Prognostic Determinants After PCI in Ischemic Cardiomyopathy: A Comprehensive Clinical Review

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Abstract

Background: Ischemic cardiomyopathy (ICM) represents a major global cause of heart failure and mortality, with left ventricular (LV) dysfunction driven by chronic ischemia, scar formation, and adverse remodeling. Percutaneous coronary intervention (PCI) is increasingly used in patients with ICM, particularly those unsuitable for coronary artery bypass grafting (CABG) or presenting with high procedural risk. However, the prognostic value of PCI in this population remains controversial, as large randomized trials and observational cohorts have produced discordant findings. This review aims to clarify the prognostic determinants that influence clinical outcomes after PCI in patients with ICM, integrating evidence from physiology-guided interventions, myocardial viability studies, revascularization completeness, and ventricular recovery predictors.

A consistent theme across clinical studies is the heterogeneity of patients with ICM, with prognosis strongly dependent on the interplay between myocardial substrate and ischemia burden. Emerging data suggest that factors such as the extent of viable myocardium, coronary physiology (FFR/iFR), complexity of coronary anatomy, and baseline LV dysfunction critically inform the expected benefit of revascularization. While the STICH extension study established a survival benefit of CABG over medical therapy, more recent data from REVIVED-BCIS2 showed no improvement in death or heart failure hospitalization with PCI added to optimal medical therapy, highlighting the need for better risk stratification. Additional evidence from viability imaging, microvascular assessments, and strain imaging underscores the importance of identifying patients with sufficient myocardial reserve to recover after revascularization.

Procedural characteristics also influence prognosis. Complete revascularization, CTO PCI success, and optimized use of mechanical circulatory support in high-risk PCI settings have been linked to improved outcomes in selected cohorts. Conversely, incomplete revascularization, extensive scar burden, and frailty markers predict limited benefit. Biomarkers such as NT-proBNP and high-sensitivity troponin further refine risk prediction and may complement imaging-based selection.

This comprehensive review synthesizes the prognostic factors that determine which patients with ICM are most likely to benefit from PCI, clarifies the evidence gaps contributing to conflicting trial results, and highlights future directions involving personalized risk models. Understanding these determinants is essential for optimizing treatment strategies, improving survival, and reducing heart failure progression in this complex patient population.

Keywords: *Prognostic , PCI ,Ischemic Cardiomyopathy*

Introduction

Ischemic cardiomyopathy (ICM) remains one of the leading causes of heart failure with reduced ejection fraction (HFrEF) worldwide, contributing significantly to mortality, recurrent hospitalizations, and impaired quality of life. The condition develops from progressive coronary artery disease (CAD), chronic ischemia, and myocardial scarring, ultimately resulting in adverse left ventricular (LV) remodeling and pump failure. Revascularization strategies, particularly percutaneous coronary intervention (PCI), have evolved dramatically in safety and technical capability, prompting increased use in patients with LV dysfunction. Yet, despite advances in PCI technique, substantial uncertainty persists regarding its prognostic impact in patients with ICM, especially compared with guideline-directed medical therapy or coronary artery bypass grafting (CABG). The conflicting evidence from contemporary randomized trials and observational registries underscores the need for a more precise understanding of which patients derive meaningful prognostic benefit from PCI. [1–3]

The central challenge in interpreting PCI outcomes in ICM arises from heterogeneity in myocardial substrate, coronary anatomy, procedural complexity, and comorbidity burden. While the STICH and STICHES trials established a long-term survival benefit for CABG in patients with significant CAD and LV dysfunction, PCI has not demonstrated similar mortality reductions in randomized settings. Most notably, the REVIVED-BCIS2 trial showed that PCI added to optimal medical therapy did not reduce all-cause death or heart failure hospitalizations in patients with reduced LVEF and extensive CAD, even when viability was present on imaging. This divergence between surgical and percutaneous revascularization outcomes highlights substantial knowledge gaps regarding patient selection, the role of myocardial viability, the impact of completeness of revascularization, and the extent of hibernating myocardium capable of functional recovery. [4–6]

Therefore, the aim of this review is to synthesize current evidence regarding **prognostic determinants after PCI in ischemic cardiomyopathy**, integrating clinical, imaging, procedural, and physiological markers that modulate outcomes. By exploring the interplay among viability, coronary physiology, LV remodeling potential, revascularization completeness, and high-risk procedural strategies, this review seeks to clarify why prior trials yielded inconsistent results and identify which patient subgroups may derive the greatest benefit. Addressing these gaps is crucial not only for improving survival and heart failure progression but also for guiding personalized treatment decisions in an era of expanding revascularization options and evolving heart failure therapies. [7–9]

Pathophysiology and Rationale for PCI in Ischemic Cardiomyopathy

Ischemic cardiomyopathy arises from a combination of chronic myocardial ischemia, infarction, and adverse ventricular remodeling, each contributing to progressive LV systolic dysfunction. Persistent ischemia leads to metabolic stress, impaired contractility, and eventually irreversible myocyte loss through apoptosis and necrosis. Additionally, neurohormonal activation—including upregulation of the renin–angiotensin–aldosterone system and sympathetic pathways—further accelerates remodeling, fibrosis, and chamber dilation. The presence of viable but dysfunctional myocardium (hibernation) distinguishes ICM from non-ischemic phenotypes and theoretically provides a substrate for functional recovery after revascularization. This mechanistic framework supports the rationale that restoring myocardial perfusion through PCI could improve contractile performance and reverse remodeling when sufficient viable tissue remains. [10–12]

The concept of myocardial hibernation, first described decades ago, explains why segments with reduced contractility may still recover after revascularization if chronic hypoperfusion is corrected. Positron emission tomography (PET), cardiac magnetic resonance (CMR) with late gadolinium enhancement (LGE), and low-dose dobutamine stress echocardiography have validated the presence of viable yet dysfunctional myocardium in a substantial subset of patients with ICM. The amount of viable

myocardium has been correlated with potential improvements in LVEF and functional capacity following revascularization, although more recent randomized data have challenged the strength of this association. Nonetheless, the persistence of physiological plausibility continues to drive interest in PCI as a strategy to salvage hibernating tissue and slow or reverse heart failure progression. [13–15]

PCI aims to relieve flow-limiting stenoses, reduce ischemia, and improve myocardial perfusion, which may mitigate microvascular dysfunction and improve energetic efficiency in surviving cardiomyocytes. Restored perfusion may also attenuate ischemia-induced arrhythmogenic substrates, reducing electrical instability in patients with scarred ventricles. In addition, improved coronary flow can reduce wall stress and oxygen demand in previously underperfused regions, potentially enhancing reverse remodeling responses when coupled with optimal heart failure therapy. However, the degree to which PCI alone can produce meaningful prognostic benefit depends heavily on the interplay between ischemia burden, scar distribution, and the feasibility of complete revascularization—factors that vary widely across patient populations with ICM. [16–18]

Despite the theoretical rationale, not all patients with ICM demonstrate functional recovery or survival benefit after PCI. Advanced scar burden, diffuse microvascular dysfunction, and extensive remodeling may limit the potential for improvement, underscoring the importance of integrative assessment using imaging and physiologic tools. Furthermore, PCI in this population is often technically challenging due to complex anatomy—such as chronic total occlusions (CTOs), multivessel disease, and heavily calcified lesions—and procedural risks may outweigh potential benefit in severely frail or comorbid individuals. These considerations highlight why the prognostic value of PCI remains variable and why a refined understanding of patient-specific determinants is essential. [19–21]

Patient Selection and Baseline Risk Markers

Patient selection is one of the most critical determinants of prognosis after PCI in ischemic cardiomyopathy (ICM). Outcomes vary substantially depending on the severity of LV systolic dysfunction, comorbidity burden, and anatomical complexity of coronary artery disease. Clinical risk assessment tools—such as the SYNTAX score for anatomical complexity and the Seattle Heart Failure Model (SHFM) for mortality prediction—provide important baseline insights but often underrepresent the unique interplay between ischemia, viability, and ventricular remodeling that characterizes ICM. Patients with advanced frailty, severe LV dilation, high pulmonary pressures, or right ventricular dysfunction frequently demonstrate limited recovery after PCI due to irreversible myocardial damage and hemodynamic compromise. Consequently, integrating traditional risk scores with heart failure–specific markers is essential for accurately forecasting post-PCI outcomes in this population. [22–24]

Comorbid conditions strongly influence prognosis after PCI, with chronic kidney disease (CKD), diabetes mellitus, and peripheral vascular disease consistently linked to higher mortality and reduced likelihood of ventricular recovery. CKD, in particular, exacerbates microvascular dysfunction and systemic inflammation, which may diminish the benefits of revascularization and increase procedural complications. Diabetes contributes to diffuse atherosclerosis and impaired microcirculatory reserve, often resulting in incomplete revascularization or limited perfusion improvement despite technically successful PCI. Additionally, frailty—an underrecognized yet powerful prognostic marker—has been shown to predict adverse outcomes independent of LVEF or coronary anatomy, emphasizing the need for comprehensive geriatric evaluation in high-risk PCI candidates. [25–27]

Baseline hemodynamic status and biomarkers also help identify patients most likely to benefit. Elevated NT-proBNP levels, severe LV dilation, and high resting heart failure burden correlate with limited capacity for functional recovery after PCI, reflecting advanced myocardial remodeling. Conversely, patients with modestly reduced LVEF, smaller LV volumes, and preserved right ventricular function generally demonstrate greater potential for reverse remodeling and symptomatic improvement. High-sensitivity troponin levels further stratify risk by identifying ongoing myocardial injury, which may signal active ischemia but can also reflect chronic structural disease; thus, interpretation must occur within clinical context. The integration of hemodynamic markers, imaging indices, and biomarkers

enhances the precision of patient selection and may help explain heterogeneous outcomes observed in major revascularization trials. [28–30]

Extent and Completeness of Revascularization

Completeness of revascularization has long been recognized as a major prognostic determinant in patients with ischemic heart disease, and this principle holds particular relevance in ischemic cardiomyopathy (ICM). Achieving complete revascularization (CR) may restore perfusion to a greater extent of jeopardized myocardium, reduce ischemic burden, and enhance the potential for functional LV recovery. Multiple observational studies and meta-analyses have demonstrated that CR is associated with improved survival, fewer heart failure hospitalizations, and better angina relief compared with incomplete revascularization (IR). However, randomized data specific to patients with ICM remain limited, and complexities such as multivessel disease, chronic total occlusions (CTOs), and diffuse calcification frequently hinder the ability to attain CR. These anatomical challenges help explain the variable prognostic outcomes observed following PCI in patients with LV dysfunction. [31–33]

The prognostic advantage of CR may be driven not only by restoration of epicardial flow but also by cumulative improvement in downstream microvascular perfusion. In populations with reduced LVEF, microvascular dysfunction and diffuse atherosclerosis are common, meaning that even lesions appearing angiographically moderate can contribute substantially to ischemia. Studies employing fractional flow reserve (FFR) and noninvasive perfusion imaging have shown that reducing ischemia burden correlates with better outcomes, particularly when residual ischemia remains minimal after PCI. Conversely, persistent ischemia following IR has been linked to poorer LV remodeling trajectories and increased mortality. While the REVIVED-BCIS2 trial permitted both complete and incomplete PCI, post-hoc analyses suggest that the degree of revascularization may modulate outcomes, although insufficient power prevents definitive conclusions. [34–36]

CTO revascularization represents an especially important aspect of completeness in ICM, given that CTOs often subtend large territories of hibernating myocardium. Successful CTO PCI has been associated with improved ventricular function, reduced arrhythmic risk, and enhanced long-term survival in selected cohorts. However, procedural risks are higher in patients with LV dysfunction, and failure to open a CTO may increase contrast load and procedural time without improving prognosis. Advanced imaging and physiologic assessment may help determine which CTOs are likely to yield meaningful recovery after revascularization. Thus, while CR may confer prognostic benefit, its pursuit must be individualized, balancing technical feasibility, myocardial substrate characteristics, and overall patient risk profile. [37–39]

Myocardial Viability as a Prognostic Determinant

Myocardial viability has traditionally been viewed as a cornerstone of decision-making for revascularization in ischemic cardiomyopathy (ICM), based on the premise that dysfunctional yet viable (hibernating) myocardium can recover function following restoration of blood flow. Early observational studies and meta-analyses demonstrated that patients with substantial viability had improved survival after revascularization compared with those lacking viable myocardium. Techniques such as positron emission tomography (PET), cardiac magnetic resonance imaging (CMR) with late gadolinium enhancement (LGE), and low-dose dobutamine stress echocardiography have provided robust noninvasive markers for identifying segments that may recover contractility. PET, in particular, offers quantitative assessment of metabolic activity, while CMR delineates transmural scar burden with high accuracy, both of which carry prognostic significance. [40–42]

Although viability has strong physiologic rationale, randomized evidence has introduced uncertainty about the extent to which it predicts improved outcomes after PCI in ICM. In the STICH viability substudy, the presence of viable myocardium was associated with better overall survival but did not significantly interact with treatment assignment—meaning that viability did not clearly identify patients who benefitted more from CABG compared with medical therapy alone. More recently, the REVIVED-BCIS2 trial also challenged the predictive value of viability for PCI benefit. Despite requiring

demonstration of viability in at least four dysfunctional segments, PCI did not improve all-cause mortality or heart failure hospitalization compared with optimal medical therapy. These findings suggest that viability alone may be insufficient for determining who benefits from PCI, particularly when substantial scar burden, microvascular disease, or diffuse cardiomyopathy coexist. [43–45]

A major explanation for the inconsistent prognostic value of viability lies in the complex relationship between viability, ischemia, and remodeling potential. Viability reflects surviving myocytes, but improvement in global LV function also depends on the extent of ischemia, mechanical dyssynchrony, and the severity of baseline ventricular dilation. Patients with extensive scar, advanced remodeling, or severely depressed right ventricular function may exhibit viable myocardium but still have limited capacity for recovery after PCI. Additionally, the threshold of viable myocardium required to translate into improved outcomes remains uncertain. CMR studies indicate that segments with >50% transmural LGE rarely recover, whereas those with <25% scar frequently improve; however, global recovery requires sufficient aggregate salvageable tissue. These nuances highlight why viability should be interpreted in conjunction with ischemia burden, coronary physiology, and LV remodeling indices rather than as a standalone prognostic marker. [46–48]

Coronary Physiology (FFR, iFR) and Ischemia Burden

Coronary physiology has become an essential tool for guiding revascularization in chronic coronary syndromes, and its relevance is magnified in patients with ischemic cardiomyopathy (ICM), where identifying flow-limiting lesions is crucial for optimizing PCI outcomes. Fractional flow reserve (FFR) and instantaneous wave-free ratio (iFR) provide lesion-specific assessments of ischemia and have consistently demonstrated superior prognostic value compared with angiographic guidance alone. Randomized trials such as FAME and DEFINE-FLAIR have shown that physiology-guided PCI reduces major adverse cardiovascular events and procedural complications compared with angiographically guided strategies. In the context of ICM, physiology may help distinguish lesions contributing to ongoing ischemia from those representing fixed scar or nonviable territories, thereby improving the precision of revascularization and potentially enhancing the likelihood of functional recovery. [49–51]

Residual ischemia after PCI is a major determinant of poor outcomes, influencing ventricular remodeling, symptom burden, and survival. Large perfusion studies, including an analysis from the ISCHEMIA trial imaging cohort, demonstrated a strong correlation between ischemia burden and adverse outcomes, with greater ischemic territory associated with higher rates of death and heart failure hospitalization. While REVIVED-BCIS2 did not mandate ischemia quantification, post-hoc analyses suggest that patients with minimal ischemia derive little benefit from PCI, aligning with longstanding observational evidence that revascularization is most valuable in those with substantial ischemia. Notably, microvascular dysfunction—a common feature in ICM due to chronic remodeling and endothelial injury—may limit the improvement in perfusion even when epicardial stenoses are relieved, further complicating prognostic assessment. [52–54]

Advances in physiologic imaging, including quantitative perfusion CMR and PET-derived myocardial blood flow, have broadened the understanding of ischemia beyond epicardial stenosis severity. These modalities provide comprehensive assessment of myocardial flow reserve (MFR), which has emerged as a powerful prognostic marker in both preserved and reduced LVEF populations. Studies have shown that reduced MFR predicts higher mortality independently of coronary anatomy and identifies patients with limited likelihood of benefit from PCI. Integrating coronary physiology with viability assessment may enhance patient selection by determining not only whether myocardium is alive but also whether it is ischemic—and therefore potentially recoverable. This combined strategy may help resolve discrepancies between randomized trial outcomes and real-world observational benefits of PCI in ICM. [55–57]

Left Ventricular Function and Remodeling After PCI

Left ventricular (LV) remodeling is a central determinant of prognosis in ischemic cardiomyopathy (ICM), and its trajectory after PCI strongly influences long-term outcomes. Improvements in LVEF

following revascularization occur predominantly when significant viable myocardium exists and ischemia is relieved effectively. Studies have shown that modest increases in LVEF—often as little as 5%—are associated with meaningful reductions in mortality and heart failure hospitalization, reflecting the sensitivity of prognosis to ventricular functional changes. However, remodeling after PCI remains highly variable, with many patients experiencing limited or no improvement. This heterogeneity is influenced by baseline scar burden, chamber dilation, and the degree of microvascular dysfunction, all of which restrict recovery despite technically successful revascularization. [58–60]

Reverse remodeling is facilitated by decreased wall stress, improved myocardial perfusion, and restoration of contractile reserve in hibernating segments. Cardiac magnetic resonance (CMR) studies consistently demonstrate that the absence of extensive transmural scar predicts greater LV end-systolic volume (LVESV) reduction and LVEF improvement after revascularization. In contrast, patients with large infarct cores or diffuse fibrosis often show minimal functional gains regardless of revascularization strategy. The STICH trial provided indirect evidence for this principle by demonstrating improved survival after CABG without substantial improvement in LVEF, suggesting that benefits may arise from mechanisms beyond simple contractile recovery. Nonetheless, smaller PCI-focused cohorts indicate that when remodeling does occur, it strongly correlates with improved symptoms, reduced arrhythmic burden, and better overall prognosis. [61–63]

Mechanical dyssynchrony and adverse geometric remodeling may further modulate the response to PCI. Patients with significant LV sphericity, papillary muscle displacement, or secondary mitral regurgitation often exhibit limited reverse remodeling because structural distortions persist even after ischemia is corrected. Additionally, persistent microvascular dysfunction—common in ICM due to chronic inflammation and endothelial damage—can blunt perfusion recovery at the tissue level despite restoration of epicardial flow. Biomarkers such as NT-proBNP and troponin have been shown to track remodeling responses, with decreases after PCI associated with favorable outcomes. However, because overall remodeling potential depends on multiple interrelated factors, prediction models combining imaging, biomarkers, and global functional parameters may offer the most accurate prognostic insight for post-PCI recovery. [64–66]

Chronic Total Occlusions and Complex Coronary Anatomy

Chronic total occlusions (CTOs) are common in ischemic cardiomyopathy (ICM), often representing longstanding obstructive disease affecting large myocardial territories. The presence of CTOs is associated with more extensive scar, greater ischemic burden, and worse baseline LV function, all of which contribute to poorer prognosis. Successful CTO PCI has been linked to improved angina control, enhanced quality of life, and in selected cases, improved LV function due to restored perfusion to hibernating myocardium. Observational registries consistently demonstrate survival benefits among patients who achieve CTO recanalization compared with unsuccessful attempts, although randomized evidence specific to ICM remains limited. The underlying principle is that CTO reperfusion may salvage viable myocardium supplied by collateral-dependent perfusion, which is typically inadequate for full metabolic recovery. [67–69]

Despite potential benefits, CTO PCI in ICM is technically challenging. Lesion complexity—characterized by heavy calcification, tortuosity, blunt stumps, and long occlusion length—contributes to procedural difficulty and increased risk. Patients with LV dysfunction face additional hazards, including heightened susceptibility to hemodynamic instability, contrast-induced nephropathy, and arrhythmic complications during prolonged procedures. The EXPLORE trial, which randomized patients with ST-elevation myocardial infarction and a concurrent CTO to CTO PCI versus no CTO PCI, did not demonstrate significant improvements in global LVEF; however, in a prespecified subgroup with LAD CTOs, there was evidence of greater LV functional recovery. Although not focused specifically on chronic ICM, these results suggest that CTO location and myocardial territory size may modulate the potential benefit. [70–72]

Advanced imaging tools and physiologic assessments have enhanced the ability to determine which

CTOs may yield meaningful prognostic improvement when recanalized. Cardiac MRI can quantify scar burden within the CTO territory, which strongly predicts the potential for contractile recovery. Similarly, PET-derived myocardial blood flow and metabolic activity can identify territories with insufficient perfusion but preserved cellular viability. CTOs supplying segments with low scar burden and preserved metabolic activity are more likely to recover after revascularization. However, CTO PCI does not universally improve outcomes, and failed attempts may expose patients to procedural risks without clinical benefit. Accordingly, the decision to pursue CTO revascularization in ICM must carefully balance anatomical feasibility, myocardial substrate, and the anticipated contribution to complete revascularization. [73–75]

High-Risk PCI and Mechanical Circulatory Support

Patients with ischemic cardiomyopathy (ICM) often undergo **high-risk PCI (HR-PCI)** because of severely reduced LVEF, complex multivessel coronary disease, chronic total occlusions, or hemodynamic instability. These features increase the risk of periprocedural hypotension, arrhythmias, and cardiogenic shock, making procedural planning critical for optimizing outcomes. The presence of extensive scar or impaired contractile reserve further limits the ability of the failing ventricle to tolerate ischemia during balloon inflation or prolonged device manipulation. As a result, the use of **mechanical circulatory support (MCS)** has expanded to stabilize hemodynamics, maintain coronary perfusion pressure, and prevent ischemia-induced decompensation during HR-PCI. Clinical registries and observational cohorts suggest that MCS-assisted PCI may improve procedural success rates and reduce hemodynamic collapse in selected high-risk patients, although definitive mortality benefits remain uncertain. [76–78]

Among MCS options, the **Impella percutaneous microaxial pump** is the most widely studied in HR-PCI. It provides forward flow support, unloads the LV, reduces myocardial oxygen demand, and augments systemic perfusion. The PROTECT II trial compared Impella with the intra-aortic balloon pump (IABP) in HR-PCI and demonstrated improved hemodynamic stability and a trend toward lower adverse events at 90 days, although the primary endpoint was not statistically different. Despite mixed randomized data, real-world USpella registry findings report improved survival and fewer major adverse cardiac events when Impella is used prophylactically in patients with severely reduced LVEF. Still, concerns remain regarding vascular complications, bleeding risk, and device-associated hemolysis, highlighting the need for selective deployment in patients with the highest anticipated hemodynamic vulnerability. [79–81]

Other MCS strategies, including **IABP**, **VA-ECMO**, and **surgically implanted support devices**, also play roles depending on patient phenotype. The IABP, despite declining routine use, may still benefit patients with modest LV dysfunction requiring short-duration support, although outcomes in ICM are less predictable. VA-ECMO offers full cardiopulmonary support but carries significant risks of bleeding, limb ischemia, and left ventricular distension; its role is generally limited to profound cardiogenic shock or salvage situations. Importantly, no mechanical support device guarantees survival benefit after PCI; rather, appropriate use mitigates periprocedural instability, enabling safer, more complete revascularization. As ongoing trials continue to evaluate MCS in HR-PCI, individualized assessment based on coronary anatomy, LV function, right ventricular performance, and comorbidity burden remains central to selecting the appropriate strategy. [82–84]

PCI vs CABG vs Medical Therapy: Prognostic Comparison

The prognostic landscape for ischemic cardiomyopathy (ICM) is heavily influenced by the modality of revascularization, with substantial differences observed between percutaneous coronary intervention (PCI), coronary artery bypass grafting (CABG), and optimized medical therapy (OMT). CABG has consistently shown survival advantages in patients with severe multivessel disease and impaired LVEF, largely supported by the STICH and STICHES studies, which demonstrated a significant reduction in all-cause mortality over 10 years. The anatomical benefit of bypassing obstructed vessels, combined with improved collateralization and protection from future plaque rupture, likely explains the durable

outcomes associated with CABG. PCI, while less invasive, does not provide the same long-term conduit protection and may be influenced more heavily by residual ischemia, incomplete revascularization, and plaque progression. This inherent distinction underlies why trials comparing PCI and CABG often show surgical superiority in patients with complex disease and LV dysfunction. [85–87]

The REVIVED-BCIS2 trial reshaped modern understanding of PCI in ICM by showing that PCI added to OMT did not improve all-cause mortality or heart failure hospitalization compared with OMT alone in patients with severe LV dysfunction and extensive CAD. Despite including patients with documented myocardial viability, PCI did not lead to improved LVEF or quality-of-life outcomes beyond early procedural benefit. These findings stand in contrast to earlier observational studies, which suggested improved LV function and survival after PCI in selected ICM patients. REVIVED raises critical questions regarding which patient subsets still stand to benefit, emphasizing that viability alone does not guarantee improvement and that ischemic burden, coronary physiology, microvascular integrity, and revascularization completeness may hold greater prognostic value. [88–90]

Comparative analyses between PCI and CABG consistently demonstrate better outcomes with CABG in patients with diffuse CAD, diabetes, or high anatomical complexity (e.g., SYNTAX score >33). However, PCI remains an important option for patients unsuitable for surgery due to frailty, comorbidities, or prohibitive operative risk. In such populations, PCI may improve symptoms, reduce angina, and facilitate better functional capacity, even without clear mortality benefit. Moreover, advancements in physiology-guided PCI, CTO techniques, and mechanical circulatory support may narrow the prognostic gap between PCI and CABG in carefully selected patients. Ultimately, the choice of revascularization strategy requires personalized evaluation, integrating anatomic complexity, myocardial substrate, comorbidity burden, and anticipated procedural risk. [91–93]

Biomarkers, Imaging, and Emerging Prognostic Tools

Biomarkers have become increasingly important in refining prognostic assessment after PCI in ischemic cardiomyopathy (ICM). Natriuretic peptides—particularly **NT-proBNP**—provide strong prognostic information on ventricular wall stress and correlate with both heart failure severity and long-term mortality. Reductions in NT-proBNP following revascularization have been associated with improved outcomes and may reflect favorable remodeling responses. High-sensitivity cardiac troponins also offer prognostic insights by identifying ongoing myocardial injury, which may arise from active ischemia, microvascular dysfunction, or chronic structural disease. Emerging biomarkers such as **galectin-3**, ST2, and markers of extracellular matrix turnover may further stratify risk by identifying patients with active fibrotic remodeling who are less likely to demonstrate functional recovery after PCI. Integrating biomarker trends with imaging and clinical data enhances risk prediction and helps identify subgroups who may derive the greatest benefit from revascularization. [94–96]

Advanced imaging modalities contribute substantially to prognostication by characterizing myocardial substrate, ischemic burden, and microvascular integrity beyond what coronary angiography can provide. **Cardiac magnetic resonance (CMR)** with late gadolinium enhancement (LGE) remains the gold standard for assessing scar burden and viability, with strong evidence showing that the extent of transmural scar predicts the likelihood of contractile recovery after revascularization. **Strain imaging** using speckle-tracking echocardiography offers additional prognostic value by detecting subtle mechanical dysfunction, with impaired global longitudinal strain associated with worse outcomes even when LVEF is preserved. **PET myocardial blood flow** quantification and flow reserve assessment further refine risk evaluation by identifying microvascular dysfunction, which strongly predicts mortality and may limit the capacity of revascularization to improve perfusion. Combining these imaging tools allows a more nuanced assessment of myocardial health and may better explain heterogeneous responses to PCI seen across clinical trials. [97–99]

Emerging prognostic tools are increasingly incorporating artificial intelligence (AI), machine learning, and integrated multimodality data to generate individualized risk predictions. Machine-learning models using CMR texture analysis, perfusion quantification, and clinical variables can identify patterns of

myocardial injury and remodeling that are not visually appreciable. Physiologic indices such as **quantitative flow ratio (QFR)** and **angiography-derived FFR** offer noninvasive approximations of lesion severity and have shown promise for simplifying ischemia assessment in patients unable to tolerate pressure-wire measurements. Furthermore, computational modeling of coronary flow, scar topology, and mechanical activation may soon help predict which patients will experience meaningful LV recovery after PCI. These innovations hold the potential to tailor revascularization strategies to patient-specific pathophysiology, bridging current evidence gaps highlighted by trials such as REVIVED-BCIS2. [100–102]

Conclusion

Percutaneous coronary intervention (PCI) in ischemic cardiomyopathy (ICM) remains a domain of significant clinical complexity, shaped by heterogeneous myocardial substrate, variable ischemia burden, and procedural challenges unique to patients with advanced ventricular dysfunction. Although the physiological rationale for revascularizing hibernating myocardium is sound, contemporary randomized and observational evidence demonstrates that the prognostic benefit of PCI is far from universal. Differences in outcomes are increasingly understood to arise not from the procedure itself, but from the interplay of viability, microvascular integrity, coronary anatomy, revascularization completeness, and remodeling potential. As such, PCI should not be viewed as a uniform therapeutic solution, but rather as a targeted intervention whose success depends on identifying the right patient phenotype.

A growing body of research indicates that improvement after PCI is most likely among patients with substantial ischemia, limited scar burden, and preserved capacity for reverse remodeling. Conversely, individuals with diffuse fibrosis, chronic adverse remodeling, or profound right ventricular impairment are less likely to experience meaningful recovery, despite technically successful revascularization. The recent divergence between CABG and PCI outcomes in major trials further underscores the importance of strategic treatment selection and highlights the limitations of relying on viability testing alone. It is now clear that prognostication must include broader assessments of physiologic flow, microvascular function, and structural remodeling.

Looking forward, the integration of advanced imaging, physiologic assessment, biomarkers, and emerging machine-learning platforms promises to transform the way clinicians tailor revascularization decisions in ICM. By leveraging multimodal data, future strategies may overcome current evidence gaps and allow more accurate prediction of which patients truly benefit from PCI. Ultimately, optimizing outcomes in ICM will require a shift from trial-level generalizations to individualized, data-driven precision medicine. In this evolving paradigm, PCI will remain an essential therapeutic option, but its prognostic value will be maximized only through careful patient selection, comprehensive risk stratification, and deliberate coordination with contemporary heart failure therapies.

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