REVIEW ARTICLE

CURRENT CONCEPTS

Myocardial Infarction Due to Percutaneous Coronary Intervention

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N Engl J Med 2011;364:453-64.

Approximately 1.5 million patients undergo percutaneous coronary intervention (PCI) in the United States every year.[1] Depending on local practices and the diagnostic criteria used, 5 to 30% of these patients (75,000 to 450,000) have evidence of a periprocedural myocardial infarction.[2,3] At the higher estimate, the incidence of these events is similar to the annual rate of major spontaneous myocardial infarction.[1] Thus, many cardiologists and internists are likely to encounter patients with coronary artery disease who have sustained a periprocedural myocardial infarction. However, the clinical significance of these events and their management remain a matter of considerable controversy and uncertainty (Table 1).[4-6] Questions that often arise include the following: Do we need to routinely screen patients for periprocedural myocardial infarction? Which patients should be observed in the hospital for a prolonged period after periprocedural myocardial infarction? What are the therapeutic implications, and what should we tell patients who sustained a periprocedural myocardial infarction despite an otherwise successful procedure? Is a periprocedural myocardial infarction prognostically equivalent to a spontaneous myocardial infarction? Is periprocedural myocardial infarction a valid end point in clinical trials? The aim of this review is to address these questions and to provide a current perspective on this issue.

Definitions and Predictors of PCI-Related Myonecrosis

Current PCI guidelines give a class I recommendation for the measurement of cardiac biomarkers (the MB fraction of creatine kinase [CK-MB], cardiac troponin, or both) in patients who have signs or symptoms suggestive of myocardial infarction during or after PCI and for those who have undergone complicated procedures.[7] In addition, a class IIa recommendation is given for routine measurements of cardiac biomarkers 8 to 12 hours after the procedure. In either case, "a new CK-MB or troponin I or T rise greater than 5 times the upper limit of normal would constitute a clinically significant periprocedural MI [myocardial infarction]."[7] The more recent consensus document on the universal definition of myocardial infarction specifically classifies cardiac-biomarker levels that are more than 3 times the upper reference limit as indicative of a periprocedural myocardial infarction and recommends measurement of cardiac troponin as the preferred biomarker.[8] Given the availability of high-sensitivity cardiac troponin assays, this guideline establishes the threshold for a diagnosis of periprocedural myocardial infarction at very low levels of myonecrosis.

The predictors of periprocedural myocardial infarction can be broadly categorized as patient-, lesion-, and procedure-related risk factors.[2] The major risk factors, in terms of both frequency and potency, are complex lesions (e.g., the presence of thrombus, stenosis of a saphenous-vein graft, or a type C lesion), complex procedures

Table 1. Evidence for and against the Clinical Significance of Periprocedural Myocardial Infarction.*

Evidence for Clinical Significance	Evidence against Clinical Significance
Patients with elevated cardiac biomarkers after PCI have evidence of focal infarction on cardiac imaging	Virtually all data correlating PMI to adverse clinical outcomes are derived from retrospective studies that have shown associations but not causal relationships
A large number of studies have shown a correlation between PMI and adverse clinical outcomes (see the Supplementary Appendix, available with the full text of this article at NEJM.org), and these studies greatly outnumber those that do not	Retrospective studies are generally limited because they cannot adequately adjust for all possible confounding variables with respect to baseline clinical, angiographic, and procedural characteristics that may determine the likelihood of both PMI and adverse outcomes
There is a positive correlation between the magnitude of postprocedural biomarker elevation and the likelihood of adverse outcomes	Most studies did not use high-sensitivity cardiac troponin assays; when these assays were used, the studies did not apply the currently recommended 99th percentile cutoff value for the upper limit of the normal range
Studies have shown that pre-PCI interventions such as statin therapy reduce the frequency of PMI and improve long-term outcomes	In most cases, PMI results in minimal myonecrosis and therefore does not substantially impair cardiac function — one of the most important determinants of outcome in coronary artery disease

* PCI denotes percutaneous coronary intervention, and PMI periprocedural myocardial infarction.

(e.g., treatment of multiple lesions or use of rotational atherectomy), and associated complications (e.g., abrupt vessel closure, side-branch occlusion, distal embolization, or no reflow).[2,9-12] In contrast, patient-related factors, such as advanced age, diabetes mellitus, renal failure, multivessel disease, and left ventricular dysfunction, are the important determinants of clinical outcomes after PCI.[2,9-11] The occurrence of periprocedural ischemic symptoms, particularly chest pain at the end of the procedure, or electrocardiographic evidence of ischemia defines the subgroup of patients most likely to have periprocedural myocardial infarction.[11,13]

MECHANISMS OF PCI-RELATED MYONECROSIS

Large periprocedural myocardial infarcts are usually due to angiographically visible complications; however, this is generally not the case in the vast majority of patients with elevated biomarker levels after PCI.[6,14,15] Cardiac magnetic resonance imaging (MRI) has confirmed two distinct locations for procedural myonecrosis: adjacent to the site of the intervention, where the injury is most likely due to epicardial side-branch occlusion, and downstream from the intervention site, where it is most likely due to compromise of the microvascular circulation (Fig. 1).[2,16] Acute myocardial injury occurs with equal frequency at the two locations and is detected on MRI in 25% of patients after PCI, with a mean infarct size of approximately 5% of the left ventricular mass.[3] The size of distal infarcts correlates directly with the extent to which the plaque volume is reduced (embolized) by PCI, since more debris is sent downstream, but this is not so for the proximal type of injury. Moreover, the composition of the plaque influences the extent of periprocedural myonecrosis. PCI for plaques with large necrotic cores leads to greater degrees of myonecrosis, whereas fibrous plaques are relatively inert in this regard.[17,18]

Embolization of plaque material has been detected on intracoronary Doppler ultrasonography during PCI. Although it occurs at each phase of the intervention, embolization is most pronounced during stent implantation.[19] Even though the number of microemboli correlates positively with the severity of myocardial microvascular dysfunction and myonecrosis, there is considerable overlap with regard to the magnitude of plaque microembolization between patients with and those without periprocedural myocardial infarction.[19,20] This finding suggests that factors other than the burden of plaque microembolization influence the likelihood of periprocedural myocardial infarction, such as the release of vasoactive factors from the atherosclerotic plaque, platelet activation, and preexisting vulnerability of the myocardium.[2]

TRADITIONAL FOCUS ON POSTPROCEDURAL MYONECROSIS

In the CK-MB and early cardiac troponin era, numerous studies evaluated the clinical significance

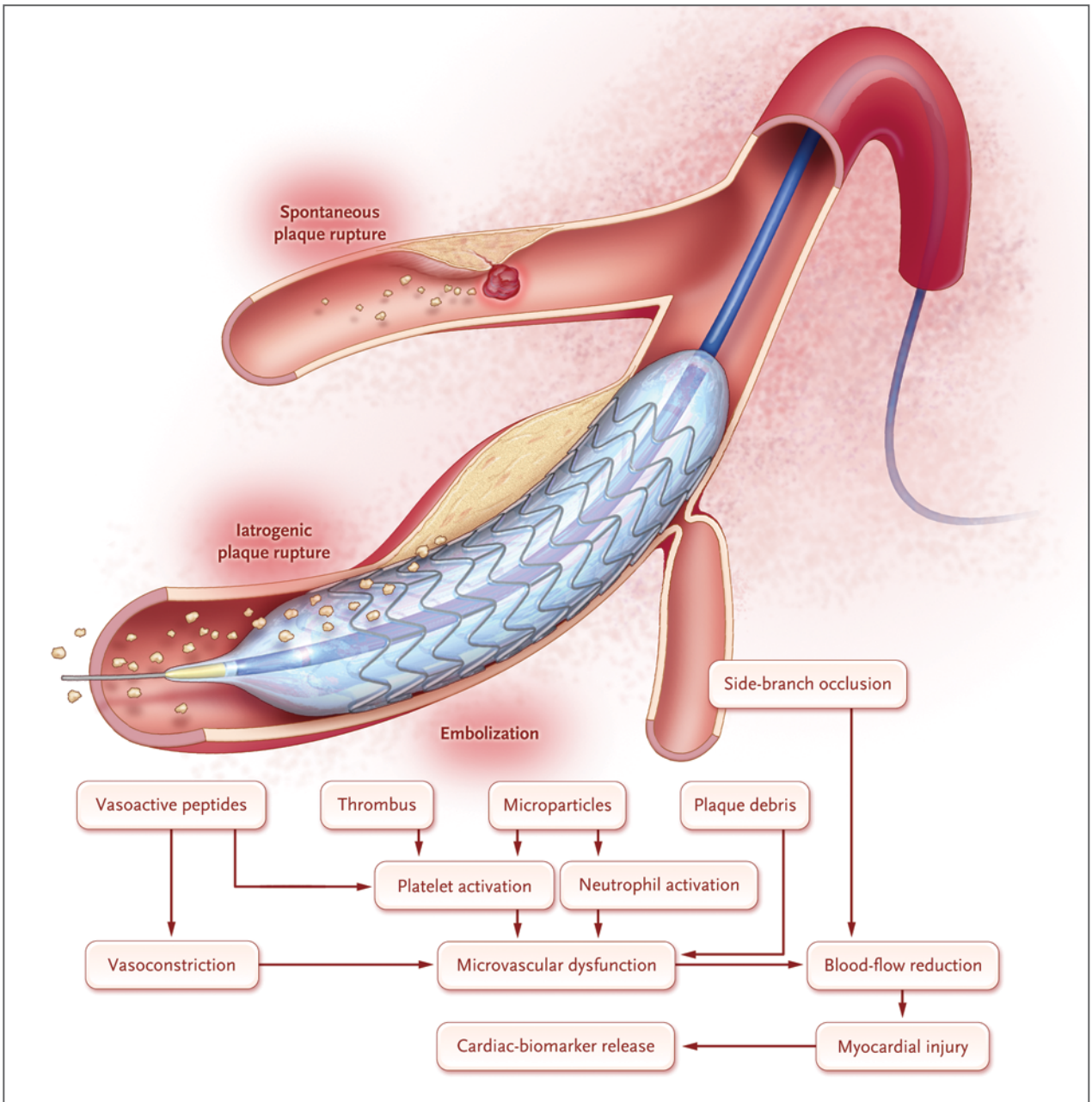


Figure 1. Mechanisms Underlying Periprocedural Myocardial Infarction.

Cardiac-biomarker elevation before percutaneous coronary intervention (PCI) is primarily due to spontaneous rupture of vulnerable plaques, epicardial thrombosis, and subsequent myocardial injury. In the absence of abrupt, PCI-related epicardial-artery closure, periprocedural myocardial infarction is related to either side-branch occlusion or iatrogenic plaque rupture by balloons and stents, which promotes microvascular injury owing to distal embolization, the release of vasoactive peptides, or both.

of cardiac-biomarker elevations after PCI, and these studies have been systematically reviewed in a previous publication.[2] The general conclusion from the retrospective analyses was that a CK-MB elevation higher than 5 times the upper limit of normal was independently associated with an increased risk of in-hospital adverse cardiac events, whereas lower levels did not appear to influence in-hospital outcomes significantly (Table 2).[21,26,40-43] Data indicating a relationship between the CK-MB level and long-term survival were less consistent. The results of several studies sug-

Table 2. Large Cohort Studies of Cardiac-Biomarker Elevation after Percutaneous Coronary Intervention (PCI), and Outcomes.*

Study	No. of Patients	Incidence of ACS	Type of Intervention	Incidence	In-Hospital Outcomes†	Length of Follow-up	Multivariate Adjusted Long-Term Outcomes
		%		%		*mo*	
Stone et al.[21]	7147	68.9	PCI		Increased risk of death (odds ratio, 8 for CK-MB >8× ULN; 67 for Q-wave MI)	24	Increased risk of death (hazard ratio, 2.2 for CK-MB >8× ULN; 9.9 for Q-wave MI)
CK-MB, >4–32 ng/ml				29.6			
CK-MB, >32 ng/ml				7.7			
Q-wave MI				0.6			
Dangas et al.[22]	4085	67.3	PCI		NA	12	Increased risk of death or MI (odds ratio, 1.5 for CK-MB >5× ULN)
CK-MB, 4–20 ng/ml				24.1			
CK-MB, >20 ng/ml				12.8			
Ajani et al.[23]	1326	85.4	PCI		NA	12	Increased risk of death or MI (odds ratio, 1.57 for CK-MB >3× ULN)
CK-MB, 4–12 ng/ml				25			
CK-MB, >12 ng/ml				20			
Kini et al.[24]‡	1675	NA	PCI		Increased risks of chest pain (58% vs. 9–12%), heart failure (35% vs. 6–7%), and increased length of stay for CK-MB >5× ULN vs. other elevations and normal levels	13±3	No association
CK-MB, 1–3× ULN				12.6			
CK-MB, >3–5× ULN				3.7			
CK-MB, >5× ULN				2.4			
Brener et al.[25]	3478	64.0	Stent placement		NA	15±15	Increased risk of death (odds ratio, 1.89 for CK-MB >3× ULN; 6.36 for CK-MB >10× ULN)
CK-MB, >8.8–26.4 ng/ml (CK-MB, >1–3× ULN)				14.5			
CK-MB, >26.4–44 ng/ml (CK-MB, >3–5× ULN)				4.5			
CK-MB, >44–88 ng/ml (CK-MB, >5–10× ULN)				2.9			
CK-MB, >88 ng/ml (CK-MB, >10× ULN)				2.4			
Ellis et al.[26]	8409	63.0	PCI		Increased length of stay for CK-MB >1× ULN	4	Increased risk of death (1.2, 1.9, and 8.9% for CK-MB <1, 1–5, and >5× ULN, respectively)
CK-MB, 8.8–40 ng/ml				13.6			
CK-MB, >40 ng/ml				3.6		48	Increased risk of death (15.1, 16.9, and 19.4% for CK-MB <1, 1–5, and >5× ULN, respectively)

Brener et al.[27]	3573	NA	PCI		NA	36	Increased risk of death (hazard ratio, 1.1 for CK-MB >10× ULN)
CK-MB, >8.8–26.4 ng/ml (CK-MB, >1–3× ULN)				21			
CK-MB, >26.4–44 ng/ml (CK-MB, >3–5× ULN)				6			
CK-MB, >44–88 ng/ml (CK-MB, >5–10× ULN)				6			
CK-MB, >88 ng/ml (CK-MB, >10× ULN)				5			
Hong et al.[28]	1693	79.0	Saphenous-vein graft PCI		Increased need for balloon pump (7.8% vs. 1.1%) and repeat PCI (4.2% vs. 1.2%) for CK-MB elevation vs. no elevation	12	Increased risk of death (hazard ratio, 3.3 for CK-MB >5× ULN)
CK-MB, 4–20 ng/ml				32.1			
CK-MB, >20 ng/ml				15.2			
Andron et al.[29]	3864	30.4	PCI		NA	6–42	Increased risk of death (hazard ratio, 1.3, 1.76, and 2.26 for CK-MB 1–3, >3–5 and >5× ULN, respectively)
CK-MB, 4–12 ng/ml				19.9			
CK-MB, >12–20 ng/ml				4.4			
CK-MB, >20 ng/ml				5.1			
Jang et al.[30]	1807	40.9	Drug-eluting stenting PCI		No association	13±7	Increased risk of death (0.5, 1.1, and 2.6% for CK-MB <1, 1–5, and >5× ULN, respectively)
CK-MB, 5–25 ng/ml				14.6			
CK-MB, >25 ng/ml				6.4			
Natarajan et al.[31]	1128	61.0	PCI		Increased risk of major cardiac events (3.8 for cTnI ≥5× ULN)	12	No association
cTnI, 1–4× ULN				7.6			
cTnI, ≥5× ULN				9.1			
Nallamothu et al.[32]	1157	36.5	PCI		NA	11±7	Increased risk of death (hazard ratio, 2.4 for cTnI ≥8× ULN, 8.9 for Q-wave MI)
cTnI, 2–5.9 ng/ml				16.0			
cTnI, 6–9.9 ng/m				4.6			
cTnI, 10–15.9 ng/ml				2.0			
cTnI, ≥16.0 ng/ml				6.5			
Q-wave MI				0.3			

Table 2. (Continued.)

Study	No. of Patients	Incidence of ACS	Type of Intervention	Incidence	In-Hospital Outcomes†	Length of Follow-up	Multivariate Adjusted Long-Term Outcomes
		%		%		*mo*	
Prasad et al.[33]	1949	47.9	PCI		Increased length of stay	26	Increased risk of death (hazard ratio, 1.2 per $\log_2$ increase in cTnT)
cTnT, ≥0.03 ng/ml				19.6			
Hubacek et al.[34]	1208	31.0	PCI		NA	24	No association
Increase in cTnT >0.1 ng/ml				20			
Feldman et al.[35]	1601	43.3	PCI		No association	25±8	Increased risk of death (hazard ratio, 1.6)
cTnI, ≥0.15 ng/ml				51.9			
De Labriolle et al.[36]	3200	0.0	PCI		NA	12	No association
cTnI, >0.30 ng/ml				23.4			
Cavallini et al.[37]	2362	45.1	PCI		NA	24	No association
cTnI, 0.15–0.45 ng/ml				19.7			
cTnI, >0.45 ng/ml				19.8			
Fuchs et al.[38]	1129	70.9	PCI		Increased risk of major adverse cardiovascular events (odds ratio, 2.1 for cTnI >3× ULN)	8	No association
cTnI, 0.15–0.45 ng/ml				15.2			
cTnI, >0.45 ng/ml				15.4			
CK-MB, >4 ng/ml				40.8			
Cavallini et al.[39]	3494	50.8	PCI		NA	24	Increased risk of death (odds ratio, 1.04 per peak CK-MB ratio unit)§
cTnI, >0.15 ng/ml				44.2			
CK-MB, >5 ng/ml				16.0			

* Plus–minus values are means ±SD. Only data from studies that included at least 1000 patients, long-term outcome data, and concentration-based biomarker analysis are shown. Hazard ratios were determined by means of a multivariate Cox proportional-hazards regression model, if available; otherwise, odds ratios were determined by multivariate analysis. Acute coronary syndromes (ACS) included angina at rest and urgent priority interventions. CK-MB denotes the MB fraction of creatine kinase, cTnI cardiac troponin I, cTnT cardiac troponin T, MI myocardial infarction, NA not available, and ULN upper limit of the normal range.

† Outcomes other than evolving myocardial infarction are shown. $P<0.05$ for all comparisons.

‡ In this study, the final analysis was based on mass immunoassay.

§ This ratio was calculated by dividing the maximum post-PCI level by the ULN or baseline level of CK-MB.

gested that any elevation in CK-MB was associated with reduced long-term survival and that there was a direct correlation between the magnitude of myonecrosis and mortality.[26,39,41,42] In contrast, other studies have shown that only large myocardial infarctions, variably defined as a CK-MB level exceeding 5 or 8 times the upper limit of normal or the presence of new Q waves, were predictive of a poor long-term outcome, especially if they were related to an unsuccessful revascularization procedure (Table 2).[21,40,43,44]

Studies evaluating the relationship between the postprocedural cardiac troponin level and long-term mortality, in general, have not excluded patients with acute coronary syndromes, many of whom would have had abnormal cardiac-biomarker levels at baseline.[31,32,35,39,45-47] Thus, the reported frequency of postprocedural elevations in cardiac troponin has been highly variable, and although some studies showed that the serum concentration of cardiac troponin was an independent predictor of survival, others did not (Table 2). The inconsistent findings were most likely due to heterogeneity of the inclusion criteria, variations in the sensitivity and specificity of the biomarker assays, different sample sizes, and differences in the duration of follow-up. Two recent meta-analyses concluded that an elevated cardiac troponin level after PCI does provide prognostic information.[48,49] Both analyses were influenced by studies from our catheterization laboratories on postprocedural cardiac troponin T elevations in which we had reached a similar conclusion.[33,50] However, the studies included in the meta-analyses (including our own) had used cardiac troponin cutoff values for normal that were higher than the currently recommended 99th percentile, thereby limiting the accuracy of their conclusions.[8]

FOCUS ON PREPROCEDURAL RISK

To date, virtually all studies of periprocedural myocardial infarction have been limited by the lack of precision with which they determined preprocedural risk. Contemporary cardiac troponin assays have greatly enhanced our ability to detect myonecrosis before and after PCI.[46,51] In a recent analysis, using the currently recommended 99th percentile value as the cutoff for a normal cardiac troponin T level, we found that approximately one third of patients who underwent nonemergency PCI had evidence of preprocedural myonecrosis.[6] These patients had a greater atherosclerotic burden and more unstable disease than patients without evidence of preprocedural myonecrosis, a finding that is consistent with previous reports.[52] Applying the universal definition of myocardial infarction to patients with normal preprocedural cardiac troponin T levels, another one third of patients sustained a periprocedural myocardial infarction after the procedure when cardiac troponin T was used to detect myonecrosis, as compared with only 1 in 15 patients when CK-MB was used.[6] The preprocedural rather than postprocedural cardiac-biomarker level was a powerful independent predictor of short-term and long-term mortality.[6] Similar findings have been reported in two additional recent studies that used cardiac troponin I within the framework of the universal definition of myocardial infarction[36,37] and in an analysis from the Evaluation of Drug Eluting Stents and Ischemic Events (EVENT) registry.[53]

These observations may seem surprising, since one might argue that the clinical effect of myocardial infarction should be the same regardless of its cause. However, most periprocedural myocardial infarcts are very small in relation to the magnitude of myonecrosis, especially in patients with stable coronary artery disease. Among patients with normal preprocedural cardiac troponin values, less than 5% have CK-MB values that are higher than 5 times the upper reference limit after PCI, and Q-wave infarctions are rare (<0.1%). Instead, CK-MB levels that are higher than 5 times the upper reference limit are generally observed in patients with elevated preprocedural cardiac troponin T.[6] Thus, it is likely that in the older studies that explored the effect of periprocedural myocardial infarction on outcomes, a large proportion of the patients who had been classified as biomarker-negative on the basis of CK or CK-MB levels at the time of PCI actually had non–ST-segment elevation myocardial infarction according to contemporary definitions. This conclusion is supported by the high proportion of patients (about 50% on average) who had acute coronary syndromes in the previous studies (Table 2, and the Supplementary Appendix, available with the full text of this article at NEJM.org).

In summary, recent studies reveal that the preprocedural cardiac troponin level is a powerful independent predictor of prognosis after PCI.

Moreover, these studies suggest that the association between postprocedural myonecrosis and outcomes after an otherwise successful PCI is, in general, a reflection of the preprocedural risk, which can be estimated by measuring baseline cardiac troponin levels with the use of contemporary high-sensitivity assays in conjunction with the clinical and angiographic characteristics of the patient.

PROGNOSTIC SIGNIFICANCE OF PERIPROCEDURAL VERSUS SPONTANEOUS EVENTS

On the basis of the traditional concept of periprocedural myocardial infarction described above, this complication has often been equated with spontaneous myocardial infarction in clinical trials.[54] The validity of this assumption has not been examined in detail, and it has been confounded by the variable definitions of periprocedural myocardial infarction used in the past. The current universal definition of myocardial infarction attempts to address this issue by introducing a specific category (type 4a) for periprocedural myocardial infarction to distinguish it from spontaneous myocardial infarction (types 1 and 2).[8]

Akkerhuis and colleagues compared the effect of periprocedural myocardial infarction as detected by CK-MB elevation with that of spontaneous myocardial infarction on 6-month mortality in a heterogeneous group of patients who had acute coronary syndromes without ST-segment elevation; the data were derived from five different clinical-trial databases.[55] The authors reported a positive correlation between CK-MB levels and mortality in both groups, although the absolute mortality was significantly higher among patients who had spontaneous myocardial infarction than among those who had periprocedural myocardial infarction. The authors concluded that the clinical significance of periprocedural myocardial infarction should be considered similar to the adverse consequences of spontaneous myocardial infarction. However, the study was conducted in the era of balloon angioplasty, before the widespread use of stents, and the analysis was not adjusted for confounding clinical variables.

To address these limitations, an analysis was conducted of data from the Acute Catheterization and Urgent Intervention Triage Strategy (ACUITY) trial (Clinical.Trials.gov number, NCT00093158) involving 7773 patients with moderate-to-high-risk, non–ST-segment elevation acute coronary syndromes who underwent PCI.[15] Periprocedural myocardial infarction and spontaneous myocardial infarction during follow-up developed in 6.0% and 2.6% of the cohort, respectively. Among patients with either type of myocardial infarction, as compared with those without myocardial infarction, unadjusted mortality at 1 year was significantly higher. After adjustment for differences in baseline and procedural characteristics between the two groups, spontaneous myocardial infarction was a powerful independent predictor of an increased risk of death, whereas periprocedural myocardial infarction was not significantly associated with an increased risk of death. Similar observations have been made among patients with diabetes and stable coronary artery disease in the Bypass Angioplasty Revascularization Investigation 2 Diabetes (BARI 2D) trial (NCT00006305), in which a first spontaneous, symptomatic myocardial infarction was associated with higher mortality, as compared with myocardial infarction induced by percutaneous or surgical revascularization.[56]

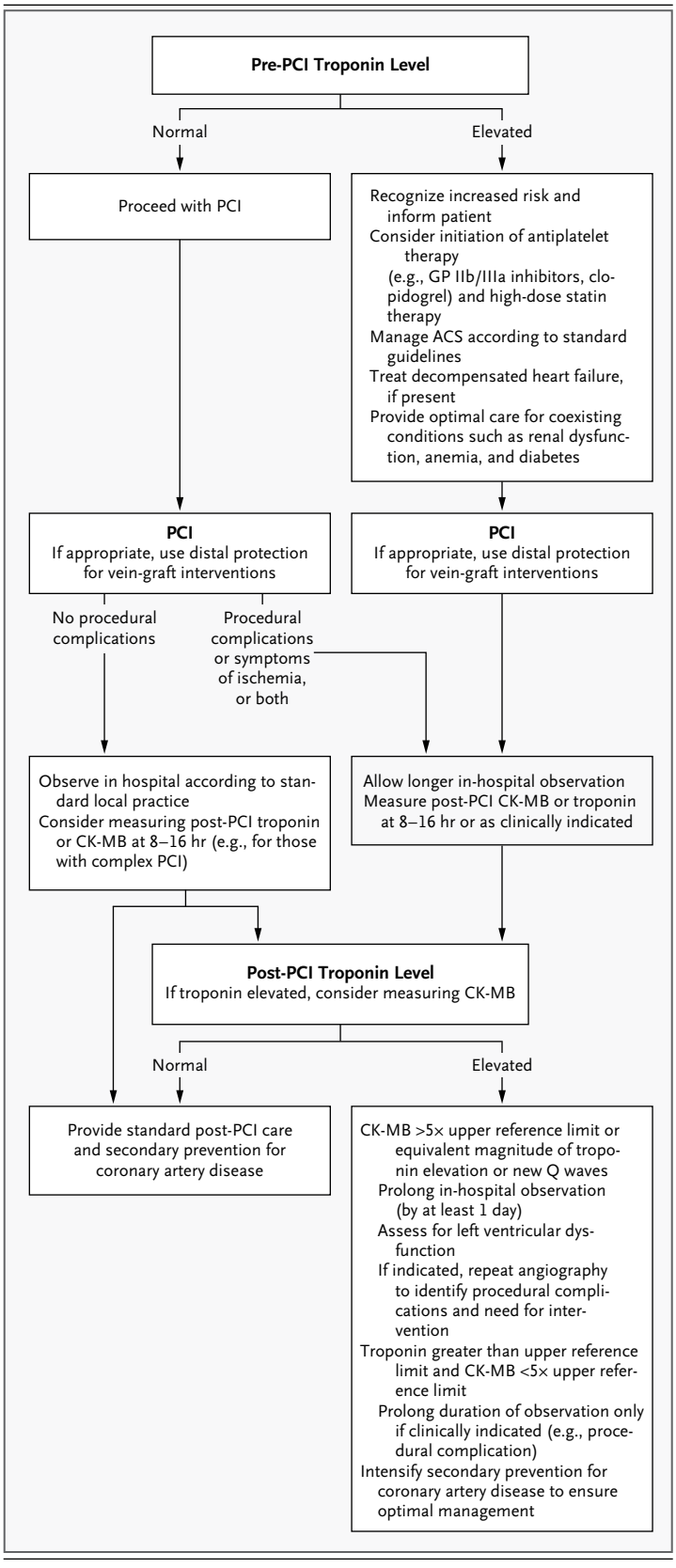
Taken together, contemporary studies indicate that spontaneous myocardial infarction is a powerful predictor of mortality. Periprocedural myocardial infarction, although frequent, is a marker of atherosclerotic burden and procedural complexity, but in most cases, it does not have important independent prognostic significance in stable coronary artery disease or in non–ST-elevation acute coronary syndromes. Although large periprocedural myocardial infarcts may affect prognosis, they rarely occur in the absence of procedural complications or in patients with normal baseline cardiac troponin levels.

AREAS OF UNCERTAINTY

There is a pressing need for the interventional community and the associated professional organizations to examine the new data and provide more practical guidelines for defining periprocedural myocardial infarction. This process should include an assessment of the appropriateness of relying on biomarkers alone and of the low threshold used for the universal definition, as compared with a definition that includes clinical criteria such as symptoms or evidence of ischemia or infarction on electrocardiography or cardiac imaging.

Figure 2. Recommendations for the Prevention and Management of Periprocedural Myocardial Infarction.

ACS denotes acute coronary syndromes, CK-MB MB fraction of creatine kinase, and GP glycoprotein.



Since most of the data on periprocedural myocardial infarction are derived from patients with normal levels of cardiac biomarkers before the procedure (predominantly those with stable or unstable angina), clearer guidelines are needed with regard to whether periprocedural myocardial infarction can be diagnosed in patients with non–ST-elevation myocardial infarction in whom biomarkers are rising before PCI and, if so, what diagnostic criteria should be used. This is probably not feasible in contemporary practice, since PCI is often performed within 24 hours after hospital admission. Another practical issue that needs to be addressed is whether the class IIa recommendation to routinely measure biomarkers after PCI is still appropriate and, if so, what the therapeutic implications of an elevated post-PCI level would be. A recent report from the National Cardiovascular Data Registry indicates that the majority of hospitals in the United States do not routinely measure cardiac biomarkers at the time of PCI.[14]

The improved understanding of the clinical significance of periprocedural myocardial infarction has important implications for the design of future randomized trials (i.e., periprocedural myocardial infarction and spontaneous myocardial infarction should not be considered equivalent clinical end points). This issue has most recently been relevant with respect to the interpretation of data from the Cangrelor versus Standard Therapy to Achieve Optimal Management of Platelet Inhibition (CHAMPION) PLATFORM trial (NCT00385138).[54] In that study, the majority of patients had acute coronary syndromes without ST-segment elevation and underwent PCI within 24 hours after presentation. This did not allow a reliable distinction between spontaneous myocardial infarction and periprocedural myocardial infarction, and led the investigators to conclude that the result of the trial "calls into question the definition of periprocedural MI used." Differentiating spontaneous myocardial infarction from periprocedural myocardial infarction will be increasingly difficult in clinical practice, since most invasively managed cases involve cardiac catheterization during a period when pre-

procedural biomarker levels would generally be rising. Thus, we would caution against including myocardial infarction as a component of the primary composite end point in future clinical trials of PCI in acute coronary syndromes or using it as a surrogate for long-term outcomes, although one might reasonably consider it as a secondary efficacy end point or a safety end point.

IMPLICATIONS FOR PRACTICE

Our recommendation is that cardiac troponin levels be routinely measured before PCI is performed (Fig. 2). A normal preprocedural level of cardiac troponin will assist in risk stratification by identifying patients in whom PCI can be performed with very low risk and who may be considered for early discharge from the hospital. In addition, a pre-PCI elevation in cardiac troponin identifies high-risk patients with complex or thrombotic lesions who may benefit from the preprocedural initiation of potent antiplatelet therapies and statins to improve outcomes.[2,57,58] Post-PCI levels should be routinely measured in patients who have undergone complex procedures, who have suboptimal angiographic results, or who have procedural complications, as well as in those who have signs or symptoms of myocardial ischemia, in order to quantify the extent of myocardial injury. However, a reasonable case can be made for not routinely measuring postprocedural cardiac troponin levels in uncomplicated, successful PCI, since it is not likely that in such cases relevant additional information can be gained that will be independent of the preprocedural risk and procedural outcomes. The role of postprocedural monitoring of biomarkers for risk stratification in the secondary prevention of coronary artery disease or as a metric of quality remains to be established.

There are no established cutoff values for cardiac troponin that define a "large" periprocedural myocardial infarct, and until such values can be clearly identified, a CK-MB level that is more than 5 times the upper reference limit, the presence of new Q waves, or both would appear to be reasonable criteria for defining a periprocedural myocardial infarction as extensive. We believe that, in general, this definition can reliably be applied only to patients with normal cardiac troponin levels before PCI. In the absence of data that can be used to help direct practice, we recommend that patients with large periprocedural myocardial infarction be monitored in the hospital for an additional day because of the reported risks of arrhythmias, hemodynamic instability, heart failure, and death (Table 2, and the Supplementary Appendix). For the purpose of preprocedural consent, one should discuss the frequency of a large periprocedural myocardial infarction (<5%) with the patient and inform the patient if it occurs after the intervention.

The care of patients with acutely elevated preprocedural cardiac troponin who sustain major periprocedural myonecrosis should, in general, be based on the guidelines for managing acute coronary syndromes. Patients whose condition unexpectedly deteriorates soon after PCI (e.g., those with recurrent and unrelenting chest pain, particularly in combination with ST-segment shifts or echocardiographic evidence of ischemia or pericardial effusion) should undergo repeat coronary angiography. The goal is to identify procedural complications that are amenable to further intervention, such as acute stent thrombosis, coronary dissection, or perforation, to limit myonecrosis and relieve symptoms. In most cases, this involves repeat PCI; it is rare in current practice for patients to require cardiac surgery.

Perhaps the most important implication for the long-term care of the vast majority of patients with periprocedural myocardial infarction is the realization that they represent a higher-risk cohort owing to a greater disease burden and more unstable disease. These patients should therefore be targeted for optimal secondary prevention based on the current guidelines. Occasionally, patients with stable coronary artery disease have extensive periprocedural myocardial infarction. The long-term care of such patients should be similar to that for patients with spontaneous myocardial infarction.

Disclosure forms provided by the authors are available with the full text of this article at NEJM.org.

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