

Grade 3 ischemia on the admission electrocardiogram predicts failure of ST resolution and of adequate flow restoration after primary percutaneous coronary intervention for acute myocardial infarction

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Background Failure of ST-segment resolution (STR) after primary percutaneous coronary intervention (PPCI) for ST-elevation myocardial infarction is associated with adverse outcome but currently cannot be predicted on admission. Our aim was to determine whether failure of STR can be predicted from clinical and electrocardiographic data available on admission and whether the adverse outcome associated with grade 3 ischemia (distortion of the terminal portion of the QRS complex) is mediated through impaired tissue reperfusion.

Methods We prospectively studied 100 consecutive patients who underwent PPCI for a first ST-elevation myocardial infarction. Multiple variables available on admission were analyzed as predictors of STR. Electrocardiograms and angiograms were analyzed by blinded investigators.

Results Grade 2 ischemia was found in 71 patients (71%) and 29 (29%) had grade 3 ischemia. Complete STR was observed in 42 (59%) of 71 patients with grade 2 ischemia as compared to 8 (28%) of 29 patients with grade 3 ischemia ($P = .004$). In a multivariate model, grade 3 ischemia was the sole predictor of failure of STR (odds ratio [OR] 0.26, 95% CI 0.1-0.72) and the strongest predictor of failure to achieve TIMI grade 3 flow (OR 0.07, CI 0.02-0.3) and TIMI myocardial perfusion grade 3 (OR 0.09, CI 0.02-0.4) after the procedure.

Conclusions Grade 3 ischemia is a strong independent predictor available on admission of failure to achieve myocardial reperfusion after PPCI, as assessed both electrocardiographically and angiographically. This association may underlie the larger infarcts associated with grade 3 ischemia and may allow the identification upon admission of patients who require more aggressive management to improve reperfusion. (*Am Heart J* 2007;153:410-7.)

Primary percutaneous coronary intervention (PPCI) is the most effective available method to reestablish coronary perfusion in patients presenting with ST-elevation myocardial infarction (STEMI). Although normal epicardial flow can be restored in >85% of cases,^{1,2} prognosis is heavily dependent upon restoration of adequate perfusion at the level of the microcirculation. Many previous studies support the hypothesis that ST-segment resolution (STR) is a surrogate for tissue-level reperfusion³ and is associated

with excellent prognosis.⁴⁻⁶ In fact, STR has been shown to be a stronger predictor of outcome than epicardial reperfusion after PPCI for STEMI.⁷

A number of investigators developed tools to predict on admission the success of epicardial recanalization, infarct size, and mortality in patients undergoing PPCI for STEMI.⁸⁻¹² However, very little data exist on the ability to predict whether or not a patient will achieve tissue level reperfusion after PPCI. A set of bedside clinical variables that would predict on admission the success of reperfusion therapy in terms of myocardial reperfusion could prove useful for the selection of treatment modalities in patients with STEMI and may provide more prognostic information than prediction of arterial recanalization alone.

Distortion of the terminal portion of the QRS complex, as originally described by Sclarovsky et al,^{13,14} is associated with larger infarcts and increased mortality in patients undergoing reperfusion therapy for STEMI. This

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Figure 1



Admission electrocardiograms of a patient with inferior ST-elevation myocardial infarction (STEMI) (**A**) and anterior STEMI (**B**) showing grade 3 ischemia. The J point is above 50% of the R-wave amplitude in leads III and aVF (**A**). There is ST elevation in leads aVL, V2, and V3. There are no S waves in leads V2-V3 (**B**).

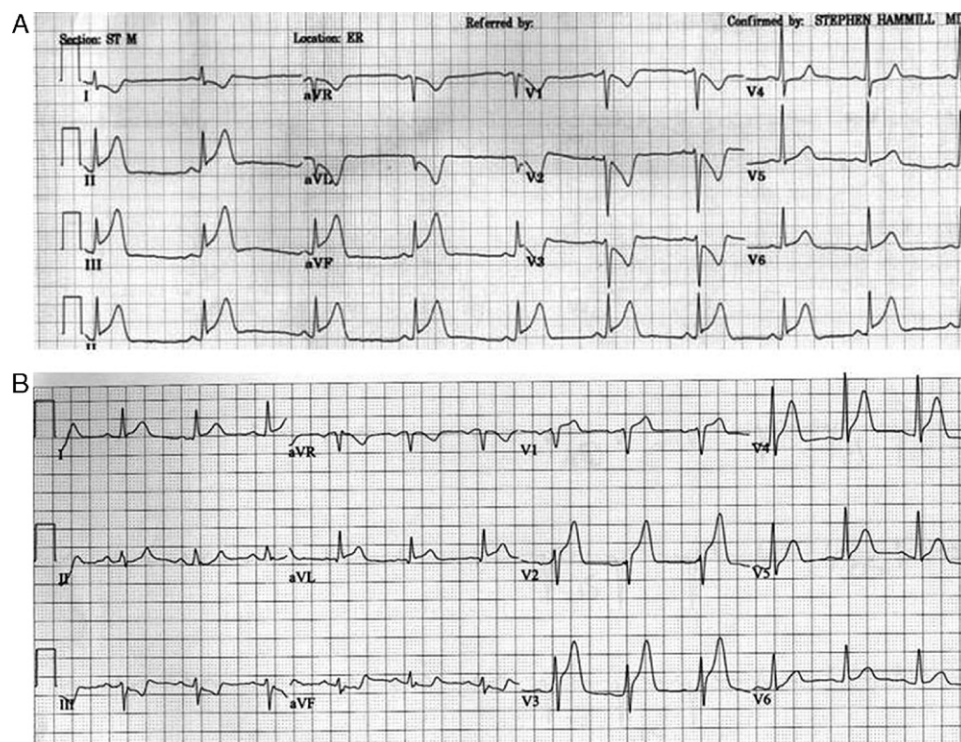
association is independent of the area at risk, time to treatment, and mode of reperfusion. Distortion of the terminal portion of the QRS was classified by Birnbaum et al¹⁵ into 2 groups: grade 3 ischemia (G3I) is characterized by absence of S waves in the right precordial leads and/or a take-off of the ST segment at >50% of the R wave in other leads (Figure 1). ST elevation that does not meet these criteria is classified as grade 2 ischemia (G2I) (Figure 2). Although the association between G3I and adverse outcome is well established, its mechanism is unclear. We have recently shown that in patients receiving thrombolytic therapy G3I on admission predicts failure of STR.¹⁶ Therefore, we hypothesized that G3I on the admission electrocardiogram (ECG) may identify patients who are at high risk of failure of myocardial reperfusion after PPCI. Such an association may offer a mechanistic explanation for the larger infarcts and increased mortality observed in these patients.

Methods

We prospectively studied all consecutive patients admitted to our coronary care unit with a first STEMI and referred for PPCI between July 2004 and April 2005. Patients were excluded if they had previous acute myocardial infarction, >12 hours of symptoms, left bundle branch block, paced or ventricular rhythm, negative T waves in >2 adjacent leads with maximal ST elevation, or incomplete or uninterpretable ECG data. Echocardiograms were obtained within 48 hours of admission. Before angiography, all patients received chewable aspirin (300 mg), clopidogrel (300 mg), and an intravenous bolus of heparin (5000 U). Platelet GP IIb/IIIa inhibitors were given in the catheterization laboratory at the discretion of the operator.

Data collection

Multiple demographic and clinical characteristics available on admission were recorded. Preinfarction angina was defined as new or worsening angina within 48 hours before admission.

Figure 2

Admission electrocardiograms of a patient with inferior STEMI (**A**) and anterior ST-elevation myocardial infarction (STEMI) (**B**) showing grade 2 ischemia. There is ST-segment elevation. However, the J point is below 50% of the R wave amplitude (**A**) and the S waves in leads V1-V3 are preserved (**B**).

Electrocardiographic analysis

The admission ECG (selected as the tracing done immediately before angiography) was analyzed in a core laboratory at the University of Texas Medical Branch at Galveston by 2 investigators (SA, YB) blinded to the patient's clinical, subsequent electrocardiographic, and angiographic data. Disagreements between the investigators were resolved by consensus. The following parameters were recorded:

1. ST segment elevation: The magnitude of ST segment elevation at the J point in each lead (except aVR) was measured manually to the nearest 0.05 mV using a magnifying glass.
2. Grade of ischemia: Patients were divided into 2 groups according to their grade of ischemia on the admission ECG, as previously described.¹³ Grade 3 ischemia (G3I) was defined as: (1) absence of an S wave below the TP-PR isoelectric line in >2 leads that usually have a terminal S configuration (leads V1-V3), or (2) ST J-point amplitude >50% of the R-wave amplitude measured from the TP-PR baseline in >2 all other infarct-related leads. Patients meeting the ST-elevation criteria but not the grade 3 ischemia criteria were classified as having grade 2 ischemia (G2I).
3. Q waves: Q waves were analyzed as categorical variables in the following manner: (0) no pathologic Q waves or shallow pathologic Q waves, defined as a depth >25% of the R wave and width >40 milliseconds (in the absence of criteria for deep pathologic Q waves); and (1) deep pathologic Q waves, defined as either Q waves with total loss of R wave or deep Q waves of >5 mm (0.5 mV) in the limb leads and >10 mm (1 mV) in the chest leads. Q waves in lead III only were not read as pathologic.¹⁷
4. ST segment elevation resolution: ECGs were obtained immediately upon the patient's return to the coronary care unit after PPCI, within 15 to 30 minutes from completion of the procedure. Two end points of STR were examined: 70% or 50% reduction in ST elevation in the single lead with maximum ST elevation on the baseline ECG.

Angiographic analysis

Angiograms were read by 2 experienced angiographers (CC, SY) who were blinded to all clinical and electrocardiographic data. Multivessel disease was defined as the presence of >70% luminal diameter stenosis in >1 major epicardial vessel. A validated score for severity of coronary artery disease¹⁸ was calculated in every case. Flow in the infarct-related artery was

Table 1. Comparison of patients with successful ($\geq 70\%$) versus unsuccessful ST resolution—baseline, electrocardiographic, and laboratory characteristics

	Failure of STR (n = 50)	Successful STR (n = 50)	P value
Baseline characteristics			
Age, y*	61 $\pm$ 12	58 $\pm$ 11	.25
Male	37 (74)	40 (80)	.63
Smokers	18 (36)	24 (48)	.22
Diabetes mellitus	13 (26)	10 (20)	.48
Peripheral vascular disease	1 (2)	3 (6)	.31
Hypertension	19 (38)	13 (26)	.20
Dyslipidemia	35 (70)	35 (70)	.90
Prior use of aspirin	14 (28)	9 (18)	.22
Prior use of β -blockers	5 (10)	7 (14)	.54
Prior use of statins	10 (20)	7 (14)	.42
Preinfarction angina	15 (30)	18 (36)	.52
Heart rate*	76 $\pm$ 16	81 $\pm$ 28	.28
Systolic blood pressure (mm Hg)*	132 $\pm$ 19	131 $\pm$ 29	.85
Time from symptoms to admission (min)	222 $\pm$ 169	198 $\pm$ 175	.47
Time from symptoms to balloon (min)	256 $\pm$ 176	232 $\pm$ 184	.50
Platelet glycoprotein IIb/IIIa inhibitors	29 (58)	25 (50)	.42
30-d mortality	3 (6)	2 (4)	.65
Electrocardiographic findings			
Grade 3 ischemia	21 (42)	8 (16)	<.01
Q waves on admission	9 (18)	5 (10)	.24
Laboratory results			
Admission troponin T level (ng/mL)*	0.08 $\pm$ 0.13	0.34 $\pm$ 1.11	.13
Peak troponin T level (ng/mL)*	6.82 $\pm$ 6.89	4.06 $\pm$ 4.32	.06
Admission CK level (U/mL)*	201 $\pm$ 395	275 $\pm$ 605	.48
Peak CK level (U/mL)*	2611 $\pm$ 2257	1628 $\pm$ 1740	.02
Admission glucose level (mg/dL)*	173 $\pm$ 79	154 $\pm$ 103	.30
Echocardiographic results			
Significant LV dysfunction	34 (76)	19 (48)	.01

Data are numbers (%).

CK, Creatine kinase.

*Mean $\pm$ SD.

graded according to the TIMI scale¹⁹ and grouped for the purpose of this analysis as TIMI flow = 3 or TIMI flow < 3. The corrected TIMI frame count²⁰ and the TIMI myocardial perfusion (TMP) grade²¹ were also determined. TIMI myocardial perfusion was grouped for the purpose of this analysis as TMP grade = 3 or TMP grade < 3. Adverse procedural result was defined as the presence of dissection, coronary emboli, no-reflow, a significant (>50%) residual stenosis, or residual thrombus. No-reflow was defined by the presence of TIMI flow < 3 at the end of the procedure in the absence of a flow-limiting stenosis.

Echocardiographic analysis

Significant left ventricular (LV) dysfunction was defined as moderate or severe LV systolic dysfunction (corresponding to an LV ejection fraction <40%), as assessed by echocardiography.

Statistical analysis

The primary end point was complete STR, defined as STR $\geq 70\%$ immediately after PPCI. A secondary end point was STR $\geq 50\%$ immediately after PPCI. Continuous variables are expressed as mean $\pm$ SD. The χ^2 test was used to compare dichotomous variables, and Student *t* test was used for continuous variables. Demographic, clinical, and electrocar-

diographic variables present on admission were evaluated as univariate predictors of STR and of angiographic findings. Factors identified as univariate predictors of STR as well as clinically important variables were then entered in a logistic regression model to identify independent predictors of ST segment resolution. The multivariate model included age, sex, time to admission, preinfarction angina, previous aspirin therapy, diabetes, grade of ischemia, and Q waves on admission.

Grade 3 ischemia was also tested as a predictor of angiographic signs of reperfusion, including TIMI flow grade, TIMI frame count, TMP grade, and an adverse procedural result. The multivariate model for TIMI flow grade and TMP grade included age, sex, time to treatment, preinfarction angina, prior aspirin therapy, diabetes, admission glucose level, grade of ischemia, and Q waves on admission. Odds ratios and 95% CIs were calculated. The level of significance was .05. All statistical tests were done using SPSS (version 12.0) software (SPSS, Chicago, IL).

Results

Between August 2004 and April 2005, 100 consecutive patients were enrolled. Fifty (50%) of them achieved

Table II. Comparison of G2I versus G3I patients—laboratory, angiographic, and echocardiographic findings

	G3I	G2I	P value
Laboratory results			
Admission glucose level (mg/dL)	207 ± 135	146 ± 60	<.001
Admission CK level (U/mL)	483 ± 891	139 ± 133	<.001
Peak CK level (U/mL)	2992 ± 2501	1763 ± 1757	.01
Peak troponin T level (ng/mL)	8 ± 7	4 ± 5	.01
Angiographic results			
Multivessel coronary artery disease	13/29 (45)	40/71 (56)	.29
Culprit artery			
LAD	19 (65)	32 (45)	.17
RCA	8 (28)	31 (44)	
Circumflex	2 (7)	8 (11)	
Proximal culprit lesion	11/29 (38)	20/71 (28)	.34
Type C lesion vs others	22/29 (76)	52/71 (73)	.78
Angiographic score*	5 ± 2	5 ± 2	.94
Initial diameter stenosis (%)*	100 ± 2	99 ± 2	.47
Final diameter stenosis (%)*	8 ± 25	3 ± 12	.14
Visible thrombus	15/29 (52)	21/71 (30)	.04
Calcified lesion	2/29 (7)	9/71 (13)	.4
Collaterals	3/29 (10)	18/71 (25)	.09
Balloon predilatation	14/29 (48)	44/71 (62)	.21
Stent implantation	27/29 (93)	66/71 (93)	.98
IABP	3/29 (10)	5/71 (7)	.58
Initial TIMI flow grade 3 vs TIMI flow grade <3	2/29 (7)	2/71 (3)	.35
Final TIMI flow grade 3 vs TIMI flow grade <3	17/29 (59)	65/71 (92)	<.001
Final TMP grade 3 vs TMP grade <3	5/23 (22)	43/58 (74)	<.001
Initial corrected TIMI frame count*	83 ± 34	81 ± 31	.85
Final corrected TIMI frame count*	21 ± 22	11 ± 6	<.001
Adverse procedural result†	12/29 (41)	6/71 (8)	<.001
Echocardiographic results			
Significant LVD	21/27 (78)	32/58 (55)	.05

Data are numbers (%).

LAD, Left anterior descending artery; RCA, right coronary artery; IABP, intra-aortic balloon pump; LVD, left ventricular diastolic function.

*Mean ± SD.

†No-reflow, acute closure, branch compromise, residual thrombus, or dissection.

complete STR immediately after PPCI. All baseline and laboratory characteristics available on admission were similar in patients who achieved complete STR and in those who did not, except for a higher rate of G3I on admission in patients who eventually failed to achieve STR (42% vs 16%, $P < .01$, Table I). Patients who failed to achieve complete STR eventually had higher peak creatine kinase levels (2611 ± 2257 vs 1628 ± 1740 U/L, $P = .02$) and a higher rate of significant LV dysfunction (76% vs 48%, $P = .01$) than those who did achieve complete STR.

Grade of ischemia

G2I on the admission ECG was found in 71 patients (71%) and 29 (29%) had G3I. Patients in the 2 groups did not differ significantly in terms of age, sex, previous medical therapy, coronary risk factors, peripheral vascular disease, time to treatment, preinfarction angina, vital signs on admission, or admission troponin T (TnT) levels. G3I patients had higher admission glucose, higher admission and peak creatine kinase, and higher peak

TnT levels (Table II). Patients who had G3I had more often significant LV dysfunction (78% vs 55%, $P = .05$, Table II). Thirty-day mortality was 4% and 6% in patients with G2I and G3I, respectively ($P = 0.67$).

Complete STR immediately after PPCI was observed in 42 (59%) of 71 patients with G2I as compared to 8 (28%) of 29 patients with G3I ($P = .004$). The multivariate model identified G3I as the sole predictor of failure of STR (OR 0.26, 95% CI 0.1-0.72). Similar findings were obtained when the analysis was repeated with 50% STR as the end point (OR 0.41, 95% CI 0.04-0.56). Positive and negative predictive values for G3I as a predictor of failure to achieve 70% STR were 72% and 60%, respectively.

Angiographic data

Patients presenting with G3I were similar to G2I patients in terms of the extent of disease, culprit artery distribution, initial TIMI grade flow and corrected TIMI frame count, and initial and final luminal diameter stenosis (Table II). However, thrombi were visible more often in G3I than in G2I patients (52% vs 30%, respectively, $P = .04$). We observed a trend toward more

Table III. Multivariate predictors of TIMI flow grade 3 and TIMI myocardial perfusion grade 3

	TIMI flow grade 3			TIMI myocardial perfusion grade 3		
	OR	95% CI	P value	OR	95% CI	P value
Male sex	24	1.6-360	.02	1.78	0.4-8.5	.48
Age	0.99	0.9-1.1	.74	0.95	0.89-1.1	.71
Time from symptoms to admission (median)	0.37	0.09-1.34	.13	0.21	0.06-0.8	.02
Pathologic Q waves	2.35	0.41-13	.34	0.19	0.04-1.1	.06
Preinfarction angina	0.58	0.15-2.23	.43	0.74	0.20-2.7	.65
Diabetes	0.11	0.08-1.67	.11	1.06	0.18-6.33	.95
Prior aspirin therapy	5.1	1.1-24	.04	1.27	0.28-5.7	.76
Glucose level on admission	0.99	0.98-1	.24	0.99	0.98-1	.19
G3I	0.07	0.02-0.33	.01	0.09	0.02-0.39	.01

All ORs are the odds when having the attribute to achieve TIMI grade 3 flow or TIMI myocardial perfusion grade 3.

frequent visible collaterals in G2I patients than in G3I patients (25% vs 10%, respectively, $P = .09$). Despite these similar findings on the diagnostic angiogram, patients presenting with G3I had substantially lower rates of final TIMI grade 3 flow and TMP grade 3 than patients presenting with G2I: 59% versus 92% ($P < .001$) and 22% versus 74% ($P < .001$), respectively. Correspondingly, G3I patients had higher final corrected TIMI frame counts than G2I patients (21 ± 22 vs 11 ± 6 , $P < .05$). An adverse procedural result was much more frequent among G3I patients than among G2I patients (41% vs 8%, $P < .05$).

Multivariate analysis showed that G3I was the strongest predictor of failure to achieve TIMI grade 3 flow (OR 0.07, CI 0.02-0.3), followed by male sex (OR 24, CI 1.6-360) and prior aspirin therapy (OR 5.1, CI 1.1-24) (Table III). When the same model was used for prediction of TMP, G3I (OR 0.09, CI 0.02-0.4) and pain to admission time (OR 0.21, CI 0.06-0.8) independently predicted failure to achieve TMP grade 3 (Table III).

Discussion

The primary finding of this study is that distortion of the terminal portion of the QRS (G3I) on the presenting ECG in patients scheduled for PPCI for STEMI is a very strong independent predictor, available on admission, of failure to achieve myocardial reperfusion as assessed both electrocardiographically and angiographically. Furthermore, G3I is the strongest predictor of an adverse procedural outcome.

Early risk assessment of acute STEMI patients referred for PPCI has been previously attempted mostly by constructing risk scores²²⁻²⁴ or by assessing the prognostic value of a limited number of parameters such as the admission glucose level or the white blood cell count.²⁵⁻²⁷ A few studies assessed the ability of the admission ECG to predict the short- and long-term outcome of PPCI. Parameters such as bundle branch block,^{22,28} sum of ST elevation²⁹, and the number of Q waves³⁰ were found to correlate with poor prognosis.³¹

The adverse prognostic implication of G3I in patients given reperfusion therapy has been well demonstrated. These patients are prone to larger infarcts as compared to G2I patients despite a similar area at risk^{15,32} and have higher rates of reinfarction and mortality.^{15,33-35} In the setting of PPCI, Birnbaum et al³² found that G3I on admission was associated with higher rates of mortality and reinfarction.³⁶ Other investigators showed that G3I predicted angiographic no-reflow.^{36,37} The mechanism underlying this association remains unclear. We have recently shown in a retrospective study that G3I on admission was the strongest predictor of failure to achieve complete STR after thrombolytic therapy.¹⁶ The present prospective work extends these observations to patients treated by PPCI. We have systematically and prospectively examined multiple clinical, electrocardiographic, and angiographic parameters, including the grade of ischemia, as predictors of ST-segment resolution. The study was conducted in the era of combined antithrombotic treatment and routine stenting for the treatment of acute STEMI. Baseline characteristics in patients with G2I and those with G3I were generally similar, except for higher admission glucose levels in the latter. This parameter was not found, however, to be an independent predictor for TIMI flow or for the TMP grade. Electrocardiograms (read in a core laboratory) and angiograms were analyzed by investigators blinded to other aspects of the data. Our electrocardiographic analysis revealed a substantial, highly significant difference in the rate of complete ST-segment elevation resolution between patients presenting with G2I versus G3I (59% vs 28%, respectively, $P = .004$). The angiographic comparison of patients with G2I and patients with G3I showed that most initial angiographic variables (with the exception of visible thrombus) were similar in the 2 groups. In contrast, the final angiographic result was markedly different in the 2 groups: patients presenting with G3I had worse final TIMI flow grades, TIMI frame counts, and TMP grades as well as a higher rate of adverse procedural result. These findings are consistent with

the higher rates of failure of ST resolution and of significant LV dysfunction in these patients.

Taken together, our studies in patients given thrombolysis or PPCI offer a mechanistic link between G3I and the poor outcome observed in these patients. Apparently, G3I is a powerful predictor of poor tissue-level reperfusion, possibly because of severe ischemic damage to the microvasculature.

Our findings have 2 important implications. First, we identified a simple measure, available on admission, that is a powerful predictor of failure of STR and of myocardial reperfusion post-PPCI for STEMI. Determining the grade of ischemia does not require any calculations and can be readily performed after brief training with the use of a ruler. The simplicity of this measurement and its substantial prognostic value may merit incorporating the grade of ischemia into automatic electrocardiogram analysis algorithms. Second, we offer a mechanism for the well-described association between G3I on admission and poor prognosis, as manifested by larger infarcts and increased mortality. It appears that these patients have a substantially lower rate of myocardial reperfusion after arterial recanalization. This may be explained in part by the larger clot burden and the poorer collateral supply observed in these patients. These observations may allow future investigators to identify on admission patients who are at high risk of failure of myocardial reperfusion and specifically direct efforts aimed to improve myocardial reperfusion (eg, intracoronary vasodilators, IIb/IIIa inhibitors) at this high-risk subgroup.

Our study has some limitations: although prospective and blinded, it is relatively small. Furthermore, some admission clinical parameters such as history of heart failure, renal failure, Killip class, and serum creatinine were not routinely recorded. Further confirmation of these observations in larger studies would be useful.

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