

Cardiac Outcomes After Acute Myocardial Infarction: Determinants, Prognostic Markers, and Contemporary Risk Stratification

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ABSTRACT

Background: Acute myocardial infarction remains a major determinant of cardiovascular mortality and morbidity despite substantial improvements in reperfusion, pharmacotherapy and secondary prevention. Outcomes after infarction are heterogeneous and extend beyond short-term mortality to include cardiogenic shock, heart failure, adverse ventricular remodeling, recurrent myocardial infarction, recurrent ischemia, ventricular arrhythmias, sudden cardiac death, mechanical complications, stroke, rehospitalization and composite major adverse cardiovascular events. The magnitude of myocardial injury and the quality of reperfusion provide the central biological link between the acute event and subsequent prognosis. Ischemic time, infarct size, infarct location, left and right ventricular function, epicardial coronary flow, microvascular obstruction, intramyocardial hemorrhage, collateral circulation, multivessel coronary disease and completeness of revascularization modify this risk. Clinical factors including age, renal dysfunction, diabetes, hemodynamic status and heart failure at presentation further influence outcomes. Contemporary imaging has expanded risk assessment beyond left ventricular ejection fraction: global longitudinal strain, scar burden, infarct size, microvascular obstruction and adverse remodeling provide incremental prognostic information. Risk scores such as TIMI and GRACE remain clinically useful, while biomarker and multimodal imaging approaches offer increasingly refined stratification. This review integrates the mechanisms and determinants of early and long-term cardiac outcomes following acute myocardial infarction, with particular attention to angiographic predictors and stable patients presenting beyond conventional reperfusion windows. Understanding the interaction between coronary anatomy, myocardial viability, microvascular reperfusion and ventricular remodeling may enable more individualized follow-up and secondary prevention after infarction.

Keywords: acute myocardial infarction, cardiac outcomes, MACE, ventricular remodeling

Introduction

The prognosis of acute myocardial infarction has changed profoundly with coronary care units, antithrombotic treatment, fibrinolysis, primary PCI, high-intensity lipid lowering, neurohormonal therapy and systematic secondary prevention. Yet improvements in population-level survival should not obscure the considerable residual risk among individual patients. Death, heart failure, recurrent ischemic events and arrhythmias remain concentrated in patients with extensive myocardial injury, delayed reperfusion, hemodynamic compromise, multivessel coronary disease and major comorbidities. [1]

The phrase “cardiac outcomes” encompasses endpoints operating over different time scales. During the first hours and days, malignant ventricular arrhythmia, cardiogenic shock, acute heart failure and mechanical complications predominate. Over subsequent weeks and months, adverse remodeling, persistent LV dysfunction, recurrent infarction, stent-related events and heart-failure hospitalization become increasingly important. Years later, recurrent atherothrombotic disease, chronic ischemic cardiomyopathy, sudden death and comorbidity determine prognosis. [2]

Major adverse cardiovascular events are widely used as composite outcomes but lack a single universal definition. Three-point MACE usually includes cardiovascular death, MI and stroke, while other studies add urgent revascularization, unstable-angina hospitalization or heart failure. This variability is directly relevant to interpretation of observational studies. The supplied protocol appropriately specifies a three-point MACE endpoint of recurrent MI, stroke and cardiovascular mortality at six months, allowing the outcome to be interpreted consistently within that study.

A major research gap is the integration of early invasive markers with downstream clinical events. Conventional risk factors and EF are well established, but angiographic variables—including culprit-vessel patency, TIMI flow, corrected TIMI frame count, myocardial blush and collateral grade—potentially provide immediate information on both epicardial and tissue-level reperfusion. Their relationship to myocardial viability, ventricular recovery and MACE is particularly relevant among late-presenting MI patients.

Infarct Size as the Biological Foundation of Outcome

Infarct size represents the cumulative result of the area at risk, ischemic duration, residual flow and successful reperfusion. Larger infarcts destroy more contractile myocardium, lower LV systolic function, raise wall stress and increase susceptibility to adverse remodeling and heart failure. They also provide a larger substrate for ventricular tachyarrhythmia and are associated with greater neurohormonal activation. [3]

Time to reperfusion therefore influences prognosis predominantly through myocardial salvage. The maxim “time is muscle” is physiologically sound, although the relationship is modified by collateral circulation, intermittent occlusion and preconditioning. Delayed presentation generally results in greater irreversible injury, but marked interpatient variability means that clinical outcome cannot be predicted from time alone. This is particularly evident among stable late presenters in whom retained antegrade flow or collateral supply may preserve function. [4]

CMR provides highly reproducible quantification of infarct size and increasingly serves as a mechanistic endpoint in reperfusion studies. Infarct size correlates with subsequent LV function and remodeling, although microvascular obstruction may provide prognostic information beyond infarct size alone. The interaction between the amount of necrosis and the quality of tissue-level reperfusion therefore offers a more complete

description of post-MI injury. [5]

Epicardial Reperfusion and TIMI Flow

Successful restoration of the infarct-related artery is a major determinant of outcome after STEMI. TIMI grade 3 flow represents normal epicardial antegrade perfusion and is associated with better myocardial salvage and prognosis than impaired flow. Yet TIMI grading remains categorical and visually assessed, prompting development of quantitative approaches such as corrected TIMI frame count. [6]

Corrected TIMI frame count measures contrast transit to standardized distal coronary landmarks and can expose delayed coronary flow despite apparently patent epicardial anatomy. High frame counts may reflect residual epicardial resistance, distal embolization, microvascular dysfunction or the slow-flow phenomenon. In observational research, cTFC therefore offers an objective continuous variable suitable for correlation with viability and clinical outcomes. [7]

The protocol's division of patients into totally occluded IRA, patent IRA and slow-flow groups is clinically meaningful because these patterns may represent markedly different ischemic histories. A patent vessel at delayed angiography may reflect spontaneous reperfusion, whereas persistent occlusion may coexist with either completed scar or collateral-supported viable myocardium. Slow flow may indicate impaired microvascular function even after restoration of luminal patency.

Myocardial Blush and Microvascular Reperfusion

Epicardial success does not guarantee myocardial reperfusion. Distal thromboembolization, capillary damage, endothelial swelling, vasospasm, inflammatory activation, edema and intramyocardial hemorrhage can prevent effective tissue perfusion after the artery has been opened. This no-reflow phenomenon identifies a form of reperfusion failure associated with larger infarcts and impaired LV recovery. [8]

Myocardial blush grade provides an angiographic estimate of tissue perfusion. Reduced blush after otherwise successful PCI identifies patients in whom contrast fails to enter or clear normally from the myocardial microcirculation. The combination of TIMI 3 flow and preserved blush is consequently more informative than TIMI flow alone when assessing the success of reperfusion. [9]

CMR can characterize the same pathophysiological process more directly. Microvascular obstruction appears as a hypoenhanced region within the enhanced infarct core, while intramyocardial hemorrhage identifies particularly severe microvascular damage. Meta-analytic and pooled data consistently associate MVO with larger ventricular volumes, lower EF and greater rates of MACE.

More recent long-term analyses suggest that the prognostic signal of MVO is strongest during the first several years after STEMI. Persistent MVO is associated with adverse remodeling and poorer outcomes compared with transient or absent MVO, supporting the concept that the microcirculation is a major determinant of post-infarction risk rather than a purely imaging-defined curiosity.

Collateral Circulation and Outcome

Coronary collaterals can maintain residual perfusion when the epicardial culprit artery becomes occluded. By reducing the severity of ischemia within the area at risk, collaterals may slow progression of necrosis, preserve viable myocardium and support LV functional recovery. Their importance is most evident in patients with delayed or chronic coronary occlusion. [10]

The Rentrop classification provides a practical angiographic measure of collateral recipient-vessel filling.

Although it does not directly quantify collateral pressure or absolute blood flow, greater filling indicates established alternative channels. Meta-analytic studies involving STEMI populations have generally linked more developed collateral circulation with smaller infarct size or improved outcomes, although associations can be confounded by duration of underlying coronary disease and differences in reperfusion treatment. [11]

For late-presenting STEMI, collateral assessment may be particularly informative because it helps explain why time from symptom onset does not map uniformly to viability or LV dysfunction. Its prognostic role is best evaluated together with IRA patency, myocardial blush and later functional or perfusion imaging rather than in isolation.

Left Ventricular Ejection Fraction

LVEF remains one of the most powerful and clinically accessible predictors of outcome after MI. Reduced EF reflects the integrated effects of infarct size, stunning, pre-existing myocardial disease and loading conditions. Patients with severe LV systolic dysfunction have greater risks of death, heart-failure hospitalization and ventricular arrhythmia, and EF remains central to decisions regarding guideline-directed heart-failure therapy and primary-prevention ICD assessment. [12]

Early EF can, however, be misleading because stunned but viable myocardium may recover after reperfusion. Conversely, an apparently preserved EF can coexist with significant regional injury or impaired longitudinal function. Serial assessment is therefore important, particularly when decisions about device therapy or long-term prognosis depend on ventricular function. [13]

LV end-systolic volume and the trajectory of ventricular remodeling provide additional information. Progressive chamber dilation, increasing end-systolic volume and deterioration of EF indicate maladaptive remodeling and are associated with subsequent heart failure and mortality. Neurohormonal activation, altered wall stress, infarct expansion and changes in extracellular matrix all contribute. [14]

Global Longitudinal Strain

Speckle-tracking echocardiography quantifies myocardial deformation and can identify systolic impairment not captured by EF. Longitudinal fibers are predominantly located in the subendocardium and are particularly susceptible to ischemic injury, making global longitudinal strain sensitive to myocardial infarction. [15]

More abnormal GLS after MI correlates with larger infarct burden, lower EF and adverse events. Regional strain can also characterize the infarct territory and potentially predict recovery. The technique is attractive because it can be performed within routine echocardiography without ionizing radiation, though image quality, vendor differences and loading conditions affect measurements. [16]

In the supplied protocol, strain analysis at follow-up can therefore serve as a functional counterpart to perfusion imaging. Improvement in deformation may indicate recovery of stunned or viable myocardium even when conventional EF changes modestly, while persistently impaired GLS may identify residual myocardial dysfunction.

Heart Failure After Myocardial Infarction

Heart failure is one of the most important consequences of MI and may occur during the acute hospitalization or develop months to years later. Its incidence varies according to population, infarct definition, reperfusion era and diagnostic criteria. Contemporary reviews identify older age, diabetes, renal disease, previous MI, anterior infarction, large infarct size, reduced EF and incomplete reperfusion among major determinants.

Acute HF after infarction reflects loss of contractile myocardium, ischemic dysfunction of remote segments, mitral regurgitation, arrhythmia or mechanical complications. Pulmonary congestion and higher Killip class at presentation are strongly associated with mortality. In older registry data, the combination of HF or LV systolic dysfunction after MI accounted for a disproportionately large share of in-hospital deaths.

Long-term HF arises through a combination of residual systolic dysfunction, progressive ventricular remodeling, recurrent ischemia or reinfarction, functional mitral regurgitation and comorbid myocardial disease. Prevention therefore begins during the index event with rapid reperfusion and continues through evidence-based secondary prevention and treatment of LV dysfunction.

Cardiogenic Shock

Cardiogenic shock represents the most severe manifestation of pump failure after acute MI. Patients develop hypotension and systemic hypoperfusion because the damaged ventricle cannot maintain adequate cardiac output. Extensive LV infarction is the commonest mechanism, although RV infarction and mechanical complications must be recognized because management differs. [17]

The landmark SHOCK trial established the foundation for contemporary early revascularization in MI-related cardiogenic shock. Although 30-day mortality was not significantly lower in the early-revascularization group, mortality at six months was reduced, supporting an invasive strategy for suitable patients.

The optimal PCI strategy in shock is also distinct from that in hemodynamically stable multivessel MI. In CULPRIT-SHOCK, culprit-lesion-only PCI with the option of staged procedures produced a lower 30-day risk of death or renal-replacement therapy than immediate multivessel PCI. This observation illustrates the need to interpret “complete revascularization” according to the patient's hemodynamic context.

Ventricular Arrhythmia and Sudden Cardiac Death

Ventricular tachycardia and ventricular fibrillation occur through different mechanisms at different stages after MI. Early arrhythmias are often driven by acute ischemia, metabolic abnormalities and reperfusion, whereas late ventricular tachycardia typically depends on re-entry through heterogeneous scar and border-zone tissue. [18]

Severe LV dysfunction remains a major marker of long-term sudden-death risk, but EF alone has limited sensitivity because many sudden deaths occur in patients with EF above traditional ICD thresholds. Scar extent, peri-infarct heterogeneity, ventricular ectopy, autonomic markers and CMR tissue characterization are therefore being investigated to improve risk stratification. [19]

The timing of EF reassessment matters because early ventricular dysfunction may recover. Implantation of a primary-prevention ICD very early after infarction has not reproduced the survival benefit observed in chronic ischemic cardiomyopathy, partly because reductions in arrhythmic death can be offset by competing non-arrhythmic mortality during the vulnerable early period. [20]

Recurrent Myocardial Infarction and Recurrent Ischemia

After surviving the index infarction, patients remain at risk from the culprit lesion, nonculprit coronary disease and systemic atherosclerotic activity. Recurrent MI may result from stent thrombosis, progression or rupture of a different plaque, untreated severe stenosis, graft disease or inadequate adherence to secondary-prevention therapy. [21]

Multivessel CAD is common in STEMI and identifies greater atherosclerotic burden. Contemporary

randomized trials have shown benefit from planned complete revascularization of significant nonculprit lesions in hemodynamically stable patients. The COMPLETE trial demonstrated that complete revascularization reduced major cardiovascular outcomes compared with culprit-lesion-only PCI in STEMI with multivessel disease.

FFR-guided strategies have also been evaluated. DANAMI-3-PRIMULTI showed that staged FFR-guided complete revascularization reduced the composite primary outcome compared with treatment of the infarct-related artery alone, with much of the difference driven by subsequent revascularization.

This evidence should not be extrapolated directly to cardiogenic shock, where immediate multivessel PCI can be harmful, or to stable patients with a persistently occluded IRA several days after infarction, where OAT did not show benefit from routine late PCI. Clinical state and timing therefore determine how anatomy translates into treatment and outcome.

Mechanical Complications

Ventricular septal rupture, papillary muscle rupture with acute severe mitral regurgitation and free-wall rupture are uncommon in the PCI era but remain catastrophic. Risk rises with delayed reperfusion, older age and larger transmural infarction. Mechanical complications often present with sudden hemodynamic deterioration several days after MI, although timing varies. [22]

Papillary muscle rupture most frequently involves the posteromedial papillary muscle because of its single coronary blood supply and produces acute severe mitral regurgitation, pulmonary edema and shock. Ventricular septal rupture creates an acute left-to-right shunt, while free-wall rupture may cause hemopericardium and tamponade. Rapid echocardiographic diagnosis and multidisciplinary surgical or structural intervention are essential.

The persistence of these complications reinforces a central theme: even in modern practice, delayed or failed reperfusion can convert an otherwise survivable MI into an event with major structural consequences.

Right Ventricular Infarction

Right ventricular involvement is particularly relevant in inferior STEMI and can cause severe hypotension despite relatively preserved LV systolic function. Clinical outcome depends on the extent of RV injury, associated LV infarction, tricuspid regurgitation and maintenance of atrioventricular synchrony. [23]

The RV often demonstrates greater potential for recovery than the LV because of lower oxygen requirements, dual coronary supply and favorable transmural perfusion characteristics. Nonetheless, severe RV dysfunction increases short-term mortality and complicates volume and vasopressor management. Echocardiography and CMR can define RV size and function when the clinical picture suggests disproportionate right-sided failure.

Renal Dysfunction and Cardiorenal Interaction

Renal dysfunction at presentation is strongly associated with mortality after MI. Chronic kidney disease identifies a population with greater age, diabetes, vascular disease, calcification, anemia and bleeding risk, while acute kidney injury may develop through shock, congestion, inflammation or nephrotoxic exposure. [24]

Renal impairment also complicates treatment because it alters antithrombotic dosing, increases bleeding risk and may restrict use of particular diagnostic or therapeutic interventions. Creatinine is consequently incorporated into the GRACE risk model and remains an easily obtained marker with substantial prognostic significance.

Diabetes and Metabolic Risk

Diabetes is associated with more diffuse and multivessel coronary disease, endothelial dysfunction, microvascular abnormalities and impaired myocardial metabolism. Patients may also present later because ischemic symptoms are atypical or less prominent. These mechanisms contribute to greater risks of recurrent MI, HF and cardiovascular mortality. [25]

Modern outcome modification extends beyond glucose lowering. SGLT2 inhibitors and GLP-1 receptor agonists have transformed cardiovascular risk reduction in appropriate populations, although their specific initiation after MI must follow the relevant evidence base and individual indications. Intensive lipid lowering, smoking cessation, blood-pressure control and cardiac rehabilitation remain core components of secondary prevention.

Age, Frailty, and Comorbidity

Age is one of the strongest predictors in post-MI risk models. Older adults have lower physiological reserve and higher prevalence of renal dysfunction, anemia, cerebrovascular disease, calcified coronary anatomy and bleeding risk. Yet chronological age alone does not determine procedural benefit, and contemporary decisions should account for frailty, cognition, life expectancy and patient goals. [26]

The coexistence of multiple prognostic domains also explains why two patients with similar angiographic infarcts can have very different outcomes. A moderate infarct in a frail patient with severe CKD may carry greater overall mortality than a larger infarct in a younger patient without comorbidity.

Clinical Risk Scores

The TIMI risk score for STEMI was developed as a simple bedside score incorporating readily available clinical features and demonstrated substantial discrimination of short-term mortality risk. Its simplicity makes it useful for initial categorization, although modern reperfusion practice and newer biomarkers have altered baseline event rates since the original derivation.

The GRACE model was developed across a broad ACS population and incorporates age, heart rate, systolic blood pressure, creatinine, Killip class, cardiac arrest, ST deviation and biomarker elevation. It predicts in-hospital and post-discharge risk and remains widely referenced in contemporary ACS guidance.

Risk scores should support rather than replace clinical judgment. They are particularly valuable because they force simultaneous consideration of hemodynamic, renal, electrical and myocardial injury markers that clinicians might otherwise evaluate separately.

Biomarkers and Prognosis

Cardiac troponin confirms myocardial injury and its concentration relates broadly to infarct magnitude, although timing, assay characteristics, renal function and reperfusion influence the observed peak. Serial measurements therefore contribute more to diagnosis and estimation of injury than a single isolated value. [27]

Natriuretic peptides reflect ventricular wall stress and are associated with heart failure and mortality after MI. Higher concentrations can identify risk not fully captured by EF, partly because they integrate systolic dysfunction, diastolic pressure, renal interaction and neurohormonal activation. [28]

Inflammatory markers such as C-reactive protein and leukocyte indices have prognostic associations but are less specific. Future approaches increasingly combine clinical variables, laboratory biomarkers, angiographic parameters and imaging rather than relying on any single marker.

CMR-Based Prognostic Phenotyping

CMR can move risk stratification beyond EF by measuring infarct size, transmural scar, myocardial edema, microvascular obstruction, hemorrhage and biventricular function. Among these markers, MVO has repeatedly demonstrated a strong association with adverse remodeling, HF and cardiovascular events. [29]

Scar heterogeneity may also contribute to ventricular arrhythmia by generating conducting channels through the peri-infarct zone. Quantitative mapping and automated tissue segmentation could eventually integrate arrhythmic and heart-failure risk in a single examination, although standardization and incremental clinical utility must be demonstrated before widespread routine use.

Adverse Ventricular Remodeling

Post-infarction remodeling begins within hours and continues for months. Infarct expansion thins and dilates the injured region, while increased wall stress promotes hypertrophy and dilation of remote myocardium. Neurohormonal activation initially helps maintain cardiac output but subsequently contributes to fibrosis, chamber enlargement and functional deterioration. [30]

The clinical consequence is the transition from an acute focal ischemic event to chronic ischemic heart failure. Reperfusion limits the initial trigger by reducing infarct size, while ACE inhibitors or ARBs, mineralocorticoid receptor antagonists in appropriate patients, beta-blockers and contemporary heart-failure therapies modify downstream remodeling and events.

Serial echocardiography is therefore more than measurement of EF. LV end-diastolic and end-systolic volumes, regional wall motion, mitral regurgitation, RV function and strain provide a longitudinal picture of remodeling and recovery.

Late Presentation and Outcome

Patients presenting late after STEMI remain clinically important in regions where prehospital delay, access to PCI, atypical symptoms and health-system factors are common. Delay generally predicts less salvage and more complications, but this population is biologically heterogeneous. Some have persistent total occlusion with completed infarction; others have spontaneous reperfusion, slow flow, collateral-dependent viable myocardium or critical multivessel disease. [31]

This heterogeneity supports early anatomical characterization in selected patients rather than treating “late STEMI” as a single pathological entity. The supplied protocol's distinction between occluded, patent and slow-flow IRA groups provides a useful framework for studying whether the angiographic phenotype predicts subsequent perfusion, functional recovery and MACE.

The therapeutic interpretation requires caution. In stable asymptomatic patients with a persistently occluded IRA days after MI, OAT demonstrated no routine clinical advantage of PCI. In contrast, ongoing ischemia, acute HF, hemodynamic instability or serious arrhythmia changes the treatment indication substantially. [32]

Revascularization and Long-Term Outcome

Primary PCI remains the preferred reperfusion strategy for STEMI when performed promptly by an experienced system. Compared with fibrinolysis in historical randomized evidence, primary PCI reduced reinfarction, stroke and short-term mortality when timely access could be achieved. [33]

Among hemodynamically stable MI patients with multivessel disease, the evidence has shifted toward planned complete revascularization. COMPLETE and earlier trials demonstrated reductions in recurrent ischemic

outcomes, supporting treatment of significant nonculprit disease either during the index hospitalization or in a staged manner according to anatomy and clinical circumstances. [34]

Revascularization must still be separated from the question of viability in a chronic or late occluded territory. Demonstrating living myocardium does not guarantee that PCI will improve prognosis, just as extensive scar does not eliminate every potential reason for revascularization. Symptoms, ischemia, anatomy, feasibility, ventricular function and expected procedural risk remain central.

Contemporary Secondary Prevention

Long-term outcome after MI is determined not only by the initial infarct but also by prevention of the next event. Antiplatelet therapy, intensive LDL-cholesterol reduction, smoking cessation, physical activity, cardiac rehabilitation, blood-pressure management and optimal diabetes treatment substantially modify recurrent risk. [35]

The 2023 ESC ACS guideline emphasizes comprehensive post-ACS secondary prevention and individualized antithrombotic strategies balancing ischemic and bleeding risks. The 2025 ACC/AHA multisociety ACS guideline similarly incorporates contemporary evidence across antiplatelet therapy, lipid lowering, revascularization and secondary prevention and now supersedes several older US ACS recommendations.

Modern prognosis therefore depends on an entire continuum of care: symptom recognition, emergency transport, reperfusion, procedural success, inpatient complication management, ventricular reassessment, secondary-prevention therapy and long-term adherence.

Linking Viability and Outcomes

Viability and outcome should not be treated as independent themes. Preserved myocardium can support subsequent LV recovery, while extensive nonviable scar increases the likelihood of persistent systolic dysfunction, adverse remodeling and ventricular arrhythmia. The relationship becomes especially relevant in late MI, where angiography may suggest residual perfusion and imaging can determine whether this translates into surviving myocardium. [36]

Yet contemporary randomized evidence warns against assuming that viable myocardium automatically predicts a prognostic benefit from revascularization. In ischemic LV dysfunction, STICH viability analyses did not demonstrate a significant interaction between viability and the survival effect of CABG, and REVIVED similarly found that the extent of viable dysfunctional myocardium did not identify patients benefiting from PCI.

For outcome prediction, scar burden and microvascular injury may therefore be at least as important as the amount of apparently viable tissue. This conceptual shift has major implications for future post-MI research: models should integrate viable myocardium, irreversible scar, MVO, coronary anatomy and global clinical risk.

Future Risk Stratification

Artificial intelligence and machine-learning models can combine dozens of variables derived from history, ECG, laboratory results, angiography and imaging. Such approaches may identify nonlinear interactions not represented by conventional regression scores. Their main challenge is transportability: excellent performance in a single-center or retrospective dataset does not guarantee generalizability across health systems or populations. [37]

Conclusion

Cardiac outcomes after acute myocardial infarction are determined by the interaction between ischemic injury, epicardial and microvascular reperfusion, ventricular function, coronary anatomy, systemic comorbidity and secondary prevention. Infarct size, cardiogenic shock, heart failure, reduced LV function, microvascular obstruction, multivessel disease and renal dysfunction identify particularly high-risk patients, while successful reperfusion and comprehensive long-term therapy improve prognosis. Angiographic indices including TIMI flow, corrected TIMI frame count, myocardial blush and collateral filling may provide early insight into both myocardial preservation and future risk, especially in late-presenting infarction. Integrating these variables with perfusion imaging, strain and clinical outcomes offers a rational approach to individualized post-MI risk stratification.

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