

Optimizing Revascularization Outcomes in Chronic Coronary Syndromes

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Abstract

The value of coronary revascularization in Chronic Coronary Syndromes (CCS) depends on whether it delivers the benefit that justified intervention. Anatomical, physiological, and plaque assessment inform patient selection, but each answers a different clinical question. Applying evolving trial evidence requires attention to the populations studied, outcomes measured, and durability of treatment. Procedural optimization and complete revascularization require the same clinical judgment, with imaging and devices serving defined treatment goals. Medical therapy remains essential regardless of the revascularization strategy. Follow-up connects the procedure to sustained prevention, rehabilitation, and assessment of symptoms and clinical events. Reviewing these outcomes against the original indication, together with the patient's priorities, provides a practical basis for improving care.

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Introduction

Technical success is important, but the value of coronary revascularization lies in its benefit to the patient. In Chronic Coronary Syndromes (CCS), this may mean relief of limiting angina or improved prognosis in an appropriate clinical setting.^{1,2} For Percutaneous Coronary Intervention (PCI) and surgery alike, patient selection, procedural quality, and sustained care must remain aligned with that goal (Figure 1). Evolving evidence and patient priorities guide decisions throughout this process.

Patient Selection and Treatment Planning

The first decision is what revascularization is expected to achieve. Extended follow-up of the ISCHEMIA trial found no significant all-cause mortality advantage with an initial invasive strategy over conservative management at a median of 5.7 years.³ Significant Left Main (LM) disease and Left Ventricular Ejection Fraction (LVEF) <35% were excluded; these findings cannot settle the prognostic question in such patients. Significant LM disease, extensive multivessel disease, and severe ventricular dysfunction require separate consideration.^{1,2} Symptom relief is a distinct benefit, supported by placebo-controlled PCI evidence in patients receiving little or no antianginal therapy and the small ORBITA-CTO trial in symptomatic single-vessel chronic total occlusion.^{4,5} These are reasons to define the treatment goal, with medical therapy continuing whichever strategy is chosen.¹

Anatomical assessment establishes stenosis severity, distribution, and complexity. When appropriate, Coronary Computed Tomography Angiography (CCTA) can exclude obstructive disease or define its extent, identifying high-risk anatomy for invasive assessment.¹ Angiographic diameter stenoses $\geq 70\%$ in non-LM arteries or $\geq 50\%$ in the LM are conventionally considered significant, although anatomy alone does not establish a PCI indication.²

For intermediate stenoses, physiology connects anatomical findings to the decision to intervene. Wire-based hyperemic and nonhyperemic assessment provides established thresholds, including Fractional Flow Reserve (FFR) ≤ 0.80 and instantaneous wave-free ratio ≤ 0.89 .¹ Angiography-derived approaches offer alternatives, although randomized noninferiority findings remain specific to the platforms tested and their one-year composite endpoints.⁶⁻⁷ CT-derived FFR can clarify selected stenoses, with interpretation limited by image quality, borderline values, and diffuse disease.⁸ The distribution of disease also shapes expectations: observational Pullback Pressure Gradient (PPG) data associate focal disease with less residual angina after PCI.⁹ Recognizing diffuse disease helps explain why treating a discrete stenosis may leave symptoms unresolved.

Plaque characterization broadens risk assessment beyond ischemia, without by itself establishing a PCI indication. CCTA identifies low-attenuation plaque and positive remodeling; Intravascular Ultrasound (IVUS), Optical Coherence Tomography

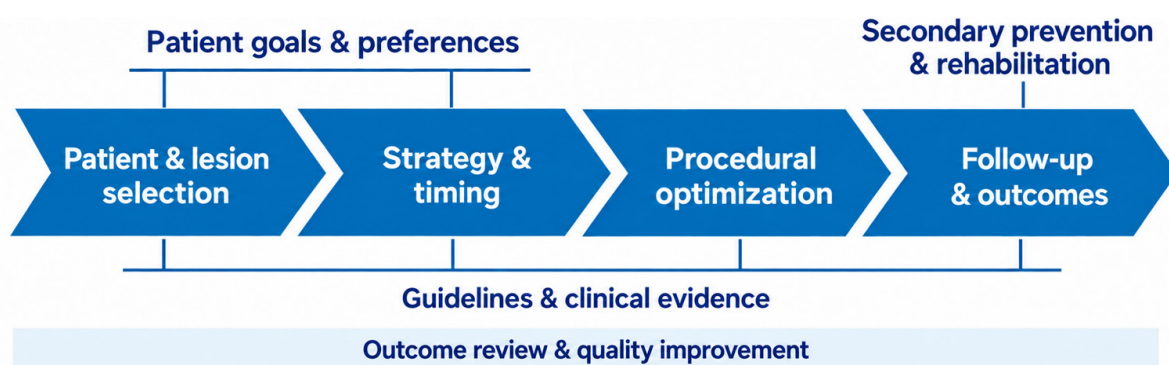


Figure 1. A framework for optimizing revascularization outcomes in chronic coronary syndromes. Planning integrates patient and lesion selection with decisions on strategy and timing. Procedural optimization is followed by assessment of recovery and long-term outcomes. Patient goals and preferences inform treatment decisions, while guidelines and clinical evidence inform all stages. Secondary prevention and rehabilitation support continuing care. Outcome review supports quality improvement across the entire process. The framework applies to PCI and CABG. PCI, percutaneous coronary intervention; CABG, coronary artery bypass grafting.

(OCT), and Near-Infrared Spectroscopy (NIRS) add information on plaque burden, Thin-Cap Fibroatheroma (TCFA), and lipid content.^{1,10-11} A maximum lipid core burden index within a 4-mm segment (maxLCBI4mm) >400 has been associated with subsequent nonculprit events.¹¹ Whether treating such plaques prevents clinical events remains unsettled. PREVENT reduced a two-year composite including revascularization and angina hospitalization by adding PCI to medical therapy for selected, imaging-defined non-flow-limiting plaques, without establishing reduced death or Myocardial Infarction (MI).¹⁰ The smaller PROSPECT ABSORB trial improved lumen dimensions but did not demonstrate fewer clinical events and was not powered for clinical efficacy.¹² Plaque findings can strengthen preventive care; routine preventive PCI still requires stronger randomized evidence.

When revascularization is indicated, the choice between PCI and Coronary Artery Bypass Grafting (CABG) depends on anatomical complexity, ventricular function, diabetes, frailty, and the feasibility of complete revascularization. Guidelines favor CABG in suitable surgical candidates with diabetes and multivessel disease involving the Left Anterior Descending (LAD) artery.² Longer follow-up helps refine the discussion. In three-vessel disease without LM involvement, FAME 3 found no significant five-year difference in the death, stroke, or MI composite, but more MI and repeat revascularization after PCI.¹³ NOBLE found no significant ten-year all-cause mortality difference for unprotected LM disease without additional complex lesions; nonfatal events were not assessed beyond five years.¹⁴ These results do not make the strategies interchangeable. Heart Team discussion needs to place prognosis alongside recovery, procedural risk, and the likelihood of further treatment. Timing in CCS allows for this discussion and medical optimization, while remaining responsive to symptoms and risk.¹⁻²

Procedural Quality and Optimization

Once PCI is chosen, imaging is valuable when it changes how the procedure is performed and confirms the result. CCTA can support planning; IVUS or OCT guides lesion preparation, sizing, and assessment of expansion and complications.¹ Intravascular imaging guidance reduced five-year target-vessel failure in complex PCI, whereas separate LM and complex high-risk trials did

not demonstrate superiority for their primary endpoints.¹⁵⁻¹⁷ A network meta-analysis combining randomized and observational studies associated intravascular imaging guidance with fewer major adverse cardiovascular events and stent thromboses in calcified lesions.¹⁸ Differences in populations, protocols, and follow-up limit comparisons. The guideline recommendation for intravascular imaging in complex PCI therefore needs to be applied through explicit optimization targets.¹

The 2026 European LM consensus illustrates how such targets depend on anatomy and technique. Suggested Minimum Stent Area (MSA) ranges are 10–12 mm² in the LM and 7–8 mm² at the LAD ostium for provisional or two-stent PCI. At the circumflex ostium, the benchmark is a Minimum Lumen Area (MLA) ≥4 mm² after provisional crossover, versus an MSA of 6–7 mm² with two stents.¹⁹ These values complement assessment of vessel dimensions, relative expansion, edge injury, malapposition, and stent deformation. Pursuing an absolute area without considering vessel size risks turning an optimization target into a reason for overexpansion.¹⁹

Contemporary Drug-Eluting Stent (DES) remains the reference treatment for de novo PCI.²⁰ Drug-Coated Balloons (DCBs) offer an option for selected in-stent restenosis,¹ while their broader use in de novo disease depends on successful lesion preparation and readiness for provisional stenting when needed.²⁰⁻²¹ The trade-off is durability: a sirolimus-balloon strategy met one-year noninferiority for target-vessel failure in the intention-to-treat analysis, not per protocol; both this and a paclitaxel-balloon strategy resulted in more repeat revascularization than DES.²⁰⁻²¹ Indonesian observational studies reported encouraging one-year outcomes in selected patients after DCB-only angioplasty in vessels ≥3.0 mm and completed hybrid DES–DCB procedures for true distal LM bifurcations.²²⁻²³ These experiences merit larger prospective comparative studies. Bioresorbable scaffolds offer temporary support, but their clinical role remains less established: IRONMAN II demonstrated noninferiority for two-year in-segment late lumen loss, not clinical equivalence.²⁴ Choosing among these approaches requires matching the need for scaffolding and the result of lesion preparation to the strength of evidence for a durable outcome.

Complete revascularization requires a plan for the disease left untreated, whether the procedure is

PCI or CABG. That plan balances residual disease against procedural burden and, for surgery, graft targets and durability.¹ After PCI, residual ischemia helps identify clinically important remaining targets. A prespecified FAME 3 analysis associated a residual functional SYNTAX score >8 after PCI with more five-year death, MI, or stroke than complete CABG, although the subgroup comparisons were not randomized.²⁵ Physiology-guided target selection is less established for CABG.² The practical implication is to document treated lesions, deliberately deferred disease, and targets reserved for staging, rather than equating a successful procedure with completion of treatment.

Follow-up and Long-Term Outcomes

Follow-up begins with completing the agreed plan. For staged PCI in CCS, physiology can guide treatment or deferral of residual intermediate lesions, while symptoms, renal function, and procedural burden inform timing.¹ Remaining targets, the proposed schedule, and responsibility for reassessment need to be clear. The postinfarction evidence addresses a different setting: the European Society of Cardiology (ESC) guidance recommends complete revascularization during index PCI or within 45 days in hemodynamically stable patients with ST-segment elevation MI and multivessel disease undergoing primary PCI.²⁶ That window cannot be transferred automatically to elective CCS practice. After PCI or CABG, reassessment returns to whether further treatment is justified by residual disease, recovery, and the original goals.¹

Sustaining benefit also means closing the gap between preventive targets and everyday treatment. After revascularization, Low-Density Lipoprotein Cholesterol (LDL-C) should be <55 mg/dL with a ≥50% reduction from baseline; <40 mg/dL may be considered for recurrent vascular events despite maximally tolerated statin-based therapy or for polyvascular disease.^{1,27} In the seven-center Indonesia-INTERASPIRE survey, only 8.5% of 402 participants achieved LDL-C <55 mg/dL.²⁸ This is not a national estimate, but it brings access, tolerability, and adherence into the same discussion as treatment intensity.

For escalation of lipid-lowering therapy, clinical outcomes and plaque changes provide different kinds of evidence. In a prespecified VESALIUS-CV analysis of stable patients with prior PCI, LDL-C ≥90 mg/dL, and no previous MI or stroke,

evolocumab reduced the composite of coronary heart disease death, MI, or ischemic stroke.²⁹ A recent NIRS meta-analysis found a numerical reduction in maxLCBI4mm with Proprotein Convertase Subtilisin/Kexin type 9 (PCSK9) inhibitor-based regimens, but neither this subgroup result nor the overall pooled effect was statistically significant.³⁰ Treatment intensity remains guided by LDL-C and clinical risk.^{1,27}

Secondary prevention also requires blood-pressure and diabetes control, smoking cessation, and antithrombotic therapy that balances ischemic protection against bleeding.¹⁻² Maintenance treatment deserves periodic reconsideration. In HOST-EXAM, patients event-free during 6–18 months of dual antiplatelet therapy had better net clinical outcomes at ten years with clopidogrel than aspirin, without a significant all-cause mortality difference.³¹ This supports considering clopidogrel for maintenance monotherapy after PCI, while reviewing the regimen as bleeding risk and tolerability change.¹⁻²

Rehabilitation connects prevention with recovery. The 2026 ESC Guidelines bring exercise, education, psychosocial support, and preventive care together in comprehensive cardiac rehabilitation.³² Referral is more meaningful when barriers to participation are addressed and the program fits the patient's capabilities. Persistent angina also deserves explanation: residual epicardial disease, microvascular dysfunction, and vasomotor disorders may require different responses.¹ Symptoms, functional capacity, and quality of life therefore belong alongside clinical events and repeat revascularization in the assessment of benefit.

These outcomes provide a basis for service improvement through registries and risk-adjusted review.² In Indonesia, regional disparities in catheterization access make coordination between referring clinicians and treating centers particularly important.³³ Recording treatment goals, completion of the agreed plan, preventive therapy, and patient-reported recovery allows services to examine where an appropriate indication failed to translate into benefit. Procedure volume alone cannot answer that question.

Conclusion

In CCS, well-informed revascularization decisions connect an appropriate indication with careful execution and sustained care. New trials refine those decisions by clarifying which patients

benefit, from which strategy, and over what period. The enduring measure of quality is whether patients receive the benefit that justified intervention.

List of Abbreviations

CABG	Coronary Artery Bypass Grafting
CCS	Chronic Coronary Syndromes
CCTA	Coronary Computed Tomography Angiography
DCBs	Drug-Coated Balloons
DES	Drug-Eluting Stent
ESC	European Society of Cardiology
FFR	Fractional Flow Reserve
IVUS	Intravascular Ultrasound
LAD	Left Anterior Descending
LDL-C	Low-Density Lipoprotein Cholesterol
LM	Left Main
LVEF	Left Ventricular Ejection Fraction
maxLCBI4mm	maximum lipid core burden index within a 4-mm segment
MI	Myocardial Infarction
MLA	Minimum Lumen Area
MSA	Minimum Stent Area
NIRS	Near-Infrared Spectroscopy
OCT	Optical Coherence Tomography
PCI	Percutaneous Coronary Intervention
PCSK9	Proprotein Convertase Subtilisin/Kexin type 9
PPG	Pullback Pressure Gradient
TCFA	Thin-Cap Fibroatheroma

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