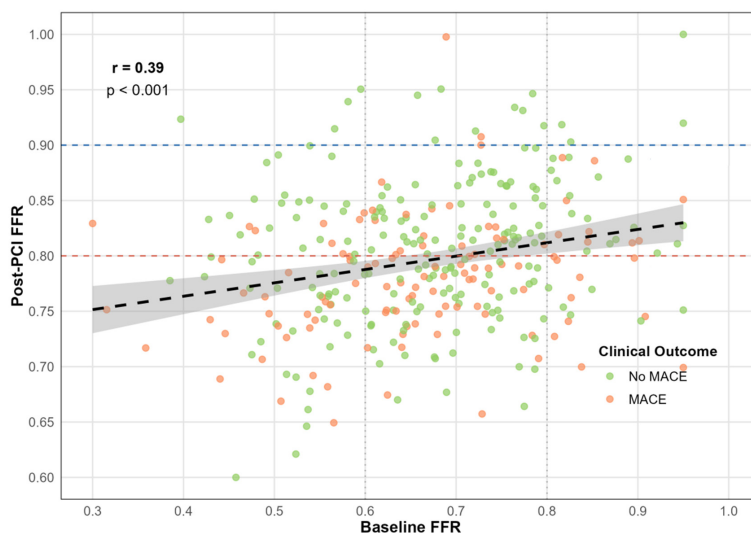


Figure 2. Scatter plot illustrating the relationship between baseline fractional flow reserve (FFR) and post-percutaneous coronary intervention (PCI) FFR. Each point represents an individual patient, colored by clinical outcome (major adverse cardiovascular events [MACE] versus no MACE). The dashed regression line indicates the correlation between baseline and post-PCI FFR ($r = 0.39$, $P < 0.001$), with the shaded band representing the 95% confidence interval; horizontal reference lines mark the post-PCI FFR thresholds of 0.80 and 0.90. FFR, fractional flow reserve; MACE, major adverse cardiovascular events; PCI, percutaneous coronary intervention.

excluding 12 patients lost to follow-up did not alter the principal findings.

Discussion

This retrospective analysis identified five independent predictors of clinical outcomes after FFR-guided PCI in diffuse long coronary lesions: suboptimal post-PCI FFR (< 0.90), lesion length ≥ 30 mm, diabetes mellitus, severe calcification, and baseline FFR ≤ 0.60 . Most importantly, achieving post-PCI FFR ≥ 0.90 was associated with a 63% relative reduction in MACE, independent of baseline anatomical and clinical features.

Management of diffuse coronary disease has historically been challenging for the interventional community. Unlike focal stenoses, diffuse lesions exhibit complex morphology and extensive atherosclerotic burden, which contribute to higher complication rates and inferior long-term outcomes [13]. Prior registries have identified diabetes, lesion length, and calcification as principal drivers of stent failure [14-16]. Our findings are consistent with these observations and extend the current paradigm by integrating these static markers with real-time intracoronary physiology.

The most striking finding in our cohort is the strong predictive value of post-PCI FFR. While the FAME-era literature established the value of physiological assessment for guiding the decision to stent, the prognostic significance of post-stenting FFR - particularly in diffuse disease - has remained underexplored. Our analysis demonstrates that having a post-PCI FFR ≤ 0.80 at the end of the procedure

confers a markedly increased risk of MACE compared with optimized outcomes. Even the

Table 4. Univariate and multivariate Cox regression analysis for predictors of MACE

Variable	Univariate HR (95% CI)	P	Multivariate HR (95% CI)	P
Age ≥ 75 years	1.42 (0.93-2.17)	0.108	-	--
Male	0.89 (0.62-1.29)	0.543	-	-
Diabetes mellitus	2.21 (1.56-3.14)	<0.001	1.84 (1.26-2.67)	0.003
Chronic kidney disease	1.73 (1.14-2.63)	0.010	1.38 (0.89-2.15)	0.153
Previous MI	1.45 (0.95-2.22)	0.087	1.24 (0.79-1.94)	0.342
Clinical presentation (ACS vs stable)	1.22 (0.86-1.74)	0.265	-	-
hs-CRP >3 mg/L	1.86 (1.29-2.67)	0.001	1.34 (0.91-1.97)	0.138
Lesion length ≥ 30 mm	2.37 (1.64-3.41)	<0.001	1.95 (1.32-2.89)	0.002
Reference vessel diameter <2.75 mm	1.98 (1.39-2.82)	<0.001	1.42 (0.97-2.08)	0.070
Severe calcification	2.12 (1.46-3.08)	<0.001	1.41 (0.94-2.12)	0.101
Severe calcification requiring plaque modification	2.15 (1.49-3.09)	<0.001	1.78 (1.21-2.63)	0.004
Bifurcation involvement	1.31 (0.89-1.92)	0.168	-	-
Diffuse disease index >0.40	1.72 (1.20-2.46)	0.003	1.28 (0.87-1.89)	0.205
Total stent length >40 mm	1.93 (1.36-2.75)	<0.001	1.32 (0.89-1.97)	0.168
Number of stents ≥ 2	1.67 (1.17-2.39)	0.005	1.22 (0.82-1.81)	0.335
Post-PCI FFR <0.90	3.22 (2.19-4.75)	<0.001	2.68 (1.78-4.02)	<0.001
Baseline FFR ≤ 0.60	2.12 (1.48-3.04)	<0.001	1.64 (1.12-2.41)	0.012
Absolute FFR improvement <0.20	1.19 (0.83-1.71)	0.342	-	-
Statin use at discharge	0.79 (0.38-1.64)	0.526	-	-

ACS, acute coronary syndrome; CI, confidence interval; FFR, fractional flow reserve; HR, hazard ratio; hs-CRP, high-sensitivity C-reactive protein; MACE, major adverse cardiovascular events; MI, myocardial infarction; PCI, percutaneous coronary intervention (No further abbreviations require definition; all present abbreviations are now defined).

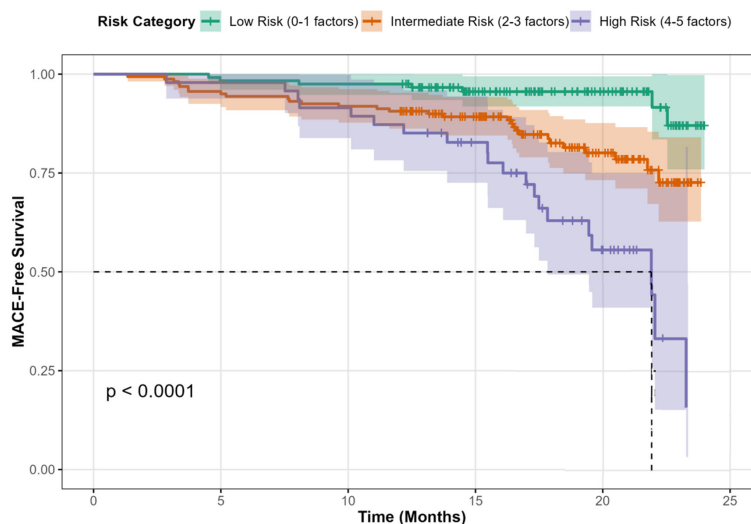


Figure 3. Kaplan-Meier curves for the multivariable risk stratification model. The vertical axis shows major adverse cardiovascular events (MACE)-free survival and the horizontal axis shows follow-up time in months. Patients are stratified into low-risk (0-1 factors), intermediate-risk (2-3 factors), and high-risk (4-5 factors) categories according to the number of independent predictors present, with between-group differences assessed by the log-rank test ($P < 0.0001$). Shaded bands represent 95% confidence intervals. MACE, major adverse cardiovascular events.

intermediate range (0.81-0.89), often regarded as acceptable, carried a significantly elevated risk. This finding suggests that the conventional ischemic threshold of 0.80 is insufficient as a procedural target in diffuse disease. These observations align with data from the FFR-SEARCH registry, which similarly identified post-PCI FFR as the principal determinant of target vessel failure [17], and with meta-analytic evidence that post-procedural functional indices outperform conventional angiographic endpoints in predicting outcomes [18]. Our study uniquely contextualizes these findings within the challenging setting of diffuse long lesions. In such lesions, angiographically satisfactory re-

sults may mask subtle mechanical issues, such as suboptimal stent expansion, edge dissection, or unresolved microvascular dysfunction, that limit final blood flow.

These findings support a reactive, physiology-guided optimization strategy as the standard of care for extensive revascularization. When post-PCI FFR remains suboptimal, operators should systematically address the underlying physiological barrier using high-pressure post-dilation, additional stenting of tandem lesions, and intravascular imaging to identify mechanical abnormalities. Whether a protocol-based application of these optimization steps improves outcomes warrants confirmation in randomized trials, but our retrospective findings strongly support this hypothesis.

Lesion length itself was an independent predictor of adverse events. Lesions exceeding 30 mm necessitate overlapping stents [19], increasing the risk of geographic miss, limiting uniform drug elution, and promoting excessive neointimal hyperplasia at stent edges. Importantly, lesion length remained predictive in our Cox model even after adjustment for total stent length, suggesting that the underlying biological aggressiveness of diffuse plaques - not merely mechanical stent burden - contributes to adverse outcomes.

Diabetes mellitus further amplified atherosclerotic risk, with an 84% increase in MACE rate among diabetic patients in our cohort. The diabetic state, characterized by endothelial dysfunction, chronic inflammation, and hypercoagulability, impairs vascular healing after stenting [20]. Disease progression in these patients often outpaces the antiproliferative capacity of contemporary drug-eluting stents. Therefore, in diabetic patients undergoing intervention for diffuse disease, procedural success must be complemented by rigorous secondary prevention, including aggressive lipid-lowering and glycemic control.

Severe coronary calcification acted as a structural barrier to optimal stenting. Lesions requiring rotational atherectomy or specialized cutting balloons were independently associated with adverse outcomes [21]. Calcification limits stent expansion, leading to malapposition and altered shear stress, which predispose to thrombosis and restenosis. Our results suggest

that even when contemporary plaque modification tools successfully facilitate stent deployment, the biological signature of severe calcification - likely reflecting an advanced necrotic core - continues to influence long-term risk.

Finally, the baseline physiological state was prognostically informative. Severely depressed baseline FFR (<0.60) consistently predicted late events independent of angiographic severity [22]. In diffuse disease, such profound pressure decline reflects not only focal narrowing but also extensive longitudinal disease burden and a large ischemic myocardial territory, suggesting a persistent biological risk even after mechanical revascularization.

Combining these five variables produced a clinically useful predictive matrix. Patients with four or five risk factors had a greater than 40% probability of recurrent ischemic events within two years, warranting intensive surveillance and prolonged dual antiplatelet therapy. Conversely, patients with optimal hemodynamics and limited clinical risk factors can reasonably be managed with standard maintenance protocols.

Several limitations of this work should be acknowledged. As a single-center retrospective observational study, our findings may be subject to selection bias and unmeasured confounding. Intravascular imaging was used selectively rather than systematically, limiting our ability to correlate physiological gradients with specific structural abnormalities. Non-hyperemic resting indices (iFR, RFR) were not systematically recorded. The 24-month follow-up may underestimate the late restenosis characteristic of diffuse disease. Despite these limitations, the strong prognostic value of post-PCI hemodynamics is compelling, supporting a paradigm shift from angiography-guided to physiology-guided treatment. Forthcoming platforms such as the FFR-REACT trial [23] are expected to validate this optimization paradigm, while emerging wire-free physiological assessment modalities (quantitative flow ratio, vessel FFR) may broaden access to these techniques [24].

Conclusion

Treatment of diffuse long coronary lesions remains a complex clinical and technical chal-

lenge. This study identifies five prognostic factors that determine clinical outcomes after FFR-guided PCI: post-PCI FFR, lesion length, baseline ischemic severity, severe calcification, and diabetes mellitus. Among these, post-PCI FFR is the most critical determinant. Achieving post-PCI FFR ≥ 0.90 substantially reduces the risk of subsequent cardiovascular events, supporting a shift from angiography-guided to physiology-guided optimization in the management of diffuse coronary disease.

Disclosure of conflict of interest

None.

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